A mathematical model of receptive field reorganization following stroke

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Abstract—Insufficient blood transport to neurons in the brain due to blocked or ruptured blood vessels (stroke) can lead to damage or death of cells, causing functional impairment. Intact neurons surrounding a stroke-like lesion have been shown to adapt to the damage by expanding their sensory receptive fields in the direction towards the lesion, thereby restoring information processing capacity within the cortex. We developed model of the effect of focal ischaemia on the performance of a neuronal population code, in order to study physiological parameters that could be influenced to enhance recovery from stroke. Our findings show that recovery of the accuracy of the population code is optimal by a specific amount of receptive stroke. Our findings show that recovery of the accuracy of the population code is optimal by a specific amount of receptive plasticity. This plasticity may be influenced by changing the parameters that could be influenced to enhance recovery from a neuronal population code, in order to study physiological model of the effect of focal ischaemia on the performance of information processing capacity within the cortex. We developed fields in the direction towards the lesion, thereby restoring to adapt to the damage by expanding their sensory receptive fields. Studies on stroke recovery associate restoration of function with reorganization in the brain [1], [2], [3]. The reorganization of neural activity that follows after stroke is very important in producing functional recovery. Moreover, it has been observed that reorganization after stroke can lead to an enlargement of the receptive fields (RFs) in the region surrounding the lesion [4-12].

Several observations have shown that following stroke, the levels of γ-aminobutyric acid (GABAergic) inhibition in neighboring brain areas drop and others have shown that a reduction of GABAergic inhibition may favor cortical plasticity ([13], [14], [15], [16]). From these findings it has been hypothesised that the brain supports recovery from lesion by decreasing GABAergic inhibition and thereby facilitating plasticity and reorganization of the cortical representation in surrounding areas. The expansion of the receptive fields can then be explained by the dis-inhibition caused by reduction in GABA. Changes in levels of GABA only affect the inhibitory parts of receptive fields but leave the excitatory parts unaffected. This disrupted balance between excitation and inhibition causes the receptive field to expand, where dis-inhibition is apparent, asymmetrically from its original position towards the lesion.

A remarkable feature of sensory cortices in the brain is that the sensory world can be mapped topographically onto the cortical surface. This means that neighboring points in the sensory field evoke activity in neighboring regions of the cortex. Since receptive fields overlap, each point is monitored by a population of neighboring cells, rather than a single cell, and when a point is stimulated the population of neurons whose receptive fields include that particular site are excited. In this work, we present a computational model of a topographically mapped population code which includes a focal lesion as well as a process for receptive field enlargement (plasticity). The model simulates the recovery processes in the brain, and allows us to investigate mechanisms which increase the ability of the cortex to restore lost brain functions. Changes in the degree of plasticity of the receptive fields of the neurons, which could potentially be influenced to enhance information transfer through the cortex post stroke, were incorporated into the model. This allowed the exploration of effects resulting from changes in the concentration of this parameter on the level of functional restoration. Neurons close to the damaged region expand their receptive field more than those neurons further away in the cortex.

We use Fisher Information in order to calculate how much information a neural response carries about the stimuli. We estimate the Fisher Information carried by the topographic map before and after the stroke. We find that by tuning the receptive field plasticity to a certain value, the information transfer through the cortex after stroke can be optimized.

II. Methods

For simplicity we use a simple Gaussian receptive field which does not take into account any dependency in the orientation of the object. A centre-surround receptive field can be represented by a two-dimensional spatial Gaussian function. Our model is based on the assumption that a population of neurons with Gaussian receptive fields can be mapped topographically onto a two dimensional lattice. The model could therefore be taken to represent neurons which exhibit centre surrounding receptive field such as retinal ganglions cells adapting to a retinal lesion. However, this will also be used to describe the recovery process of the envelopes of cortical receptive fields. That is, functional recovery due to the reorganization of neuronal activity that takes place in the cortex after a stroke-like lesion will be investigated. The modeling will be based on the finding that intact neurons surrounding the lesion partially restore cortical function by taking up some of the role of the damaged neurons [13], [14].
The prototypical cortex model

Simple cells in the primary visual cortex are selective not only to for the size of the object but also for their orientation. However, for sake of simplicity we choose to use a simple Gaussian model which it does not include orientation dependency. The core of the model is an uncorrelated population of $n$ neurons, each defined by a two dimensional centre-surround Gaussian receptive field, $G(x, y)n$, with $x$ and $y$ representing the spatial coordinates of the stimulus, and $x_0, y_0$ the centre of the receptive field for a given neuron. In order to name explicitly the excitatory and inhibitory part, we can write this equation as

$$G(x, y)n = \pm (f^0_{exc}(x, y)n + f^0_{in}(x, y)n) \tag{1}$$

where $f^0_{exc}(x, y)n$ and $f^0_{in}(x, y)n$ are defined as,

$$f^0_{exc}(x, y)n = \frac{1}{2\pi \sigma^2_{cen}} \exp \left(\frac{-(x-x_0)^2 + (y-y_0)^2}{2\sigma^2_{cen}}\right) \tag{2}$$

and

$$f^0_{in}(x, y)n = -\frac{B}{2\pi \sigma^2_{sur}} \exp \left(\frac{-(x-x_0)^2 + (y-y_0)^2}{2\sigma^2_{sur}}\right) \tag{3}$$

The standard deviation $\sigma_{cen}$ determines the width of the central part of the curve and $\sigma_{sur}$ determines the width of the annular surrounding region. The value of $B$ determines the balance between excitatory and inhibitory regions.

