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A Model-based Interpretation of Phantom Pain
—*Conservative Body Schema and Flexible Somatotopy*—

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Some patients with amputations are known to still experience presence of the amputated limb (phantom limb). Phantom limb is found to be accompanied by reorganizations of somatotopic representations at cortical and subcortical levels due to deafferentation, which also discloses presence of body schema, i.e., integrated image of body. In addition, phantom limb is often recognized with painful sensation, i.e., phantom pain, which is suggested to be caused by abnormally high activations in thalamocortical neurons in the deafferentated area. In this paper, simulations using a self-organizing neural network model were performed to clarify contribution of axonal sprouting and body schema to reorganization of cortical representation, and to explore a possible mechanism underlying abnormally high thalamocortical activation. The results showed that axonal sprouting could cause reorganization of cortical mapping with an aid of facilitatory inputs referring to the conserved body schema even after deafferentation. In addition, removing thalamic recurrent inhibitions was shown to induce high and sustained activations in thalamocortical neurons in the deafferentated area. Consequently, this study suggested that discordance between the reorganized cortical representation and physiological process referring to the body schema could give rise to phantom pain. Phantom pain is a clue disclosing how body schema is constructed and maintained, which might be an important subject also for robotics dealing with an artificial body.

KEYWORDS: phantom pain, body schema, reorganization, self-organizing network, somatosensory cortex

1 Introduction

How does human know that parts of her/his body belong to her/him and she/he can move them voluntarily? Why is a touch on her/his body felt as the touch just on her/his body, not on the other's? These questions may sound nonsense. However, there do exist special situations under which these questions emerge with actuality. Phantom limb, the subject of our study, is one of such special situations.

Some patients with amputations are known to still experience the presence of the amputated limb (phantom limb). Phantom limbs are often painful. 80% or more of amputees were reported to suffer from the phantom limb [1]. There have been clinical and physiological studies for relieving the pain and exploring the underlying mechanisms [2].

So far, cortical and non-cortical mechanisms have been proposed for phantom limb [3]. The cortical mechanisms are believed to involve reorganization of somatotopic mapping after amputation[1]. That is, cortical area used to represent the deafferentated part of the body was observed to be invaded by representations of the intact parts [6, 7]. These findings led to hypothesis that neuronal responses in the invaded area to afferent inputs from the intact parts of the body might cause phantom sensation [3, 6]. Otherwise, non-cortical mechanisms include free nerve endings, neuromas and spontaneous activations in spinal cells which represent the amputated part. However, phantom sensation was known to be evoked or augmented by stimulating the intact part of the body [8], which suggests that the non-cortical mechanisms are not only the possibility.

So far, reorganization of cortical mapping has been simulated by self-organizing neural network models [3, 9, 10]. Although they are differently structured in detail, principally these models can be categorized as a reaction-diffusion system, in which difference in diffusion constants of activator and inhibitor helps autonomous pattern formations [11]. In a neural system, activator and inhibitor correspond to excitatory and inhibitory connections, respectively; formed patterns correspond to self-organized neuron groups in each of which neurons are strongly coupled each other via excitatory connections. Spatial distribution of thus organized neuron groups constructs a cortical topographic mapping. Note that in such a system, once patterns are formed, they are quite robust against any perturbations. Therefore, additional mechanisms are required for the formerly organized mapping to be reorganized. Extensively divergent afferent inputs as Pearson et al. assumed could reorganize somatotopic mapping after deafferentation through neuron's competitive input selection, because in their case each neuron had inputs from all over the hand surface, glabrous and dorsal [10]. However, such a totally divergent afferent

[1] short-term and long-term changes in cortical and subcortical response properties are known to result after amputations [4, 5]. In this paper, the long-term change possibly accompanied by anatomical reconstruction is studied.

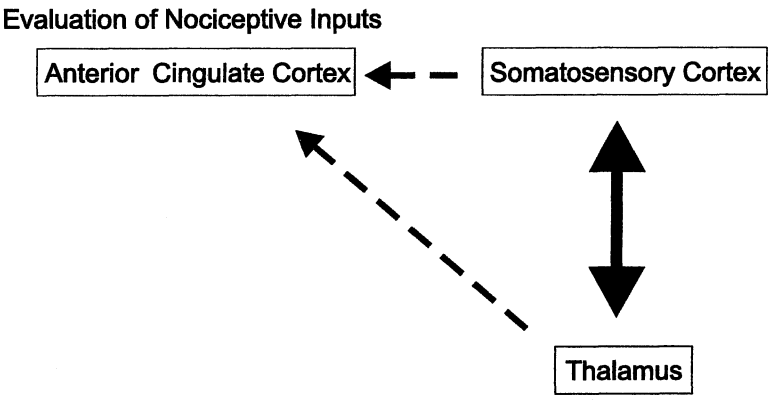

Fig. 1 Neural processing of pain sensation.

projection is not found for all the cortical somatotopical representations [12]. On the other hand, with a restricted degree of divergence in afferent projection, Xing and Gernstein reported that there were some difficulties in simulating reversion of reorganized connections and boundary shift of representation after deafferentation [13]. Extended cortico-cortical connections found in a large-scale reorganization and axonal sproutings might emerge to reconstruct robust cortical mapping [14, 15, 16].

Actually, phantom pain could not be elucidated only by reorganization of cortical somatotopic map. Mechanisms deceiving somatosensory system and inducing pain sensation with phantom limb should be understood. The deceiving mechanism is considered to involve an integrated image of the body, which is referred to as "body schema" or "body image" [17, 18]. How the body schema affects somatosensory process is discussed in the next section. Exploration for the mechanism inducing pain sensation with phantom limb should be done in the light of cognitive process of pain. Recent fMRI and PET studies clarify that the anterior cingulate cortex, categorized as the limbic system, is activated during pain sensation in addition to the thalamus and somatosensory cortex, which differentiates brain response to the painful stimuli from the non-painful [19, 20]. These findings suggest that localization and evaluation of painful stimuli are separately performed in the somatosensory cortex and the cingulate cortex. Because anatomically, the anterior cingulate cortex is known to receive dense axonal projections from the thalamic nuclei [21, 22], activations of thalamic neurons are supposed to be necessary for pain sensation (see Fig. 1). This coincides with that central pain is associated with abnormally high activities in thalamic neurons [23, 24].

