Pointwise Organizing Projections

Teuvo Kohonen
Helsinki University of Technology
Neural Networks Research Centre
Espoo. Finland
teuvo.kohonen@hut.fi

Abstract - *The SOM algorithm was introduced to create abstract-feature maps. It is not correct to use it as a model of pointwise neural projections such as the somatotopic maps or the maps of the visual field, because the SOM does not transfer signal patterns: the winner-take-all function at its output only defines a single response. This presentation introduces a new self-organizing system model related to the SOM that has a linear transfer function for patterns all the time. Starting from a randomly interconnected pair of neural layers, it creates a pointwise ordered mapping from the input layer to the output layer.*

Key words - brain map, neural projection, self-organizing network, POP, SOM

1 Introduction

There exist two main types of brain maps: 1. Pointwise ordered *projections* from a receptive surface onto a cortical area (e.g. the visual and somatosensory maps). 2. *Feature maps,* which are ordered along with some sensory feature value (e.g. color) or a computed entity (such as the spatial location or orientation of the subject or an object). The self-organizing map (SOM) paradigm [1] was originally only meant to explain the Type 2 (abstract feature) maps. All previous attempts to construct Type 1 (pointwise projective) maps artificially have been either defective or erroneous. The scope of this talk is to discuss the possibility of creating Type 1 (pointwise projective) maps in a new self-organized fashion related to the SOM.

The SOM was not the first model of cortical organization (cf., e.g., the line detector model of v.d. Malsburg of 1973 [2] and the "nerve field" model of Amari of 1980 for the Type 1 pointwise maps [3]). Unfortunately, none of these attempts was a success. The main weakness of the earlier models was that they were based solely on excitatory and inhibitory lateral connections, and the Hebbian rule of synaptic plasticity of the afferent connections. For instance, the model of v.d. Malsburg was "brittle," because the reported patchwise ordering ensued when using fixed lateral connectivity values, some of which were defined with the accuracy of two or three decimals, and no attempts have later been made to improve or generalize the model. In the nerve-field type models, on the other hand, a genetically defined *preordering* of the connectivity had to be assumed; this order was then shown to be stable. However, when starting with the disordered initial state, no global ordering was ever obtained; only a weak tendency of *patchwise* ordering has been observed.

My original, biologically inspired solution to create *globally* ordered abstract-feature maps, starting from a *disordered* state, was to assume two different kinds of state variables: 1. By means of very-short-range lateral excitatory connections and somewhat-longer-range lateral inhibitory connections, a winner-take-all (WTA) function can be implemented, which concentrates the neural activity onto a small spot. 2. A separate effect spreads some kind of plasticity control to the spatial neighborhood of the winner. The essence of these two separate functions, viz. the WTA and the neighborhood function, respectively, was dressed into the mathematical SOM algorithm, which no longer refers to any specific biological components. Some people, however, have tried to implement the pointwise projective (Type 1) ordered maps, e.g., the somatosensory or visual maps, by means of the SOM algorithm, too, using the coordinates of the receptive surface as a two-dimensional input to the SOM array. This is totally wrong, first, because the sensory systems have no means to decode and transfer the coordinate values of a stimulus to the brain, second, because the input coordinates correspond to the stimulation of only one point in the input layer, and third, because the WTA only defines a single output response. Contrary to that, many brain maps produce plenty of concurrent output responses to a multimodal stimulus pattern.

In the biological realms, genetic information defines a very rough initial order of the neural projections. Refinement of this order begins already prenatally, by means of endogenous signals generated by the network itself. The final resolution of the mapping, and optimization of the neural resources (magnification factor) are achieved postnatally, according to the sensory experiences. It has been demonstrated by Chang and Merzenich [4] that exposing newborn rats to continuous moderate-level acoustic noise, the development and refinement of the tonotopic maps will be delayed long beyond normal periods. Nakahara et al. [5] have showed that the exposure of infant rats to complex tone sequences results in altered auditory cortex organization. These observations, among many others, prove that the input-driven organization of the brain maps is a fact and needs a new theoretical model. I shall demonstrate the formation of such a mapping, starting with random interconnectivity.

