Biol. Cybern. 72, 197–206 (1995)

Biological Cybernetics

A neural network model of phantom limbs

Manfred Spitzer, Peter Böhler, Matthias Weisbrod, Udo Kischka

Section of Experimental Psychopathology, Psychiatrische Universitätsklinik Heidelberg, Voss-Strasse 4, D-69115 Heidelberg, Germany

Received: 18 February 1994/Accepted in revised form: 7 July 1994

Abstract. This paper presents a detailed clinical description of phantom limbs and a neuronal network model that provides a comprehensive and parsimonious explanation of otherwise inexplicable or at least unrelated phenomena. Simulations of self-organizing feature maps (Kohonen networks) that had been trained to recognize input patterns were deprived of parts of their input in order to simulate partial deafferentation. This leads to reorganization processes that are shown to be driven by input noise. In patients with an amputated limb, this noise is generated by dorsal root ganglion sensory neurons which are known to fire irregularly upon laceration. According to this model, the long-standing debate concerning non-cortical vs. cortical contributions to the generation of the phenomenon of phantom limbs can be resolved in that it is the peripherally generated noise that causes cortical reorganization. The model can be tested and may have therapeutic implications.

1 Introduction

The term phantom limb was introduced by Mitchell (1871) to denote sensations of the presence of an extremity that has been lost. These occur in 80%–100% of amputees (Jensen and Rasmussen 1989), but only rarely and negligibly in paraplegics (i.e. patients with lesions of the spinal cord). For example, after the amputation of a leg, patients still experience the presence of the leg, feel the foot in a certain position, experience movements of the leg as well as tingling or itching sensations from the leg's skin. Phantom limbs are often painful.

From the extensive literature on the phenomenon, the following clinical features of phantom limbs can be summarized (cf. Buchanen and Mandel 1986; Carlen et al. 1978; Cronholm 1951; Jensen et al. 1984; Katz 1992; Poeck 1963; Spitzer 1988; Weiss and Fishman 1963):

(1) The more severe the trauma, the more likely and/or the more extensive is the phantom limb. Elective amputations, for example, cause fewer and less pronounced phantoms than traumatic amputations.

(2) Phantoms change over time. This is known as (a) 'telescoping' and (b) 'shrinking'. (a) In approximately one-third of the patients, the phantom limb becomes shorter, such that the hand eventually is felt as if it were attached directly to the shoulder, or the foot directly to the knee or hip. Hand or foot, as experienced in space, may even completely retract into the stump, but still be felt as a hand or foot. (b) In addition to the shrinkage in length, the size of the experienced phantom may shrink such that the hand is still felt, but like the size of a child's hand or as small as a postage stamp.

(3) In addition to the continuing experience of the presence of the amputated limb and sensations from the stump, there are referred sensations, i.e. 'stimulation of the stump evokes sensations as if the phantom were a physical reality and were itself stimulated. The phantom hand is 'represented' by the distal part of the stump, and the phantom-forearm merely by a narrow zone proximal on it. Conditions might be described by saying that the stump had 'taken over' the sensory functions of the lost limb [. . .]' (Cronholm 1951, p. 183). These referred sensations are topographically organized (see Fig. 1) and maintain the quality of the original sensation: touching the stump is experienced as a touch to the stump and a touch to a specific point on the phantom limb, a breeze at the stump is felt as two breezes, and water running down the stump is experienced as water running down the stump and the arm or the leg.

(4) Sensory acuity of the stump is increased, and the threshold of two-point discrimination is decreased, as comparisons between the skin on the stump with contralateral sites reveal. Moreover, the closer the skin area is situated towards the amputation site, the more pronounced are these effects.

(5) In cases of amputations of the forearm below the elbow, one of the surgical options is to use the remaining parts of the two bones, radius and ulna, to form a 'Krukenberg forceps', i.e., a gripping organ which can be trained to be used for simple everyday grasping tasks. In such patients, the phantom hand can get mapped onto the forceps, such that some fingers may disappear, while

Correspondence to: M. Spitzer

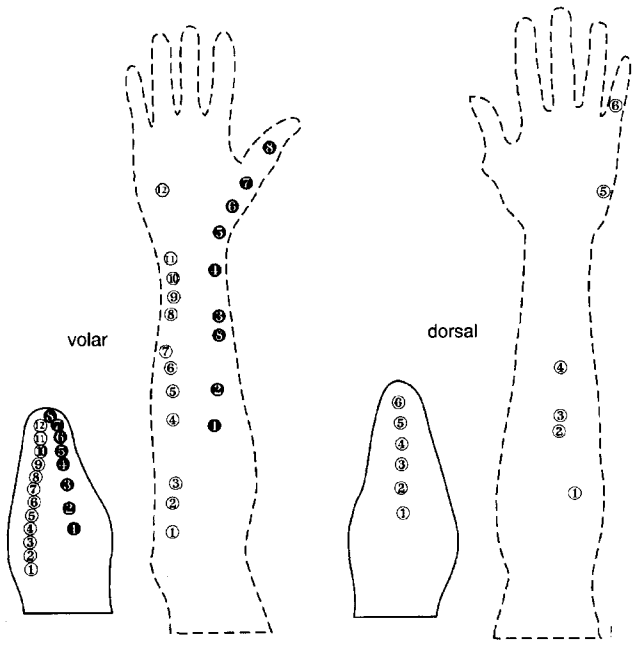

Fig. 1. Localization of referred sensations as experienced in the phantom arm and hand when the volar (**A**) and the dorsal (**B**) stump was stimulated by touching it with the blunt end of a pencil. The localization of the subjective sensations were drawn on a chart. The points on the stump that were stimulated are numbered and indicated by a *filled small circle* (•), the points of the subjective experiences are charted using an *open small circle* (◦) and corresponding numbers (redrawn after Cronholm 1951, p. 190)

others come to represent the two parts of the artificially constructed forceps. Grasping movements of the forceps were paralleled by the subjective experience of grasping with the phantom hand.