In this paper, simulation studies are carried out to clarify the following three points. (1) how axonal sprouting affects the reorganization of topographical mapping. (2) how the body schema can be modeled at physiological level and is involved in reorganization of topographical mapping. (3) how abnormally high thalamocortical activation which might induce the pain sensation is induced associated with the phantom limb. In order to achieve this, a self-organizing neural network model is developed, which faithfully mimics the anatomical structure of somatosensory pathway. Through the simulations, a possible fundamental mechanism underlying the phantom pain is suggested.

2 Body schema and phantom limb

As mentioned before, reorganization of cortical map caused by deafferentation is not sufficient for inducing phantom limb, because the invasion of representation simply implies that cortical neurons in the invaded area come to represent the intact limb stump. Provided that there exists a cognitive process which localizes the afferent inputs still referring to an old image of body structure before amputation, this process would mis-localize sensory inputs as from the intact region. Presence of such an image has been noticed, which is referred to as "body schema" or "body image" [17, 18]. That is, discordance between the body schema and the reorganized cortical mapping is supposed to be involved in phantom limb sensation. Ramachandran found that painful phantom sensation was relieved, when the amputated hand was made visually reappear by mirroring the intact hand, and the amputee patient was instructed to move "both" hands [25]. This finding could be interpreted in various ways. Nevertheless, accordance between the body schema and the cortical representation might have been restored by his procedure.

The body schema is supposed to exist in the higher-order cortical area such as the parietal lobe [26, 27]. The schema is postulated to integrate motor intention (motor command), awareness of movement (proprioception), and visual feedback (visual information) [27]. Here, the question arises how information referring to the body schema affect somatosensory process. Iriki et al. found that neuronal activities in the somatosensory cortex of a

monkey were increased by attention guided by a visual cue [28]. This finding suggests that information referring to the body schema appears as facilitation to the cortical area representing the concerned part of the body. Therefore, if the body schema is conserved even after amputation, motor intention and attention would be oriented to the amputated part of the body, which are supposed to deliver facilitation to the deafferentated cortical area.

In the following simulations, considering somatic sensation referring to the body schema, facilitation is applied to coltical neurons in addition to afferent stimulations for organizing neural connections.

3 Neural network model

3.1 Model structure

Figure 2 illustrates the model structure. The neural network model consists of 3 layers: input, thalamus, and cortex, each of which has 18 × 18 lattice nodes. The nodes in the cortical and thalamic layers correspond to neurons. The input layer consists of just input nodes. Dynamics of neuronal response implemented following Xing and Gerstein [9] is described in Appendix.

Connections between neurons are made roughly following an anatomical structure except for the cortical layer where each neuron makes both of excitatory and inhibitory synapses on target neurons: a pair of excitatory and inhibitory neurons are fused into a single neuron for simplicity. Afferent projections, input → thalamus and thalamus → cortex, are made to the neurons in the area of 3 × 3 nodes centering around an originating neuron. Corticothalamic connections from the cortex to the thalamus and lateral recurrent connections in the thalamic layer are made in a nearest neighbor basis, i.e., 8 neurons. The lateral recurrent inhibitory connections are implemented to mimic those mediated by thalamic reticular neurons [29]. As shown in Fig. 3A, within the cortical layer, excitatory axonal projections are distributed up to the 3rd neighbor centering an originating neuron, and inhibitory projections up to the 6th neighbor; for both types of projections there is no self-connection. Here, target neurons are randomly selected following density decaying associated with the distance from an originating