2 A new model for a pointwise projective self-organizing map

The objective of this presentation is to demonstrate the self-organized formation of a pointwise mapping. Without essentially losing in generality, in order to reduce the computing load, and to facilitate a simple graphic display, the input and output "layers" in this experiment are defined as one dimensional. The first choice we have to made is to decide whether we start from a completely disordered state, i.e., with randomly interconnected layers, or whether we presume some grand initial order. The latter would correspond to the state from which postnatal learning begins in biology, but restricting ourselves to such a postulated order would make the problem so easy that the simulation results would seem almost trivial. To keep the theory sufficiently general, we assume only a single process model, which, nonetheless, may undergo different phases for different input data. Let us start with random initial connections. We shall try to use *multimodal* signals all the time, in order to mimic real stimulus patterns. (I use the term "mode" in the same sense as in statistics, meaning a "peak" or local maximum. Such a "mode" may be due to a localized component function.)

All the units of the input layer are initially connected to all of the output units by random weights. As we want to use this network for signal transfer, its transfer function is assumed

as linear at all times:

$$y_i = \sum_{j=1}^{n} w_{ij} x_j \ , \tag{1}$$

where x_j is the activity at synapse j of neuron i, in these experiments $n = 50$, and w_{ij} is the variable strength of synapse j of neuron i.

In a somewhat similar sense as in the "biological" SOM algorithm, a spatially distributed *plasticity-control agent* is assumed. In this model, the local concentration z_i of the agent at unit i is assumed to be describable by the convolution of the activities y_h at the units h and some point-spread kernel g_{h-i} that describes the spreading of the agent or its effect from location h to location i:

$$z_i = \sum_{h=1-\delta}^{n+\delta} g_{h-i} y_h \ . \tag{2}$$

The values of the y vector have thereupon been extended by δ positions to the left and right beyond the borders of the output layer in order to allow the point-spread kernel to work reasonably well at the borders, too.

The concentration of the plasticity-control agent z_i, on the other hand, shall control the modifiabilities of the connections in a nonlinear, progressive fashion. Let u_i be the plasticity-control effect at location i:

$$u_i = f(z_i) \ , \tag{3}$$

where f is a positive, monotonically increasing function to be defined later on.

Conventionally, the signal-dependent modifications of the synaptic efficacies have been assumed to obey the law of Hebb: the changes are proportional to the product of input and output activities. However, if the self-ordering connections must learn from multimodal input signals, where the number and locations of the modes are variable, I have found it necessary that the changes shall be proportional not only to the presynaptic activities but also to some function of the synaptic strengths themselves. The need for such an extra condition may not be quite obvious and cannot be demonstrated here in detail. Let it suffice to mention that when a multimodal input signal pattern matches with a synaptic weight pattern, the degree of matching is mainly due to the coincidence of one of the activity modes with the strongest synapses. Learning should then be restricted locally to the neighborhood of these strongest synapses, e.g., by making the learning proportional to the synaptic strength, or otherwise the activated neurons try to learn the "irrelevant" signal modes present at their inputs, too. In this way, different neurons can simultaneously learn from different modes.

On the other hand, the postsynaptic activity in the law of Hebb is now replaced by the combined effect of the neighboring neurons. Let $w_{ij}(t)$ be the strength of the synaptic connection from input unit j to output neuron i at time t. Then the adaptation or updating equations of the interconnection strengths are assumed to read in the discrete-time formalism as

$$w_{ij}(t+1) = w_{ij}(t) + \alpha(t)[1 + \beta w_{ij}(t)] u_i(t) x_j(t) \ , \tag{4}$$

where $\alpha(t)$ is a time-variable parameter that describes the synaptic plasticity, and β is a constant.

Eq. (5) resembles the law of Hebb, with the exceptions that 1. the postsynaptic activity is replaced by the local control effect $u_i(t)$ that depends on the postsynaptic activities of the neighboring neurons, and 2. the rate of modification is further made to depend on the efficacy of the connection w_{ij} itself.