(6) Both the extent and clarity of these sensations vary greatly from case to case. Phantoms may not develop in patients with low intelligence or with senile dementia. Clinicians have furthermore stressed the importance of characterological variables in the development of phantom limbs (Schilder 1923; Zuk 1956; cf. Spitzer 1988 for a summary and critical comment of the older literature), though there is only anecdotal evidence in support of this conjecture (cf. Kartz 1992).

(7) Some patients experience discomfort or even pain if the experimenter strikes the air at the place corresponding to the experienced localization of the phantom limb (cf. Cronholm 1951).

In general, the development of phantom limbs and their clinical features appears, to be, at least in part, dependent upon central and peripheral physiological and also psychological variables. This has led to a fierce debate, described by Hoffman (1954, p. 263) as 'long and bitter with much evidence for and against each side [. . .]'. In the following two sections, evidence in favour of the central cortical and peripheral views will be presented.

2 What causes phantom limbs? The cortical view

The above-mentioned clinical observations are compatible with the view that the experience of phantom limbs is produced by a cortical mechanism. This view was held by a number of 'classic' authors. Cronholm (1951), for example, assumed the presence of 'cortical hyperexcitability' in amputees as one mechanism that causes the subjective phantom experiences. In recent years, the cortical view received further support from electrophysiological evidence of cortical reorganization due to the changes in the general patterns of input signals.

Since the work of Penfield (cf. Penfield and Rasmussen 1950), we know that the primary sensory cortex has a map-like structure. Recent studies in animals and human beings (cf. Merzenich and Sameshima 1993) show that these maps are not static, but rather are subject to constant change driven by the organism's experience. Such change not only occurs during the development stage of the organism, but throughout life until death.

To give an example: Recanzone et al. (1992a) trained monkeys to discriminate different frequencies of tactile vibrations presented to a constant location of the glabrous skin of a single finger. The detection thresholds for the trained digit decreased from a difference of 6 to 8 Hz to a difference of 2 to 3 Hz. Generalization to other fingers was limited. These findings were taken as evidence for local changes in a somatotopically organized somatosensory cortex. This interpretation was validated by electrophysiological recordings from somatosensory neurons: 'There was a greater territory of representation of the small area of skin that was stimulated in the behavioral task in trained monkeys, when compared with the representations of corresponding skin sites in the opposite hemisphere of the same monkey [. . .]' (Recanzone et al. 1992b, p. 1057). It was further demonstrated that by the training, 'distributed spatial and temporal response properties of cortical neurons are altered' (Recanzone et al. 1992c, p. 1071).

Pons et al. (1991) found cortical somatosensory maps to be capable of a particularly large degree of reorganization. Whereas it used to be assumed that limited deafferentation in adult primates leads to the reorganization of somatosensory areas of 1 to 2 mm (the size of the cortical projection zones of individual thalamic neurons), the authors found changes over distances of up to 14 mm. In one of their animals, the cortical area corresponding to the lost limb actually became responsive to stimuli applied to a region of the face.

Merzenich and his group (cf. Merzenich and Sameshima 1993) conducted a number of peripheral lesion experiments that add further evidence of dramatic cortical plasticity in adult primates. Merzenich et al. (1983) were the first to demonstrate self-organization capabilities in the somatosensory cortex after restricted deafferentation. The authors advanced the hypothesis that it is the spatiotemporal coherence of the input pattern which leads to their representation on the cortical surface. In this view, input signals compete for representational space on the cortical surface. The more similar the input signals, the closer they will eventually be

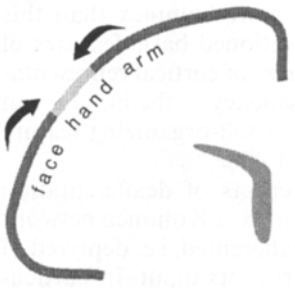

Fig. 2. Cortical map of sensory input. The homunculus depicts the relative size of the cortical fields representing sensory information from the various body parts (adapted from Penfield and Rasmussen 1950). Note that the area coding sensory input from the hand lies between areas coding sensory input from the arm and from the face

located together. The process of translocation of input representation is driven by competition between cortical neurons.

In addition to this line of electrophysiological reasoning, Ramachandran et al. (1992) reported a clinical observation which provides further strong evidence in favour of the cortical view of the genesis of phantom limbs (see also Halligan et al. 1993). In particular, the authors observed that stimulation of the face in a patient with an amputated arm can evoke localized and topographically mapped sensations in the phantom hand. As the face is not adjacent to the arm, but the cortical representation of the face is adjacent to the cortical representation of the arm (see Fig. 2), this finding supports the view that cortical reorganization drives the development of phantom limbs.

This finding led Ramachandran et al. (1992, p. 1160) to propose the hypothesis that 'tactile and proprioceptive input from surrounding tissue 'takes over' the brain areas corresponding to the amputated limb', and that 'spontaneous discharges arising from neurons innervating these tissues would be misinterpreted as arising from the missing limb'. In particular, the authors 'expect sensory input from both these regions [face and arm] to 'invade' the cortical hand area and provide a basis for referred sensations.'