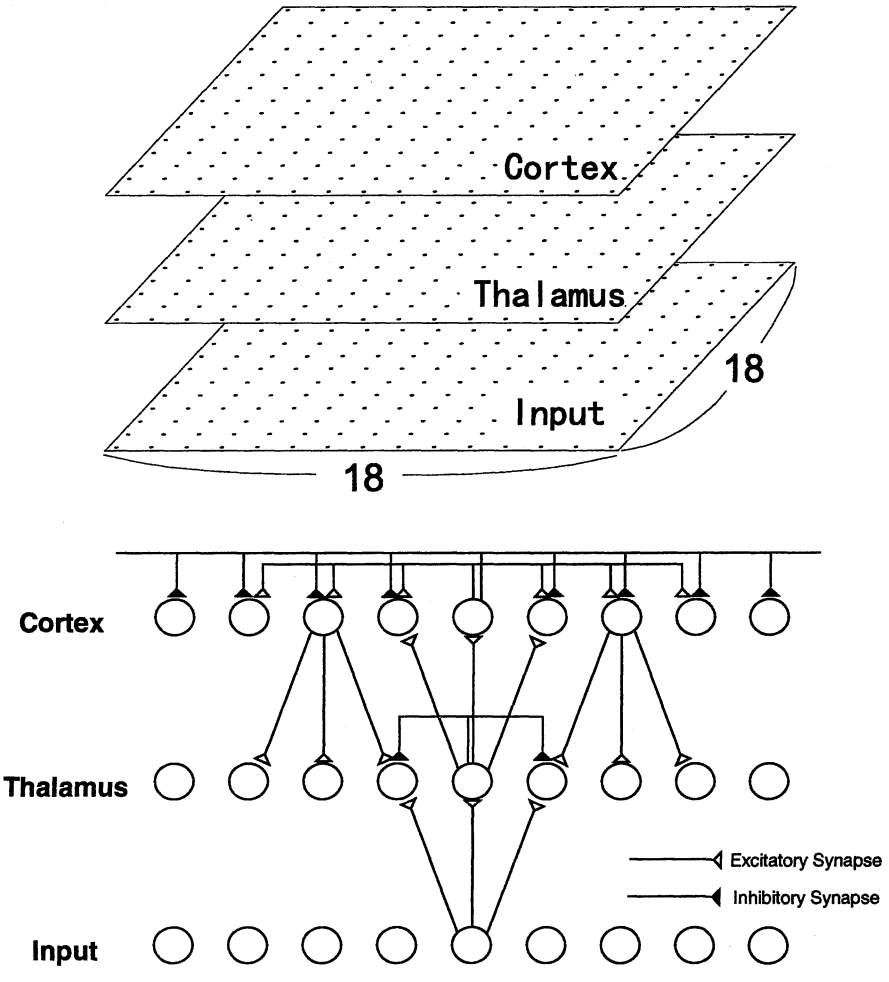

Fig. 2 Three-layered structure of the neural network model

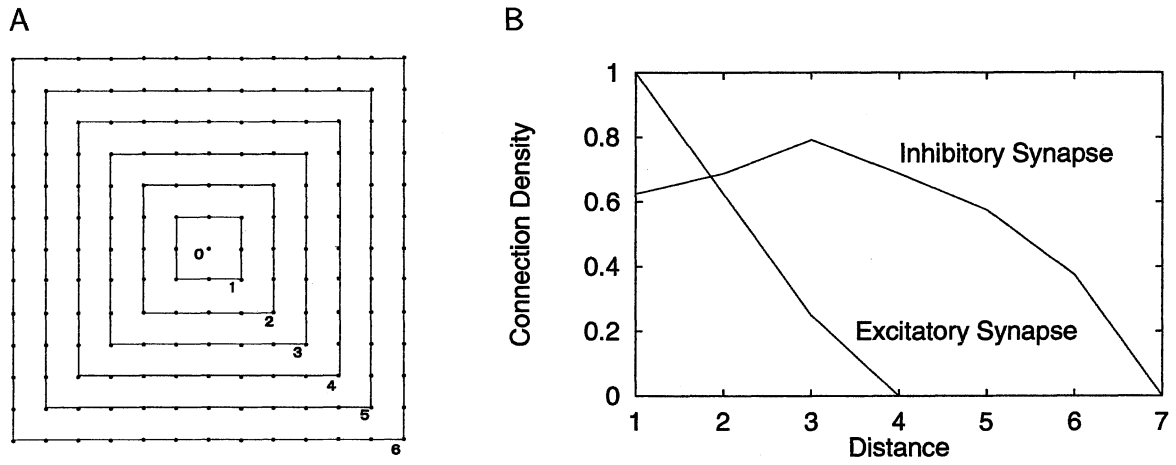

Fig. 3 **A** Distance between nodes **B** Connection density as a function of distance between nodes.

neuron as shown in Fig. 3B. These connections are made to satisfy a periodic boundary condition in order to avoid the edge effect. The corticothalamic and lateral inhibitory connections are assumed to have the delays of 8ms and 3ms, respectively, considering the number of synapses involved in each actual connection.

3.2 Network training

Network training is performed following the Hebbian and normalization rules [9]. That is, respective synaptic weights are incremented associated with the sum of EPSPs at the moment of firing of the postsynaptic neuron, and then the synaptic weights originating from the identical neuron are normalized in order to preserve the total amount of weights after the increments (for detail, see Appendix). This rule applies to the thalamocortical connections and the excitatory connections within the cortical layer. The total amounts of synaptic weights for a single neuron are 1 for the cortical excitation, 10 for the cortical inhibition, 1 for the thalamocortical connection, 5 for the corticothalamic connection, and 10 for the thalamic recurrent connection. Initial values of synaptic weights are given by uniform random numbers satisfying the predetermined total amounts.

Training of the network is done by repeating the following procedure. A training period consists of the initial stimulus-free 10ms, the stimulus 10ms and the following stimulus-free 40ms. The stimulus is given as an excitatory conductance pulse of 0.4. Then, all neuronal states are reset at the end of the period. Throughout the total period of training, receptors in the input layer are set to exhibit the background activities of 5Hz, where each of activity events is an excitatory conductance pulse of 0.775 with duration of 1ms. These parameters of background activities are selected so that neuronal activity in the cortical layer is approximately 1Hz. These rules almost follow Xing and Gerstein [9] except for the extended training period and plasticity of the thalamocortical connections.

4 Simulation results

4.1 Organization of somatotopic representation

Firstly, it is examined whether a boundary between somatotopic representations is constructed naturally by self-organization. The lattice is divided into two planes at the center, each of which is assumed to represent different somatotopic region, e.g., hand on the left half plane and face on the right.

Training is performed as follows. (1) A randomly selected plane, left or right, is stimulated successively 10 times. Input nodes in 3×3 area is stimulated simultaneously. If the stimulated area covers the center, the area is truncated at the center to obey the somatotopy. The procedure for changing synaptic weights described in Sec. 3.2 is applied every time stimulated. (2) The training (1) is repeated until the stimulation covers the whole plane. (3) The training cycle (2) is repeated 30 times. The resulting connections in the cortical layer do not show any distinct boundary between the planes (the result is omitted here). There are thick connections crossing over the center, and some neurons have the receptive fields spreading over the center. These results indicate that the self-organization here is not sufficient to construct a clear boundary of representation.

In order to implement the facilitation described in Sec. 2, a resting level of the firing threshold, H_0, of a cortical neuron in the stimulated plane is lowered from 10mV to 6mV above the resting membrane potential. As shown in Fig. 4, under this condition, there is no thick connections organized over the center between the planes. In addition, the organized receptive fields in the cortical layer scarcely spread beyond the center. Therefore, the