However, the law of Hebb and also eq.(4) as such would still be unrealistic in practice, because they only describe the conditional *strengthening* of the synapses. This would sooner or later lead to the saturation of the synapses. In reality, unused synapses regress. In this simulation, to make the connections increase and decrease, the w_{ij} are normalized by dividing each $w_{ij}(t+1)$ by the Euclidean norm of the set of the $w_{ij}(t+1)$. Such a normalization was already used in the original "dot-product SOM" algorithm, and there it was assumed to have a biological counterpart, e.g., in the redistribution of the synaptic resources within a cell.

There are still two functional forms to be defined for this system model, namely, the convolution kernel g_{h-i}, and the nonlinearity f of the effect of the plasticity-control agent. No clues can be obtained from biological data, and there are indefinitely many choices for the mathematical forms. I have tried many options with varying success. For the present report we may rest content with the choices that look reasonable and have produced successful orderings. Let us take for the convolution kernel a law that resembles the diffusion effect:

$$g_{h-i} = \frac{1}{1 + |h-i|/c} \; , \tag{5}$$

where c is a constant, similar to the diffusion length. For the nonlinear effect of the plasticity-control agent we take

$$u_i = f(z_i) = pos(e^{az_i} - b) \; , \tag{6}$$

where $pos(x)$ is 1 if $x > 1$ and zero otherwise, and a and b are free parameters. Eventually b can be taken equal to zero. (Notice that exponential laws often apply to chemical effects.)

Earlier the normalization of the updating equation (4) was discussed. The competitive-learning processes are in general rather sensitive to the condition that the values to be compared are presented in common scales. The neural systems have many means to normalize the neural signals to common scales. Therefore, to maximize the ordering tendencies, normalization was carried out in all of the following places: 1. The vector of the output values y_i was normalized with respect to its maximum element. 2. A similar normalization was performed for the z_i. 3. Moreover, the vector of the plasticity-control effects u_i was normalized with respect to the sum of its elements. The latter choice corresponds to locally and temporally limited resources of the plasticity-control agent. After more careful studies, it may be possible to simplify and combine some of these normalizations.

In the first place I want to show that the pointwise organization will result even when starting with a randomly interconnected state, and using *multimodal* input patterns. This kind of ordering would be impossible in the traditional SOM. Let the input pattern consist of K Gaussian components, centered at random in the input layer:

$$x_i = \sum_{k=1}^{K} e^{-(i-d_k)^2/2s^2} \; , \tag{7}$$

where the d_k are picked up at random from [1,50].

3 Simulations

3.1 Main result

The initial values for the w_{ij} were selected at random from the range [0,1] with uniform probability, after which, for each i, the sets of the input weights were normalized (using the Euclidean norm). The initial connectivity matrix is shown in Fig. 1a, where row i corresponds to the ith output neuron, and column j to its jth synapse, or the jth input neuron, respectively. In eq.(2), $\delta = 3$. The size of the small squares describes the value of w_{ij}.

The parameter β in eq.(4) was equal to 100. The point-spread parameter c in eq.(5) was equal to 10, and the parameters in eq.(6) were: $a = 4$, $b = 12$. In eq.(7), the displacements d_k were selected from the range [1,50] with a uniform probability, the number K of the Gaussian components was drawn at random from [1,3]. The only time-variable system parameter is the learning-rate factor α like in the SOM, but additionally, the resolution of the input signal patterns was made to improve with time. For these two functions I finally selected

$$s = s(t) = 2 + 10/(1 + .002t) \ , \tag{8}$$

$$\alpha(t) = 1/(1 + .004t) \ . \tag{9}$$

The connectivity matrix after 25 000 training cycles is shown in Fig. 1b. Like in the SOM, when using random processes for intialization and input signals, the *direction* of ordering will result as the mirror image in half of the cases.

