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A model of task-specific focal dystonia

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ABSTRACT

Task-specific focal dystonia is a task-specific movement disorder which manifests itself as a loss of voluntary motor control in extensively trained movements. The condition is most frequent in musicians. Until today, the aetiology of focal hand dystonia is not completely understood, but there is growing evidence for an abnormal cortical processing of sensory information, as well as degraded representation of motor functions. It was demonstrated that in the somatosensory cortex the topographical location of sensory inputs from individual fingers is corrupted. Occasionally, a change in sensory information of the hand may at least temporarily improve the condition. This phenomenon is called sensory trick. In this paper, we propose a model of encoding of sensory stimuli which could explain the task specificity of cortical representations of the fingers or other effectors in the context of dystonia. In the framework of this model a sensory stimulus is encoded as a signal vector of higher dimension. A part of its components directly represents the sensory stimulus, while the remaining components describe the context. This model does not only account for the task specificity, but may also explain some characteristics of the retraining process in this disorder.

1. Introduction

The general term dystonia is used to describe a syndrome characterized by involuntary sustained muscle contractions, frequently causing twisting and repetitive movements, or abnormal postures (Jankovic, 2005). If these symptoms are restricted to one body part, the syndrome is termed "focal dystonia". In task-specific focal dystonia the most prominent characteristic is the degradation and loss of voluntary control of highly overlearned complex and skilled movement patterns in a specific sensory-motor task. The highest incidence of focal task related dystonia is found in musicians (Altenmüller & Jabusch, 2009). For example, in pianists suffering from hand dystonia, piano playing might be severely compromised due to loss of fine motor control of individual finger movements; however, writing on a computer keyboard in high speed using ten finger system is not deteriorated. Interestingly, musician's dystonia seems to be clearly related to intense practice of the disturbed movement patterns. For example, fast, repetitive complex movements of the left hand in violinists are more often affected as compared to the more balancing movements of the right bowing hand. Vice versa, in pianists, dystonic movements are predominantly developed in the right hand, which is at least in the baroque, classical and romantic period more challenged as compared to the left due to the more frequent occurrence of fast figurations, trills and scales (Jabusch, Zschucke, Schmidt, Schuele, & Altenmüller, 2005). Although structural abnormalities (Black, Ongur, & Perlmutter, 1998) as well as hereditary factors (Schmidt et al., 2009) have been recently identified, it is generally agreed that overtraining plays an important role in this type of dystonia (Baur, Jabusch, & Altenmüller, 2011). This has impressively been demonstrated in an animal experiment with monkeys trained to do extensive fine motor movements of the hand developing a dystonia-like syndrome (Byl, Merzenich, & Jenkins, 1996). Consequently, it had been suggested that focal dystonia has to do with a misled self organization process. Sanger and Merzenich for example have hypothesized that it is a manifestation of an unstable sensorimotor control loop. They discussed several mechanisms which possibly could lead to a gain >1 in the feedback loop and they formulated a computational model which explains some aspects of focal dystonia (Sanger & Merzenich, 2000). For most of the characteristic phenomena, however, a satisfying theoretical explanation does not yet exist. Amongst them are the following.

- In some musicians with focal dystonia it could be observed that the somatotopic order of the somatosensory finger representations in the cortex was distorted and that the receptive fields of single fingers overlapped (Elbert et al., 1998). This may suggest that the origin is not so much instability of the control loop but primarily a corrupted mapping of sensory signals to cortical regions. In writer's cramp such a distorted mapping has also been demonstrated in the Basal Ganglia, more specifically in the

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Putamen (Delmaire et al., 2005). It has been hypothesized that such a distortion of somatotopic maps is due to maladaptive plasticity and inefficient lateral inhibition, leading to a fusion of previously separated sensory-motor receptive fields. (For a review see Hallett, 1998.)

- Musician's dystonia is in most instances task specific. As mentioned above, the condition affects the control of finger movements predominantly in the context of instrument playing. Astonishingly, it does not impair the function of the same fingers in other activities, although at closer examination minor abnormalities not related to a specific task can become obvious (Byl et al., 1996; Rosset-Llobet, Candia, Fábregas, Ray, & Pascual-Leone, 2007). This means that generally the corrupted mapping is effective only in a special context.

Among the neural networks studied in computer science there is a type which lends itself for modelling cortical mappings and particularly the self organization processes which generate them: the Kohonen network (self organized mapping, SOM, Kohonen, 1982, 1995) is an array of 'theoretical' neurons which processes a set $X = \{x^i\}$ of training signals in such a way that to each signal x^i of the set X a neuron is assigned, i.e. the signal set X is mapped to the array of neurons. An important property of this mapping is that similar signals $x,\ x'$ are mapped to neighbouring neurons. In terms of mathematics this property is called *preservation of neighbourhood*. It closely corresponds to somatotopy in neural organization.

Soon after the first SOM publications (Merzenich et al., 1984) have shown that the reorganization observed in the sensory cortex of mammals after peripheral nerve damage can be modelled as an adaptation process in a Kohonen map. Martinetz, Ritter, and Schulten (1988) demonstrated that the auditory cortex of a bat may be considered as a neighbourhood preserving map of the relevant space of ultrasonic signals. Meanwhile the SOM concept is accepted as a tool for understanding the processing of sensory signals in the brain (Sirosh & Miikkulainen, 1995; Turrigiano & Nelson, 2004; Wiemer, Spengler, Joublin, Stagge, & Wacquant, 2000).