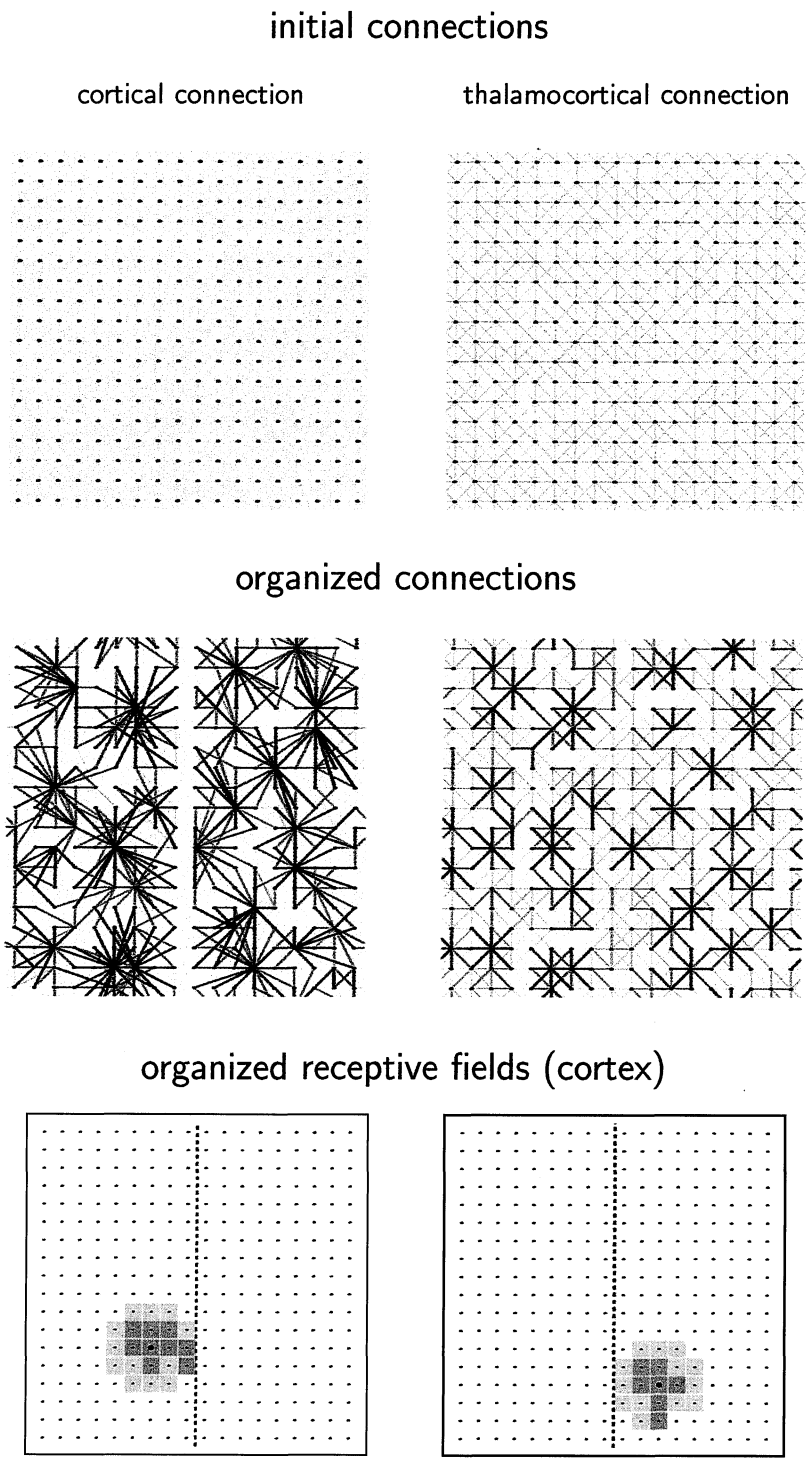

Fig. 4 Organized cortical and thalamocortical connections and typical receptive fields in the cortical layer separated by a boundary. In the top and middle panels, a thick line indicates a strong connection. In the bottom, a thicker color indicates a larger response. A dot in the center of the receptive field locates the neuron concerned.

boundary between the different somatotopic representations is shown to be successfully organized.

4.2 Reorganization of somatotopic representations

Next, reorganization of cortical mapping after deafferentation is simulated, which corresponds to reconstruction of the boundary in our model. A preliminary simulation using the trained network shown in the previous section is performed, which re-trains the whole network under the one-sided stimulation mimicing the deafferentation of the left plane. However, this shows no distinct reorganization of the boundary between the representations. This result demonstrates robustness of the constructed boundary even under the deafferentation. As men-

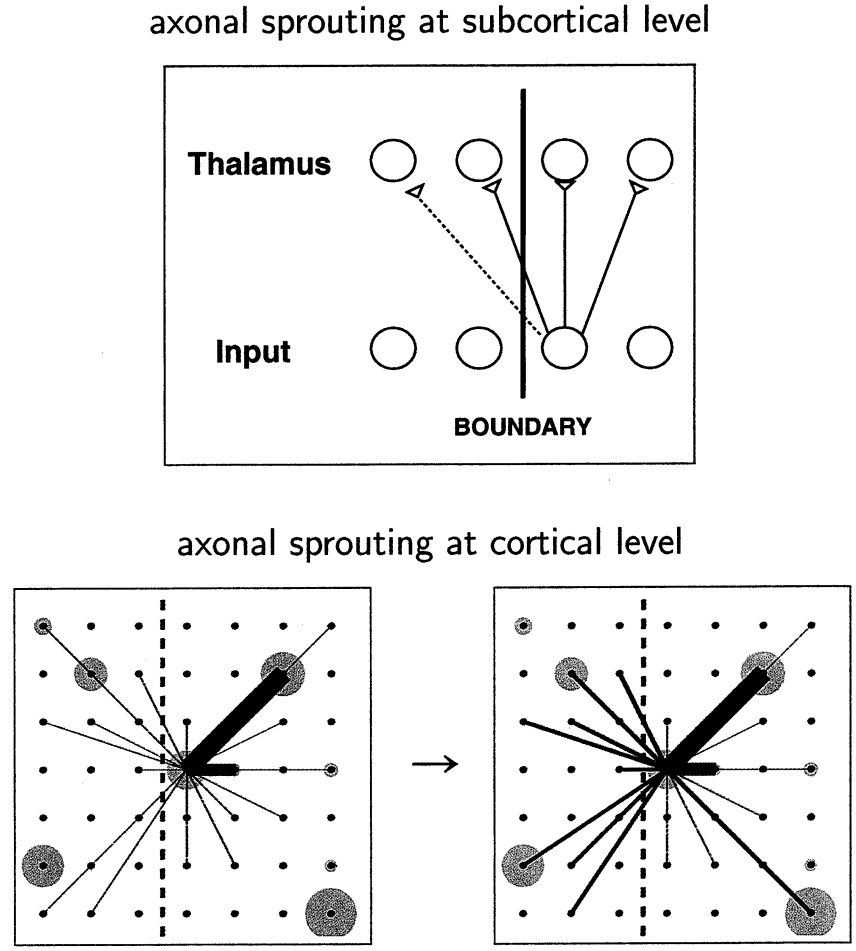

Fig. 5 Axonal sproutings in the subcortical (upper) and cortical neurons (lower). At the subcortical level 3 × 3 projection area of the receptor next to the boundary is extended to 4 × 3. At the cortical level,ineffective connections are strengthened and some connections are re-assigned to the responsive neurons which have non-negligible receptive helds,where a shaded circle indicates the extent of the receptive field.

tioned in the introduction, this robustness of formed patterns is considered to be inherent property for a self-organizing neural network.