3.2 Refinement of initial order

If one starts with a rough initial order, the training pattern can be much more complex than before. Fig. 1c delineates a roughly preordered initial state. It can be obtained, e.g., using broad unimodal input patterns (soft Gaussians) as input and the value $\beta = 0$ in eq.(4). Fig. 1d gives the ordering result when the number K of the Gaussian components in the input pattern was drawn at random from [1,10] using 50 000 training cycles.

3.3 Magnification factor

Like in the SOM, the area in the output layer occupied by a certain subset of input projections can be shown to depend on the frequency of stimulation of this subset. I have carried out numerous tests to evaluate the magnitude of this *magnification factor*, but the effect seems to depend on the particular system parameters and the signal history.

3.4 Random input

A control check was performed by using as the elements of the input vectors random numbers from the range [0,1]. The system model was otherwise the same as that defined in Secs. 2 and 3.1. Fig. 1e shows the connectivity matrix obtained after 50 000 training cycles.

Another control check was carried out starting with the roughly preordered initial state depicted in Fig. 1c, and continuing the refinement process with random inputs for 50 000

training cycles. The result is shown in Fig. 1f. One can discern a "columnar" structure, but no refinement of the initial ordering has taken place.

4 Biological conditions

The evidence for some kind of *local control* of the synaptic plasticity comes from many observations: theoretical, physiological, and behavioral. One might think that there exist plenty of chemical signaling agents in the neural realms to that end. From the point of view of the theories of self organization, however, at least the following two conditions must be met: 1. The lateral spread of the agent or effect must have a proper range, at least several millimeters, to comply with the sizes of the sensory maps. 2. The time constants of the agents or effects must be sufficiently short, of the same order of magnitude as the typical changes in input patterns, in order that the adaptation effects follow the signals.

The time constants of the neural growth factors are much too long in order that the latter could act as local plasticity-control agents.

Some years ago it was believed that the synaptic plasticity is controlled by the NO (nitric oxide) molecules, which are produced at the synaptic terminals in proportion to their activities and diffused into the extracellular space. New estimates by Philippides et al. [6] as well as Thomas et al. [7] indicate that the diffusion range of NO may be only on the order of 100 microns. This is too short a value to account for map formation.

The well-known plasticity-controlling neuromodulators like noradrenaline spread diffusely across the cerebral cortex. However, it is thinkable that the local neural signals are somehow able to affect the receptors of these neuromodulators, enhancing the plasticity-control effects locally or restricting them to the neighborhoods of signal activities. This control effect might also have a shorter time constant than that of the transmission of the neuromodulators themselves. On the other hand, it is also imaginable that the plasticity-control effects are mediated by anatomical structures such as the interneurons and their nonsynaptic control actions.

An up-to-date account of the time-dependent plasticity is given in Ref. [8].

5 Conclusions

The original Self-Organizing Map (SOM) paradigm was inspired by a need to explain the *abstract-feature maps* of the brain. Contrary to that, the pointwise projective maps such as the visual and somatotopic maps should not be described by the SOM algorithm, first of all, because the winner-take-all function only defines a single output response at a time.

This presentation introduces a new model in which the ordering of the pointwise projections is driven by the input signals. No initial order needs to be assumed. It may be proper to call it the *POP (pointwise organizing projections)*. Unlike in the SOM algorithm, the learning process does not rely on any global winner-take-all function. Especially at the later steps of learning, the map can produce an indefinite number of separate, simultaneous output responses, around each of which learning proceeds concurrently and is decoupled from the learning processes that take place at the other locations. The signal transfer mapping is linear at all times, and becomes finally a one-to-one spatial mapping over almost all of the input and output layers, let alone some minor boundary effects.