The cortical view of phantom limb causation may be summarized as follows:

- The cortex is a two-dimensional computational map-like surface.
- This surface changes according to the spatiotemporal characteristics of the input.
- If an area becomes deafferented (i.e. deprived of its input), reorganization takes place.
- Either during this process or as a result of this process, neurons representing input patterns that are no longer present due to deafferentation become activated. This activation is subjectively experienced as the phantom sensation.

3 The non-cortical view of phantom limb causation

A number of authors have suggested that phantom limbs are caused by a peripheral mechanism or by a mechanism at the level of the spinal cord. According to this non-cortical view, free nerve endings, neuromas and axons proliferating into scar tissue are regarded as the cause of phantom pain which in turn is supposed to lead to phantom sensations. Poeck (1963) quotes several authors who suggested that phantom limbs are caused by interactions between efferent autonomic and afferent sensory fibres. This hypothesis was refined and put forward in a recent review by Katz (1992). He proposed that ' a phantom limb, whether painful or not, is related to the sympathetic-efferent outflow of cutaneous vasoconstrictor fibers in the stump and stump neuromas' and that 'the paresthetic or dysesthetic component of the phantom limb may be triggered by sympathetic-efferent activity' (Katz 1992, p. 290).

In addition to peripheral nerves, the spinal cord has been implicated in phantom limb genesis. For example, from his detailed clinical observations, Cronholm concluded that hyperexcitability of the spinal cord has to be the mechanism by means of which phantom limbs come into existence. This non-cortical view of the causation of phantom limbs – either at the level of the peripheral nerve or the spinal cord – is supported by one striking clinical finding : patients with lesions of the spinal cord and the clinical condition of paraplegia rarely develop phantom sensations. If they do, these sensations are weak, they lack detail, and the sensations occur months after the onset of paraplegia. Carlen et al. (1978, p. 216) correctly point out the apparent consequence of this observation for the cortical view: If phantoms were generated by the activity of brain cells rather than cord cells, the paraplegic should report an even more vivid phantom sensation since his brain has lost even more input than an amputee's.'

In special support of this view are two cases with thoracic spinal cord lesions and additional arm amputation (Bors 1951). These cases allow the intraindividual comparison of the effects of amputation and spinal cord lesion. In both cases there was a pronounced and clearly experienced phantom of the arm but only a weak and hard to describe phantom of the lower body. This clinical observation makes it highly unlikely that individual differences account for the differences in the phantom experience of amputees and patients with spinal cord lesions. The inevitable conclusion appears to be that the presence or absence of phantom sensations in these two patient groups has to be accounted for by differences located in the spinal cord of the peripheral nerve. The non-cortical view of phantom limb causation may hence be summarized as follows:

- Amputees suffer from a lesion of the peripheral sensory nerve.
- Paraplegics with lesions of the spinal cord do not (or to a small degree) experience phantom limbs.
- Both conditions imply cortical deafferentation, which therefore cannot explain the differences.
- Phantom sensations have to be caused by a mechanism that involves the peripheral nerve endings and/or the spinal cord.

4 Combining both views: self organizing feature maps and noise

To sum up our discussion, there are two apparently incompatible views of the cause of phantom limbs. Behavioral and electrophysiological evidence, in particular the findings of Pons and Ramachandran, clearly point to the cortex as the site of plasticity and reorganization. However, the observation that paraplegics generally do not experience phantom sensations has led clinicians to conclude that phantom limbs must be generated at the level of the peripheral nerve or of the spinal cord, since that is where amputees differ from paraplegics. This apparent conundrum can be solved by a particular neural network model of the cortex and biologically plausible assumptions about the characteristics of sensory input in amputees and paraplegics. In the remaining part of this paper, this model is outlined and a brief introduction to self-organizing feature maps provided. Then, simulations performed on a personal computer with a small, commercially available neural networks package are presented, and finally, these results are discussed in the light of the clinical data. We conjecture that our model provides a parsimonious explanation for many otherwise unexplained or at least seemingly unrelated phenomena.

Self-organizing feature maps, as described by Kohonen (1989), are two-layered networks in which every input node is connected to every output node. In addition, the neurons of the output layer are connected between themselves such that every neuron is wired up with every other neuron in this layer. These connections are excitatory for nearby neurons and inhibitory for neurons further away. The net result of this is the implementation of a general functional feature of the cortex, i.e. focused local activation and lateral inhibition (Creutzfeld 1993), which is critical to the network's self-organizing property. Lateral inhibition creates competition between neurons in the layer. When an input pattern is present at the input layer, each neuron of the output layer receives a complete copy, modified by the strength (weights) of the synaptic connections between the input neurons and the output neurons. Due to the intralayer connections, only one neuron will become most activated by a given input, suppress the activity of neurons further away, and activate nearby neurons (competition). If hebbian learning is implemented, the synaptic connections between active input neurons and the 'winner' neuron are strengthened (competitive learning). Likewise, the connections between the adjacent neurons and the active input neurons are strengthened to some degree. Hence, the same input pattern will be recognized by the winning neuron with a higher probability, and input patterns similar to that input pattern are more likely to lead to the activation of a neuron in the neighbourhood of the winning neuron.

It has been shown that networks of this type have the general capability of organizing any coherent input according to its most frequent and most salient features over a given two-dimensional neuronal layer. In other words, it generates receptive fields that are ordered according to features of the input.