Axonal sprouting may be one of mechanisms reorganizing the robust organized patterns, because the sprouting could shuffle spatial distributions of excitatory and inhibitory connections. In order to implement axonal sprouting from neurons in the intact region to those in the deafferentated area, ineffective synaptic weights from neurons in the right three columns next to the center to those in the left, (deafferentated side) are strengthened up to 10 times of the lower bound (see Fig. 5), where the ineffective synapses indicate that those weakened nearly to the lower bound due to the previous training. In our simulations, the sprouting is limited to the excitatory connections. In addition, considering the subcortical sprouting, the connections from receptors in the right nearest column to the center are allowed to be extended to the left by one column: the projection area of receptors becomes 4 × 3 in the thalamic layer as shown in Fig. 5.

The training is carried out for the whole network under the facilitatory bias on the left plane, while the stimulation is applied only to the right. The training results in emergent thick connections from the right to the left over the center as shown in Figs. 6 and 7. Consequently, some cortical neurons in the deafferentated plane are shown to have their receptive fields beyond the center which used to be the boundary between representations (Fig. 7). That is, the cortical neurons which suffer from deafferentation could respond to the sensory inputs from the intact region. In other words, the cortical representation for the intact region invades that for the deafferentated.

4.3 Abnormal activations in the thalamocortical loop

Here, dynamics of the reorganized network with sprouting are studied. Figure 8 shows a response of the reorganized network to the single stimulation applied to the intact region. Because this response disappears quickly, there appears to be no room for abnormally high activations. Such a stable response is considered to be due to

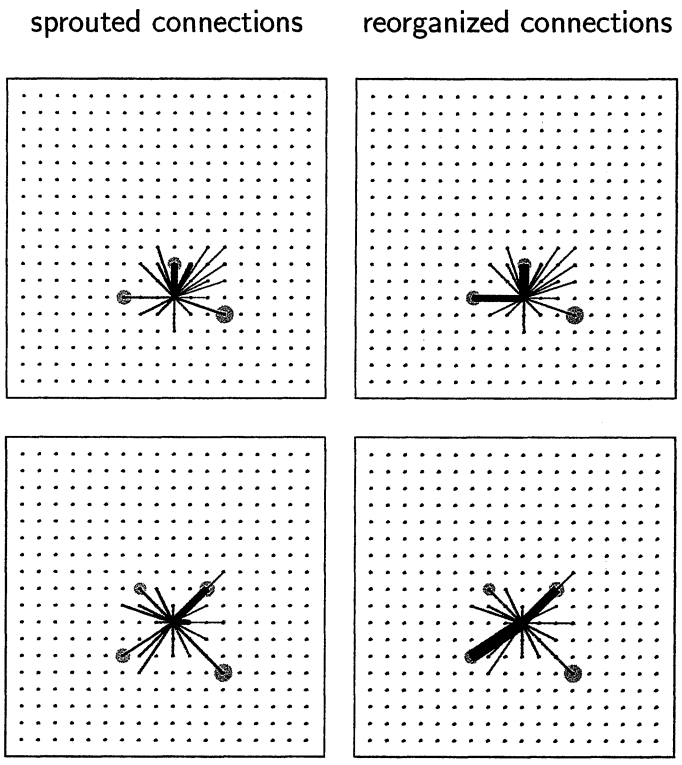

Fig. 6 Reorganized connections beyond the center by the axonal sprouting.Here,a thick line indicates a strong connection,and a shaded circle denotes the extent of receptive field of the connected neuron.

inhibitory systems included in the model i.e., cortical and thalamic inhibitions. In order to examine contributions of both inhibitory systems, cortical and thalamic responses are simulated by removing the respective inhibitory effects. Removing the cortical inhibition in the left plane results in almost the same response as the intact one shown above (the result is omitted). Figure 9 shows a response under no thalamic recurrent inhibition in the left plane. This response is initially evoked in the right plane, which is propagated to the left and sustained at an extremely high level. The results here suggest that weakened thalamic recurrent inhibition in the deafferentated area could induce abnormally high activation through the thalamocortical-corticothalamic loop.

5 Discussion

Here, the mechanism underlying phantom pain was explored by the simulations using the self-organizing neural network model. The results are summarized as follows. (1) The axonal sprouting facilitated reorganization of boundary in the cortical mapping, which was not realized merely by training under the deafferentated condition. (2) Physiological process referring to the body schema was simulated as facilitation to the cortical neurons, which contributed to organizing the firm boundary in cortical mapping, and cooperated with the sprouted connections in reorganizing the boundary. (3) With abolition of thalamic recurrent inhibition, abnormally high activation was sustained in the thalamocortical circuits representing the deafferentated region, which was not induced by removing the cortical inhibitions. Through these simulations, a possible mechanism underlying Phantom pain is suggested as follows. Physiological process referring to the conserved body schema even after amputation and axonal sprouting cooperate to reorganize the cortical mapping. The thalamic recurrent inhibition in the deafferentated area undergoes degradation associated with reorganizing process, which makes the thalamocortical network hyper active. Induced abnormally high activation in the deafferentated thalamocortical area is recognized as phantom pain due to conservation of the body schema.