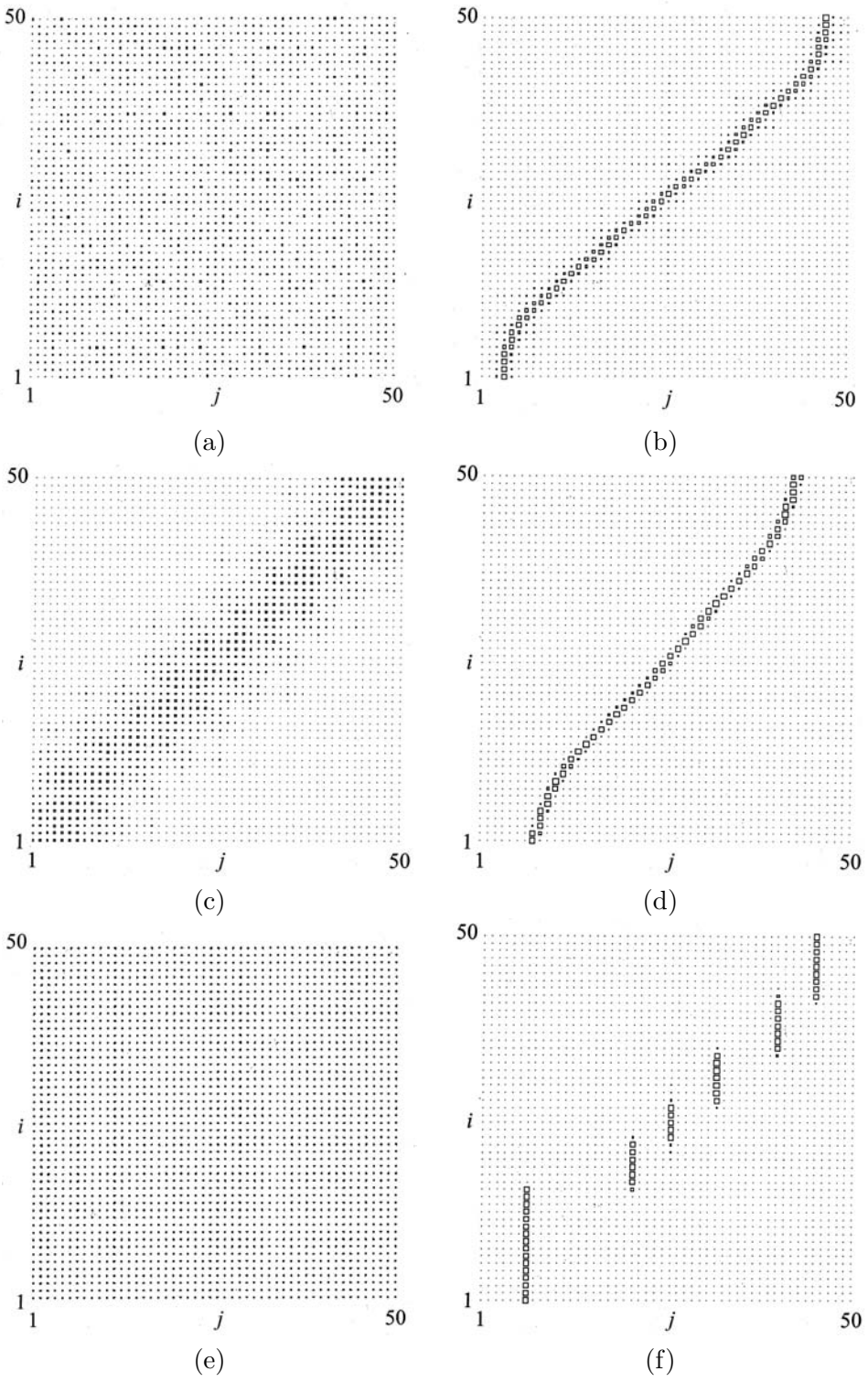

Figure 1: (a) Initialization (b) Main result, one to three modes (c) Rough initial order (d) Refinement, one to ten modes (e) Random input (f) Rough initial order followed by random input

In biology, the ordered neural projections are roughly preprogrammed genetically, and prenatal endogenous signals are also at work in defining the neonatal order of the maps. Although global ordering seems to be possible even when starting with a disordered initial state, some results in this work indicate that if there exists even a slight degree of initial order, this order will be improved markedly by the sensory experiences, and the refinement process then tolerates more complex stimuli than when starting from a complete disorder.

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