The fact that after amputation of a digit the corresponding cortical region by and by takes over new functions was impressingly modelled in the simulation experiment (Merzenich et al., 1984). Yet this raises the question how stimuli are encoded as signals and whether the mapping of different stimuli σ, σ' to different neurons N, N' implies a corresponding wiring of signal paths. If there were simple signal paths from sensors to neurons of the cortex then it were difficult to explain how the connections are modified during the self organization process and how they are switched to another cortical region after an amputation. A more plausible assumption is that each stimulus from a certain body region is encoded as a set of simultaneous signals which are transmitted to the cortex via one and the same bundle of paths. A similar assumption was already made in the paper by Merzenich et al. (1984). Here a stimulus is represented by a two-component signal vector $\vec{x} = (x_1, x_2)$ where x_1, x_2 are the coordinates describing the location of the stimulated sensor. This assumption was justified within the scope of a principal demonstration. However the (Cartesian) coordinate pair x_1, x_2 refers to a pair of rectangular axes which have no natural relationship to biological structures. It is not plausible that a sensor in the hand surface will produce signals x_1, x_2 which depend on the choice of a special coordinate system.

Therefore we propose a simple model which explains the encoding of stimuli as high-dimensional signal vectors $\vec{x}$. This is the reason why a sensory stimulus was denoted above not as a single signal x but as a *signal vector* $\vec{x} = (x_1, x_2, \ldots, x_k)$. The encoding of stimuli is based solely on the concept of distance. It does not refer to a special coordinate system.

If one accepts the hypothesis that each stimulus is represented by a high-dimensional vector $\vec{x}$ then it needs only a slight generalization of this picture to explain the task specificity of focal dystonias.

The function principle of self organized mappings is sketched in Section 2. Here naturally we must restrict ourselves to those features which are important in the context of the present paper. Section 3 describes the model of stimulus encoding, i.e. the generation of a signal vector $\vec{x}$ for a given stimulus σ. The model is tested by a simulation experiment which in a certain sense is a remake of the experiment by Merzenich et al. (1984). It reveals that the neighbourhood preserving mapping between hand and sensory cortex as well as the reorganization of this mapping after amputation of a digit is correctly reproduced. In Section 4 it is shown that application of the same principles to more general types of stimuli leads to an explanation of task specificity. This is not only of theoretical interest. In attempts to cure musician's dystonia by retraining, one of the problems is the so called deprogramming, i.e. the destruction of a corrupted cortical representation. If the mechanism of task specificity were known this would facilitate the planning of deprogramming strategies.

2. Cortical representations as self organized mappings: Kohonen networks

A structure which lends itself for modelling cortical representations is the Kohonen network (Self Organized Mapping, SOM). It is a mapping of a signal set $X \subset \mathbf{R}^m$ to a regular, typically two-dimensional grid of $n \times n$ simulated neurons $N_{p,q}$. A signal $\vec{x} \in X$ is given in parallel to all neurons and the neuron $N_{p,q}$ with maximal output y, the so called winner neuron, is considered the picture of $\vec{x}$. We will denote it by N_{p^c,q^c}. The mapping is neighbourhood preserving and since preservation of neighbourhood corresponds to what is called somatotopy in biology, Kohonen mappings are interesting for modelling cortical representations. In the present paper a somewhat different SOM, the DCNG SOM (Müller, 2009) (Data Cluster to Neuron Groups), was used. Therefore we recapitulate the training process of the Kohonen SOM only so far as it is necessary for explaining the DCNG SOM and the motivation for its use.

In the Kohonen SOM the output y of a neuron $N_{p,q}$ with input $\vec{x}$ is

$$y_{pq} = (\vec{x},\ \vec{w}_{p,q}). \tag{1}$$

Here the weight vector $\vec{w}_{p,q}$ describes the synaptic couplings of the signal components $x_1, x_2, \ldots, x_m$ to the neuron $N_{p,q}$ and $\phi(u)$ is a monotonically increasing function of the argument u.

In training a Kohonen SOM for a mapping of X the elements $\vec{x} \in X$ are given in random order to the net N. For each $\vec{x}$ the winner neuron N_{p^c,q^c} is determined. Now all neurons $N_{i,j}$ of the net are adapted to $\vec{x}$ according to

$$\vec{w}_{i,j} := \vec{w}_{i,j} + g(|i - p^c|, |j - q^c|; B) \cdot \alpha \cdot (\vec{x} - \vec{w}_{i,j}). \tag{2}$$

The parameter α determines the overall strength of the adaptation. The function $g(a, b; B)$ is 1.0 for $a = b = 0$ and decreases with increasing arguments a, b over a range $\approx B$. Thus not only the competency of the winner neuron for $\vec{x}$ is reinforced but the w-vectors of the neighbouring neurons too are somewhat shifted to $\vec{x}$. This makes sure that the mapping of X to $\{N_{p,q}\}$ is smoothed and becomes neighbourhood preserving. As in most adaptive systems the adaptation strength α is reduced gradually during the training process. In order to achieve neighbourhood preservation over the whole net as well as in detail the neighbourhood parameter B must start with a value of the order $N/2$ and is to be successively reduced. In several simulation experiments it has been shown that the Kohonen SOM can explain reorganization processes observed in the mammalian sensory cortex (Parpia, 2011). Nevertheless it has at least two properties which are biologically implausible.