The concentrically distributed excitation and wide-spread inhibition in the neural network model were shown to subserve robust self-organization of neuron groups in each of which neurons were tightly coupled by excitatory connections. Such a difference in the spatial distributions of cortical excitatory and inhibitory connections roughly reflects those in the neocortex [30]. As mentioned previously, this set-up of activator and inhibitor distributions is general one for pattern formation in a reaction-diffusion system [11]. The robustness of formed patterns was also found in the simulation study by Xing and Gernstein [13]. With a restricted degree of divergence in afferent projection, they reported that the reorganized connections could not be fully reversed, and that boundary shift of cortical mapping after deafferentation needed training with more intensive stimulation than

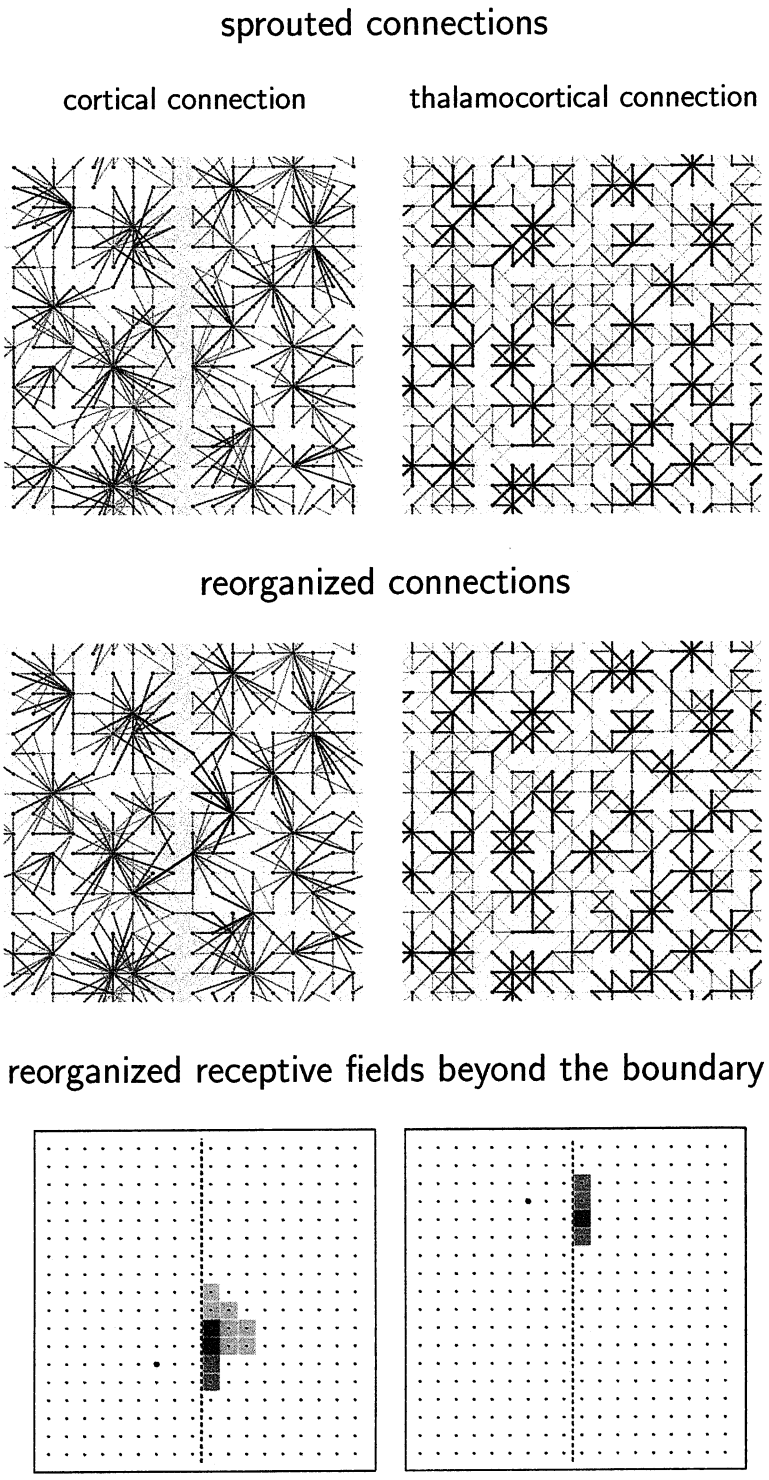

Fig. 7 Reorganized cortical and thalamocortical connections by axonal sprouting and typical receptive fields beyond the center in the cortical layer. In the top and middle panels, a thick line indicates a strong connection. In the bottom, a thicker color indicates a larger response. A large dot in the left plane locates the neuron concerned.

did the organization of cortical mapping [13]. If the cortical neurons receive extensively divergent afferent projections, robust mapping once organized could be easily reorganized by changing input arrangement, e.g. deafferentation [10]. This naturally leads to hypothesis that divergent projections from subcortical systems underlie reorganization of somatotopic map [4]. However, a large scale reorganization observed, for example, after arm amputation, seems not to be explained only by the divergent projections [16]. In such a case, axonal sprouting which potentially shuffles the spatial distributions of activator and inhibitor could be another possible mechanism [16, 27]. Our result in Sec. 4.2 suggest that extended axonal sprouting and divergent afferent projec-