While the cortex is surely more complex than this type of network, the above-mentioned basic features of the cortex – dependency of the size of cortical representations upon the frequency and saliency of the input – can be plausibly modelled with such self-organizing feature maps (cf. Ritter and Kohonen 1989).

In order to simulate the effects of deafferentation caused by amputation on the cortex, a Kohonen network was trained and then partly deafferented, i.e. deprived of a topologically constrained part of its input. In particular, the simulation was designed to study the redistribution of neuronal representations of the input patterns. Therefore, the distribution of representations of the input was recorded under various operational circumstances. Activation of neurons in the deafferented part of the network was found to be dependent upon input noise.

5 Simulations

A simple implementation of self-organizing feature maps was used to simulate the effects of amputation (i.e. of the sudden removal of parts of the input) on a trained network. A 10×10 neuron Kohonen network was trained to recognize the characters of the alphabet, as represented by an array of 5×7 input nodes (see Fig. 3, top). After about 400 training cycles, using a decreasing size of the neighbourhood (i.e. the set of neurons surrounding a winning neuron which receive activation from the winning neuron), the characters of the alphabet were spread across the map in an orderly way, i.e. salient features of the characters determined the spatial distribution of the winning neurons within the network (see Fig. 3, middle).[1] This trained network was saved and used as the basis for further simulations.

Amputation was simulated by removing the characters from the input file which had come to be represented in one quadrant of the map. The feature map was then trained again with this restricted set of input patterns. When the neighbourhood size was kept to zero or one, this procedure did not lead to the activation of neurons within the deafferented part of the network, i.e. to the rapid redistribution of the winning neurons over the entire network. However, such activation of neurons in the deafferented area took place if noise was added to the input (Fig. 3, bottom).

Noise was implemented by adding random numbers (generated from a standard C-language random number generator) to the input patterns, which under conditions of no noise consisted of patterns of the values 0 and 1. At a given noise level x, random numbers from $-x$ to x were added to the input signal before presenting it to

[1] Simulations were performed using an IBM-compatible 80386 CPU personal computer running inexpensive and easy to use software intended for use in teaching the neural network approach (cf. Caudill and Butler 1992). A learning constant of 0.15 and a hexagonal network architecture were used. At the beginning of the simulations the weights were randomly distributed (but normalized); the neighbourhood size was first set to 3, and after 100 and 200 training cycles reduced to 2 and 1, respectively, in order to obtain a reasonably well-trained network

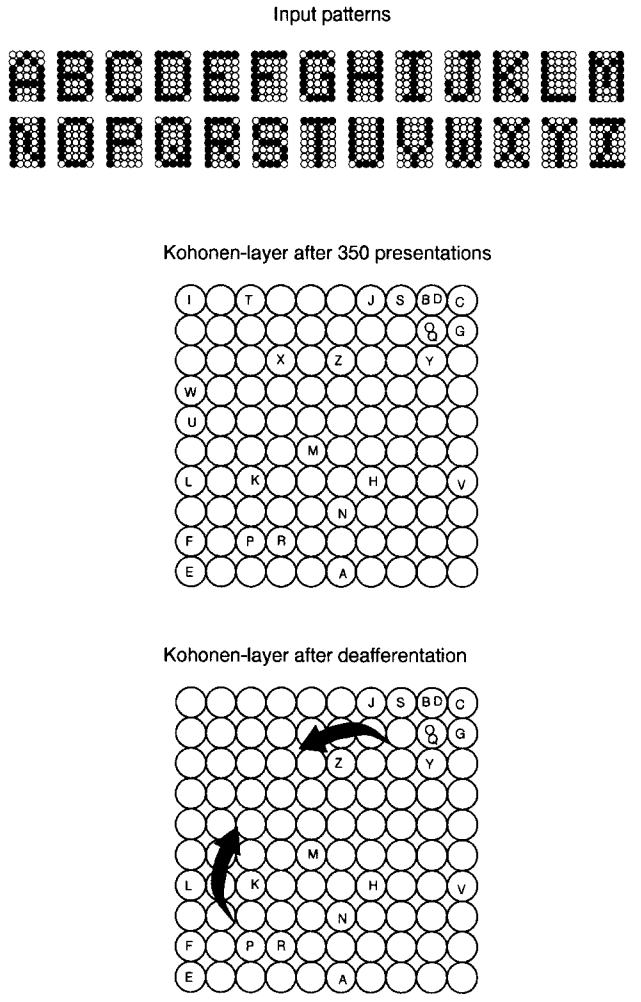

Fig. 3. Input patterns (*top*), trained Kohonen layer (*middle*) and effects of simulated deafferentation (*bottom*) in the simulated network model. Black circles represent active neurons at the top. In both Kohonen networks shown, *characters on circles* indicate winning neurons, i.e. neurons that represent the respective input patterns. *Arrows* (*bottom*) indicate the direction of expected and – in the presence of input noise – observed 'invasion' of winning neurons into the deafferented part of the network

the network. As all input signals were scaled in the range of 0.0 to 1.0, possible noise levels fell into this range. Moreover, the addition of noise to each input signal was thresholded such that all input values remained in the range of 0.0 to 1.0. To give an example: under the condition of a noise level of 10%, random values between -0.1 and 0.1 were added to each of the 35 numbers (originally 0 or 1) of the 5×7 input pattern (cf. Caudill and Butler 1992, p. 20). This manipulation effectively degraded the clarity of any given input pattern and, with increasing noise, allowed it to 'resemble' other input patterns.