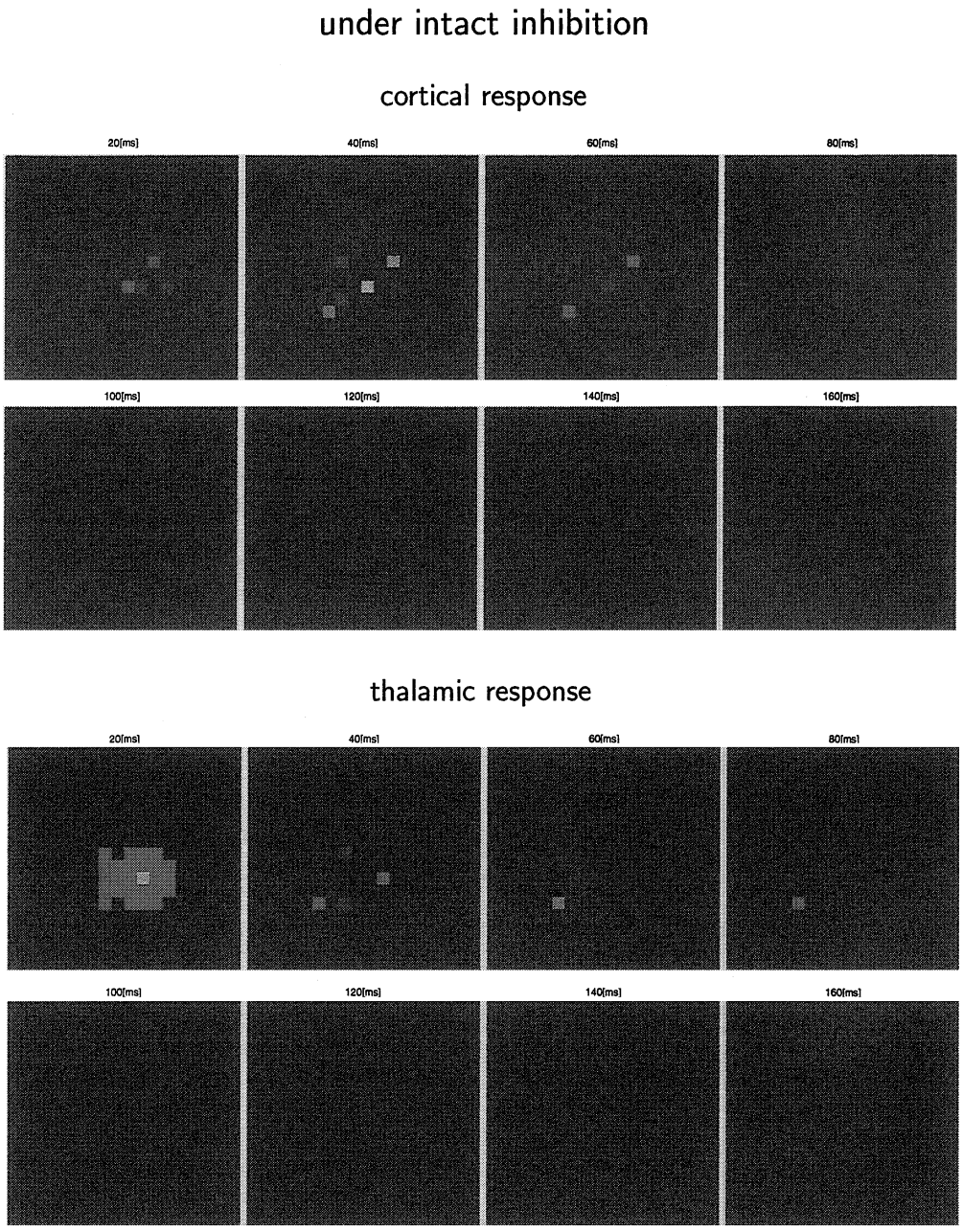

Fig. 8 Cortical and thalamic responses to the stimulus applied to the right plane (intact side) under intact inhibitions. A response increases in order of blue → green → yellow → orange → red.

tions are cooperatively involved in reorganizing the cortical mapping after amputation.

In our simulation, abolishment of the thalamic recurrent inhibition was found to sustain abnormal activations in the deafferentated thalamocortical area, which might underlie painful sensation in phantom limb. The thalamic recurrent inhibition was implemented to mimic a neurophysiological effect of thalamic reticular neurons, which are anatomically known to receive corticofugal projections [29]. Although direct physiological evidence is not known, activities of thalamic reticular neurons might be weakened through lack of corticofugal and afferent activations in the deafferentated area. Actually, such a disinhibition associated with reorganization of cortical mapping has been suggested for patients with pathological pain [23, 31]. Abnormal organization of cortical representation was observed in patients with repetitive strain injury and focal hand dystonia. Since they suffered from pathological pain, these diseases might be regarded as another case that discordance between the cortical representation and the body schema induces painful sensation [27]. In addition, patients with dystonia were found to have overexcitable motor cortex due to loss of inhibition [31]. Although its relation with changes in cortical sensory representation is unclear, motor and sensory overexcitations may share a common mechanism, which deserves further investigation.