Under the condition of increasing noise levels, the space on the map left empty by the removal of the respective input patterns was quickly 'invaded' by representations of patterns of the restricted input. In other

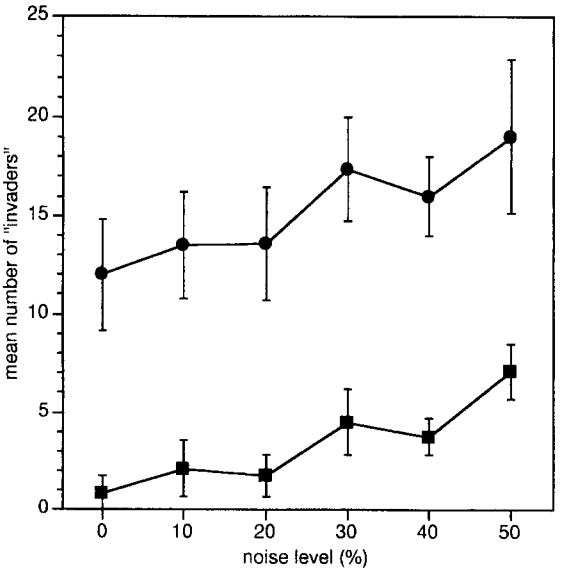

Fig. 4. Number of invading winning neurons (mean and SEM) during 10 training cycles dependent upon noise level and neighbourhood size

words, neurons that used to become activated by a pattern which was no longer present as input now became activated by a pattern which used to be coded by a neuron in the non-deafferented area.

In order to quantify these observations, simulations were run with different levels of noise and different neighbourhood sizes. Deafferentation was simulated in two similarly trained networks in which different random starting conditions had resulted in a different distribution of input patterns across the feature map. As the dependent variable, the frequency of winning neurons falling into the deafferented area was recorded. For each noise level and neighbourhood size, eight simulations were run, starting from the two different networks. In each network, deafferentation was simulated for each of the four quadrants separately.

Figure 4 presents the mean number of 'invading' representations at different noise levels and neighbourhood sizes during 10 cycles of input presentation (i.e. each pattern of the restricted input set was present 10 times as input). A two-factor analysis of variance (ANOVA) with neighbourhood size and noise level as variables revealed a significant effect of neighbourhood size ($F(1/84) = 87.3$, $P < 0.0001$) and noise level ($F(5/84) = 2.7$, $P = 0.027$), while there was no interaction between the two factors. The increasing number of invading representations with increasing noise levels was highly significant, as validated by contrast analysis (linear trend, $F\ (1/90) = 5.96$, $P = 0.017$). As can be seen from Fig. 4, in a well-trained network with clear coherent predictable input and a steep activation function (i.e. a neighbourhood size of 1) and well-established weights, the system appears to be rather stable with only a small tendency for any redistribution of representations. Increasing levels of noise, however, produce an increasing number of invading

neurons at both simulated neighbourhood sizes.[2] Figure 4 also demonstrates that while redistribution of representations depends upon neighbourhood size, i.e. upon the activation function of the winning neuron, the effect of noise is additive.[3]

In order to investigate further the nature of the noise-driven 'invasions' of winning neurons activated by input patterns that used to be represented within the non-deafferented areas, we analysed the previous coordinates of the representations that 'invaded'. This was based upon the following line of reasoning: because of the self-organizing feature of the Kohonen layer, similar patterns are coded by neurons that are close together. Hence, a somewhat disorted version of, for example, the input pattern 'O' is more likely to activate (if not the 'O' neuron) a neuron coding for somewhat similar input ('Q', for example) located in the neighbourhood of the 'O' neuron. Therefore, 'invaders' should move from areas adjacent to the deafferented area rather than 'jump' in from distant areas. If the latter were the case, one might argue that what we call 'invasion' is in fact random activation of all neurons of the Kohonen layer by the noise.

As Fig. 5 indicates, this is not the case, i.e. invading representations are more likely to come from nearby sites rather than 'jump' in from distant locations. The distance effect on the frequency of invading representations was highly significant (linear trend, $F(1/35) = 12.9$; $P < 0.001$). In other words, neurons of the deafferented layer become increasingly activated with increasing noise levels. In particular, input patterns similar to those no longer present (and therefore represented by neurons relatively close to the deafferented area) lead to such activation.

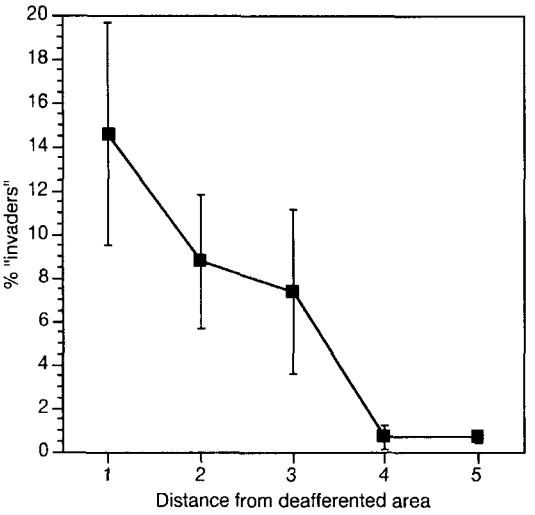

Fig. 5. Origin of the 'invading' representations. In a 10×10 network with one quadrant lesioned, the remaining neurons are 1–5 steps (i.e. changes in the x and/or y coordinates) apart from the deafferented area. For each of the light quadrants of the two networks, the representations in the non-deafferented part of the network were grouped according to their distance from the deafferented part into five categories. As the number of invaders from each of these five categories depends upon the absolute number of neurons in that category, the percentage of invaders in each group was determined. Means and standard errors of the percentage scores of 'invaders' are displayed as dependent upon the distance of the invading representation from the deafferented part of the network.