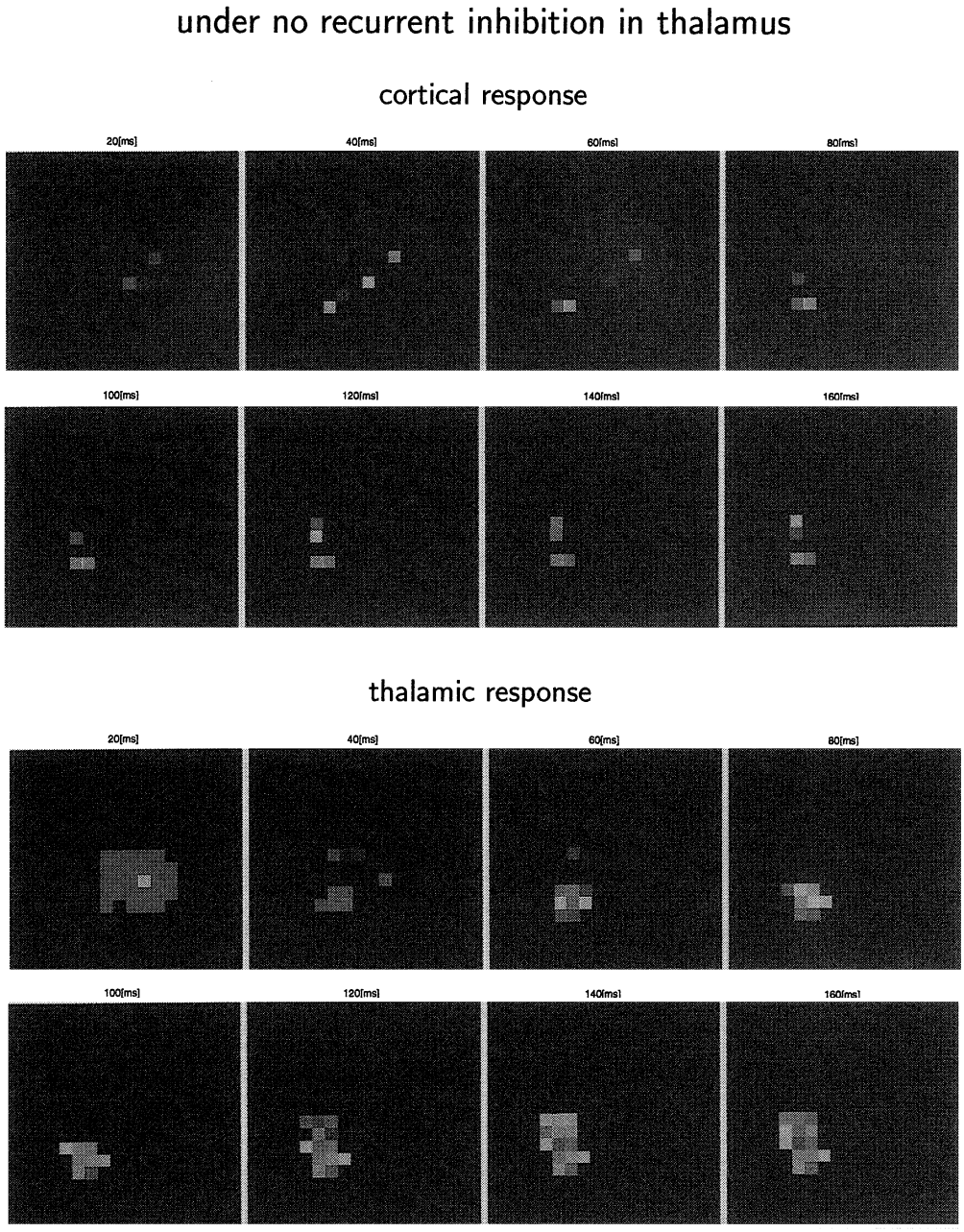

Fig. 9 Cortical and thalamic responses to the stimulus applied to the right plane (intact side) under no recurrent inhibitions in the thalamus. A response increasss in order of blue → green → yellow → orange → red.

Pain sensation in phantom limb is not confined to the special subject of physiology, because the synthesis and maintenance of the body image are deeply involved, which are considered to be essential problem in robotics pursuing highly autonomous functions. Actually, when a robot tries to mimic a motion captured by vision, reproduction of the motion referring to its own body schema should be essential [32]. Human and animals seem to easily realize this operation. How does a robot feel the integrated image of its own body? A hint to this question may be given by studying "phantoms in the brain" [33].

Appendix

A.1 Model overview

Model equations are given as follows [34, 9].

Table 1. Model parameters.

U	transmembrane potential
H	threshold
G_k	potassium conductance
S	spike variable
S_{CN}	potential change produced by the total input current to a neuron
G_i, G_e	the total IPSP and EPSP in terms of conductances
H_0	the resting threshold (10 mV above the resting membrane potential)
U_k	equilibrium potential of the potassium conductance (10 mV below the resting membrane potential)
U_E, U_I	excitatory and inhibitory synaptic equilibrium potentials (70 mV and −10 mV relative to the resting membrane potential)
T_{MEM}, T_H, T_{Gk}	time constants for the decay of U, H, G_k (10, 30, 5 ms)
T_e, T_i	the decay time constants of EPSP and IPSP (8, 20 ms)
B	the amount by which the potassium conductance increases (30)
C	the amount of threshold increase associated with U (0.65)

$$S_{CN} = [G_e \cdot (U_E - U) + G_i \cdot (U_I - U)] \times \frac{G_e + G_k}{G_i + G_e + G_k}$$

$$\frac{dU}{dt} = \frac{-U + [S_{CN} + G_k \cdot (U_K - U)]}{T_{MEM}}$$

$$\frac{dH}{dt} = \frac{-(H - H_0) + C \cdot U)}{T_H}$$

$$S = \begin{cases} 0 & if \quad U < H \\ 1 & if \quad U \geq H \end{cases}$$

$$\frac{dG_k}{dt} = \frac{-G_k + B \cdot S}{T_{G_k}}$$

Parameters are explained in Table 1. EPSP(g_e) and IPSP(g_i) is computed as follows.

$$f_e(t) = \begin{cases} 0 & , \quad t < 0 \\ w_e \cdot \frac{t}{T_e} \cdot \exp \frac{-t}{T_e} & , \quad t \geq 0 \end{cases} \tag{1}$$

$$f_i(t) = \begin{cases} 0 & , \quad t < 0 \\ w_i \cdot \frac{t}{T_i} \cdot \exp \frac{-t}{T_i} & , \quad t \geq 0 \end{cases} \tag{2}$$

where t denotes the time passed from an spike input. w_e and w_i denote synaptic weights, and T_e and T_i are time constants, 8ms and 20ms, respectively. Therefore, when spike input times are given by $t_1, t_2, \ldots, t_n$ $(t_n < t)$, g_e and g_i at time t are given by

$$g_e(t) = \sum_{j=1}^{n} f_e(t - t_j) \tag{3}$$

$$g_i(t) = \sum_{j=1}^{n} f_i(t - t_j). \tag{4}$$

Finally, G_e and G_i are given by summing up g_e and g_i for all synapses, respectively.

A.2 Rule of plastic change of synapse weights

Plastic changes of cortical excitatory synapses and thermocortical synapses obey the following rule.

1. When a neuron fires, synapses which have contributing g_e are enhanced by Δw_e.
   $\Delta w_e = r \cdot (W_e - w_e) \cdot g_e$
   w_e and W_e denote an excitatory synaptic weight and the total amount of synaptic weight for a single neuron, respectively. r is a constant, 0.3 for cortical and 0.01 for thalamocortical connections.
2. After the changes of synapses, a normalization process follows for preserving the total amount of synaptic weights. The maximum and minimum bounds of weights are set as 50% of W_e and 20% of the averaged weight, respectively.

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