6 Discussion: model and reality

We have simulated the effects of deafferentation in a simple Kohonen network. Multiple simulation runs demonstrated that redistribution of representations within the network is facilitated by input noise, the effect being additive on the effect of increasing neighbourhood size. Moreover, the redistribution is characterized by the 'invasion' of representations from areas adjacent to the deafferented network area.

From these observations of simulated deafferentation, the following model of phantom limb causation may be derived. The noise-driven activation of neurons in the deafferented area (which used to code input patterns which no longer exist) by somewhat similar input patterns corresponds to the activation of sensory neurons coding for the lost limb by input to sensory neurons coding for other parts of the body (but lying adjacent to the deafferented area). This activation is the objective correlate of the subjective experience of phantom sensations. In the absence of input noise (in paraplegic patients), the non-altered sensor input does not reach the deafferented sensory cortex, and no activation of its neurons occurs. In sum, it is the presence of input noise generated 'peripherally' that drives rapid central cortical reorganization of sensory representations in amputees.

In this discussion, we first address the biological plausibility of the model. Its virtues in providing a parsimonious account of a number of clinical and experimental phenomena are then discussed, and alternative hypotheses, experiments as well as clinical implications are considered. Finally, general remarks on noise in neuronal networks and on the epistemic status of simulation experiments follow.

Biological plausibility

This model rests on a number of assumptions which we think are biologically plausible.

(1) Its most important presupposition – that the cortex can be modelled by self-organizing feature maps – will not be commented upon, since it has already been extensively discussed (cf. Kohonen 1989; Ritter 1991).

[2] We further noticed that while increasing noise levels lead to instant activation of the deafferented neurons, these 'invading representations' often were not stable, in particular at higher noise levels and during the training cycles that directly followed deafferentation. Under such circumstances, representations may wobble for quite a while before some degree of stabilization occurs. Finally, at high noise levels we observed wobbling representations within the non-deafferented area of the network (see Discussion)

[3] We experimented with larger neighbourhood sizes, but due the small size of our network, the results were less clear-cut. At a neighbourhood size of 3, this neighbourhood includes up to almost 50% of all neurons, and ceiling effects occur with respect to the possibilities for redistribution of representations. We hope to resolve this issue by running simulations on considerably larger networks (see Discussion)

(2) When limbs are amputated, the dorsal root ganglion cell as well as the pathways to the cortex remain intact. Moreover, it is known that lesions of the primary afferent fibres lead to spontaneous activity of the dorsal root cell (cf. Devor 1984). According to the experimental findings of Welk et al. (1990), random action potentials are generated by up to 30% of lesioned sensory neurons. Hence, the assumption of the presence of extra noise in the sensory input of amputated patients is biologically plausible. Needless to say, no such noise can reach the cortex in paraplegics since the necessary afferent fibres are lesioned.

(3) The model rests on a third assumption: in self-organizing feature maps each input neuron is connected to each neuron of the Kohonen layer. The degree and type of corresponding connectivity in the neocortex are highly complex. However, given that a single pyramidal cell receives up to ten thousand input connections, it is not so much the high connectedness, but rather the presence of unused connections – presupposed by the model – which has to be addressed. It is important to keep in mind that for input representations to move from one neuron to another, there have to be connections between the neurons representing the input activity and several neurons of the output (Kohonen) layer, and as only one of these output neurons is activated, there have to be unused connections. Generally speaking, models of cortical plasticity appear to presuppose the existence of connections which are not used (cf. Anderson et al. 1990, p. 297). Is there evidence for such a seemingly superfluous arrangement?

From an evolutionary point of view, it must be mentioned that the very gain of plasticity is quite likely to be enough to justify the existence of extra connections. In other words, animals may have evolved to keep a certain proportion of unused input fibres (and not destroy them by pruning mechanisms; cf. O'Leary 1992). They thereby retain the capability for experience-dependent neural plasticity. In addition to these theoretical considerations, over the past decade empirical evidence has accumulated for the existence of 'silent' connections in the central nervous system that can be 'unmasked' by changes of the spatiotemporal input characteristics (cf. Bach y Rita 1990). Such 'functional' changes occur within minutes and involve spatial redistributions of cortical representations (shifts of receptive fields) of up to 2 mm. Within the superficial layer of the cortex (layer I), horizontal connections span 6–8 mm and display use-dependent changes in synaptic strength. They have been implicated in long-term spatial changes of cortical representations of more than 10 mm (Gilbert 1993). In addition to the process of unmasking, rapid sprouting of new axons in response to input changes has been demonstrated in the visual cortex (Antonini and Stryker 1993). Such remodelling of axonal arbors occurs within a few days and appears to blurr the boundary between functional/physiological and anatomical change. In sum, recent research has provided ample evidence for the existence of the surplus connectivity within the cortex that is needed for neuroplastic change and presupposed by the modelling of such change by feature-map-like network models.

Parsimonious accounting for clinical and experimental observations

Our model of reorganization of representations in a Kohonen network after deafferentation is not only biologically plausible, it also provides a parsimonious account for clinical and experimental observations which otherwise are unrelated or lack an explanation.

(1) Phantom limbs are experienced immediately after amputation. This may be explained by the fact that the very mechanism of amputation produces 'sensory input noise' (i.e. random activity of the dorsal root ganglion sensory neurons) which leads to some random activation in the deafferented area. Subjectively, this corresponds to tingling and itching sensations in the no longer existing limb immediately after amputation.

(2) It is known that compared to planned amputations, amputations after traumatic events are more likely to and, indeed, more rapidly produce phantom sensations. This can easily be explained as being caused by the extra incoherent input (i.e. noise) provided by the traumatic event and the time thereafter until the operation is carried out.

(3) Stimulation of body surfaces corresponding to cortical areas adjacent to the deafferented area gives rise to 'referred sensations' in the phantom limb. According to our model, the extra noise provided by the randomly firing sensory neuron leads to the activation of cortical cells within the deafferented area. This happens almost immediately after amputation. It is important to note that the representations 'invade' from areas adjacent to the deafferented area.

(4) The somatotopical organization of the referred sensations corresponds to the fact that the redistribution in feature maps is governed by the internal structure of the map.

(5) Telescoping and shrinking of the phantom is likely to correspond to the decrease of the size of the cortical area that codes for the amputated limb. These processes occur on a larger time scale and must be accompanied by neurons of the deafferented area 'unlearning' previous representations. It is likely that this process is facilitated by input noise.

(6) According to the model, amputation leads to an unused cortical area, which is taken over by adjacent areas. In other words, the deafferented cortical surface takes up computational functions of adjacent areas, which thereby increase their computational capacity. This increased capacity can be demonstrated: the receptive fields of sensory neurons in the stump become smaller, i.e. processing of sensory information from stump sensory neurons is improved as a result of the increased cortical computational surface.

(7) When the simulations were run with rather high noise levels, we observed some wobbling of the representations of input located within the non-deafferented areas: a given input no longer at every presentation led to the activation of its representing neuron. Instead, the added noise made the input activate neurons that used to code other input patterns. This led us to the prediction that there should be clinical cases where

similar 'misplacements' of sensory input might occur, caused by processes that can be expected to lead to extra noise. Such extra noise may be expected after particularly severe injuries and/or additional injuries. In such patients, touching an arm, for example, might result in the sensation of touch in the (non-amputated) leg. A search through the older (usually more descriptive) literature revealed that such cases exist. In his monograph, Cronholm (1951) described a patient with multiple fractures that led to amputation 4 months after the injury, and another patient with one leg amputated and the other also injured. Both patients reported referred sensations of the kind just described: when parts of their bodies were touched, they felt the touch at that location and, in addition, at another location of the body (but not in the phantom). In both cases, a high sensory noise level can be expected, caused in the first case, by the severity of the injury and the delay of the amputation and, in the second case, by the additional injury. In the light of our model, these clinical observations find a straightforward explanation. In our view, it is not unlikely that such experiential phenomena may occur more often but are rarely scrutinized by the physician.

(8) Lastly, the model does not rule out some influence of higher order cortical maps (coding meaningful items or events) on the redistribution of the cortical representations of sensory input. The existence of this important mechanism has been demonstrated in deaf humans who received a cochlea implant. This device consists of a transducer that converts sound waves into electrical pulses. Of course, it is not possible to mimic the pattern of action potentials generated by the cochlea, but some temporally coherent code is fed by the device directly to the acoustic nerve. Immediately after this procedure the patients are unable to make any use of the signals. They hear unintelligible 'babble' when somebody is speaking. However, after several months of adaptation, in some of these patients, reorganization of auditory cortical areas takes place such that the impulses from the cochlea are reorganized as the different phonemes produced by a speaking voice. Some patients even pass the ultimate test of engaging in a telephone conversation (lip reading is not possible under such circumstances). It is important to realize that in these patients the representation of phonologically coded words must have been 'ordered' in some way by semantic information, i.e. the information about the meaning of the phonological patterns. Hence, higher order semantic maps must have influenced the development of lower order phonological maps. It should be mentioned that the general map-like structure of the auditory cortex has been demonstrated in bats (Suga and Jen 1976; quoted in Ritter 1991) and in primates (Recanzone et al. 1993). The map found in bats has been simulated using a self-organizing feature map network (Ritter 1991). Recently, Miikkulainen (1993) has provided a theoretical framework for multiple layers of self-organizing feature maps coding for various aspects of language. Along similar lines, higher order cortical maps may influence representational redistribution processes in amputees and hence allow for the influence of 'psychological variables' on the phantom experience.

The proposed model cannot account for reorganization processes after cortical damage (see Sutton et al. 1994), and it does not explain the (extremely rare) cases of phantoms in patients with birth defects (cf. Weinstein et al. 1964). However, it belongs to the small but growing number of instances where the presence of noise in nervous systems appears to be an important part of its function (in this case, cortical reorganization; cf. Douglas et al. 1993; Maddox 1994). In this respect, it bears some resemblance to the view of Changeux and Edelman of neural plasticity, which also relies upon the existence of random variability in neural signalling in order to enable neural group selection mechanisms to work (cf. Changeux et al. 1984; Edelman and Finkel 1984). Our computational model, however, is much smaller in scope than the Darwinian selectionist approach to neuroplasticity in that it only demonstrates a single principle of neural reorganization. Moreover, we focus on the genesis of a particular kind of subjective experience, which often occurs immediately after deafferentation, rather than on the neural mechanisms that are involved in microneuroanatomic change.

Alternative hypotheses, clinical implications, and further experiments

Activation patterns of single cortical neurons in animals before and after amputation could be studied experimentally. Therefore, it should be possible to test our model and compare it with other hypotheses about the generation of phantom sensations. In particular, as Katz (1992) in his thoughtful review has proposed a peripheral mechanism involving a loop of sympathic-efferent and somatic afferent fibres, it should be possible to generate differential predictions for the two models. Contrary to our model, the model of Katz states that specific sympathetic input drives the generation of phantoms.

Moreover, as animal behavioural models of phantom limb experiences are described in the literature, strategies to prevent phantoms might ultimately be feasible. As the slow cortical redistribution processes that presumably occur in paraplegics do not (or only to a small degree) result in phantom sensations, reduction of input noise by intranerval injection of agents that either reduce the activity of dorsal root sensory neurons or even destroy them might prevent the development of phantoms. Phantom limbs result in considerable discomfort for the patient, varying from the experience of odd sensations to almost unbearable pain. Hence, effective strategies for the prevention of phantom limbs are needed. Once the basic mechanism has been found and an animal model has been developed, such therapeutic strategies come within reach – at least in cases of elective amputations (for example, in patients with bone tumors).

The present study is preliminary in nature. Limitations of the size of the network imposed by hardware and software inevitably left a number of questions open:

1. What is the time course of change in cortical representations, and how does noise influence the development of lasting changes in the distribution of representations?

2. Is there an optimal neighbourhood size for such changes to occur? (With only two levels for neighbourhood size an interaction cannot be completely ruled out.) 3. How do the data from larger scale simulations correspond to clinical observations? (A detailed prospective clinical study of cases with elective amputations, informed by the results of simulations, should be planned.) The first step to answer these questions is, of course, running simulations on larger networks.

General considerations

We want to conclude with a systematic and an epistemic note on noise in neuronal networks and on the scientific status of simulation experiments. Neural network models are often introduced as a solution to the 'hundred step problem' of programs that supposedly are run by the brain (cf. Rumelhart 1989, p. 135): human beings and primates can do many complex tasks within a few hundred milliseconds. Programming these tasks algorithmically would involve thousands of steps. As the fastest neurons in the primate cortex have switching times of 1 to 5 ms, however, the algorithms used by the brain to accomplish such tasks can only involve – at the most – about 100 steps. Hence, so the argument concludes, brains have to use the highly parallel architecture realized in neuronal network models.

In the context of the present study it is important to note that real neurons are not only at least 10^4 times slower than silicon switching devices (such as the chips used in personal computers) but also at least 10^9 times less accurate (see von Neumann 1958). It is therefore highly unlikely that the presence of 10^9 times more noise in brains compared with computers will not have a strong influence on the way in which information processing evolved in living creatures. To put the argument more strongly, it appears inevitable that brains evolved not to fight noise (with error-correction algorithms, which could only be implemented by error-prone hardware), but rather to use it, i.e. to 'build it into the system'. If this is the case, the 'signal-to-noise' ratio becomes an important general variable of information processing in living brains which has to be actively controlled and adjusted to the different needs of the organism. There is biological and psychological evidence that some neuromodulators may in fact have the function of controlling this crucial ratio (cf. Cohen and Servan-Schreiber 1992; Morrison and Hof 1992; Spitzer et al. 1993), and that learning can be impaired by a decrease of the cortical noise level. From this perspective, further investigations of the effects of noise on simulated networks as well as on the functions of biological systems appear warranted.

Finally, the epistemic status of simulations such as the one reported above should be briefly discussed. Two opponent views can be distinguished. (1) The view proposed by many neural network researchers and supported by an accumulating number of studies bridging the gap between physiology and neural computation (cf. Churchland et al. 1990; Shepherd 1990) takes these networks as a research tool. Like other empirical research tools, it is assumed that networks provide data that are sufficiently detailed and clear to be used in the testing of conflicting hypotheses (cf. Kosslyn and König 1992). (2) According to the sceptic view, however, neural network models cannot prove or disprove any hypotheses, but rather merely provide 'demonstrations' (cf. Crick 1988, 1989) or existential proofs, i.e. proofs that some model actually 'works' in a particular way. It is important to realize that this sceptic view is sufficient for the interpretation of our proposed model presented in this paper. We do not claim to have tested conflicting hypotheses; instead, we merely wanted to demonstrate the existence of a specific type of network solution for a particular set of propositions. In other words, we do not claim that our rather small-scale simulation proves that phantoms are caused by cortical reorganization processes due to noise generated in the spinal cord. We rather merely suggest the existence of a computational mechanism for redistribution of representations in Kohonen-type feature maps, and we draw parallels between the computational model and real world data. The fact that the model provides a parsimonious account for a number of clinical and experimental observations does not count as a proof. However, the model suggests further strategies for research which may lead to more detailed simulations. Such simulations in turn may provide clinicians with clues for further hypotheses that can be subjected to empirical tests in patients. Hence, even the sceptic has to acknowledge the 'heuristic value' of simulated neuronal activity, and the detailed study of such simulations is likely to prove the sceptic wrong.

Acknowledgements. We want to thank V.S. Ramachandran, M. Posner and an anonymous reviewer for helpful comments on a previous version of this paper. This work was supported by a grant from the Deutsche Forschungsgemeinschaft DFG (Project No. Sp 364/1-2) to the first author.

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