Each receptive field is constructed by adding positive and negative Gaussian functions together. The positive function represents the excitatory part of the receptive field, i.e. where positive stimulus, $s$, elicits an increase in firing rate. The negative function represents the inhibitory part, where a positive stimulus causes decreasing firing rate. The excitatory part for each neuron $n$ is termed $f_{exc}(x, y)n$, and the inhibitory part for each neuron $n$ is termed $f_{in}(x, y)n$.

The superscript $^0$ stresses that these are the definitions for the receptive field of neuron $n$ pre lesion.

Neuronal responses are complex and variable thus describing the relationship between stimulus and response is a difficult task. A simple neuronal model can estimate firing rates as instantaneous functions of the corresponding applied stimulus by assuming that contributions from different locations within the visual field sum linearly. That is, the spatial input stimulus, $s(x, y)$, is weighted linearly by the receptive field of each neuron, $G(x, y)n$. The linear response of each neuron $n$ to the input stimulus is thus generated by:

$$r_{linear}(x, y)n = \sum_{x=1}^{size_x} \sum_{y=1}^{size_y} G(x, y)n \cdot s(x, y) \tag{4}$$

where $size_x$ and $size_y$ determine the height and width of the spatially mapped visual field respectively. By adding to the model a threshold-gain function, $T$, which is appropriately bounded from above and below the firing rate will be never be negative or unrealistically large.
leave excitation unaffected. The reduction of GABA causes dis-inhibition of adapting receptive fields in the direction of the lesion. The amount of dis-inhibition apparent in neuron \( n \) is controlled by the receptive field plasticity parameter \( \gamma \) and the distance \( d_n \) between the damage and the neuron in question. The dis-inhibited inhibitory part, \( f_{in}(x, y)_n \), of a receptive field facing in the direction of the lesion can be described in terms of the original inhibitory function, \( f_{in}^0(x, y)_n \), plasticity and distance by:

\[
f_{in}(x, y)_n = f_{in}^0(x, y)_n \cdot F(\gamma, d_n)
\]

(8)

where \( F(\gamma, d_n) \) is defined as

\[
F(\gamma, d_n) = 1 - \exp\left(-\frac{d_n}{\gamma}\right)
\]

(9)

Eq. (9) fulfills the requirement that the dis-inhibition should increase with increasing values of \( \gamma \) and decrease with increasing values of \( d_n \) but the equation is purely hypothetical and this relationship remains to be fitted to experimental data.

The total receptive field structure is given by the summation of the Gaussian excitatory and inhibitory functions. Since the excitatory part, \( f_{ex}^0 \) is left unaffected during the recovery process, the new total receptive field \( G \) is expanded from its original position towards the lesion and has the form:

\[
G(x, y)_n = f_{ex}^0(x, y)_n + f_{in}^0(x, y)_n \cdot \left[1 - \exp\left(-\frac{d_n}{\gamma}\right)\right]
\]

(10)

According to eq. 8 - 10, neurons close to the damage experience greater dis-inhibition and therefore greater expansion of their receptive fields than neurons further away in the cortex.

Analytical approach

In the following, we denote the cortical spontaneous neuronal activity \( r \), and making use of the Central Limit Theorem, assume that \( r \) is normally distributed about \( f_n \).

\[
P(n | xy) = \frac{1}{\sqrt{2\pi}\sigma_n^2}\exp\left(-\frac{(n - f_n(x, y))^2}{2\sigma_n^2}\right)
\]

(11)

where \( f_n(x, y) \) is a 2-D well shape tuning function. Fisher information can be calculated as

\[
I = -\int_0^R dr \log P(r | xy) \left[\frac{\partial^2}{\partial x^2} \log P(r | xy)\right]
\]

(12)

As we described, the receptive field is made up of an excitatory and inhibitory part \( f = f_{in} + f_{ex} \), which may also account modifications in the receptive field when a localized damage take place. This is, considering that a finite number of cells have been removed the inhibitory part changes after the lesion as states in eq (9):

\[
f_{in} = f_{in}^0(x, y)_n (1 - \exp(-d(n)/(\gamma(n))))
\]

(13)

were \( d(n) \) is the minimal distance from a given cell to the damage and \( \gamma(n) \) is defined as the plasticity after the lesion removes the neuron of cartesian coordinates \( x_i, y_j \).

\[
d(n) = \sqrt{(x_n - x_i)^2 + (y_n - y_j)^2}
\]

Notice that \( x_i \) and \( y_j \) correspond to the position \((i, j)\) of the sites which have been removed. After some algebra, and by assuming \( R \to \infty \) this leads to

\[
I = I_{ex} + I_{in} + I_{in-ex}
\]

(15)

where \( I_{ex} \) and \( I_{in} \) are the excitatory and inhibitory contributions respectively. But, \( I_{in-ex} \) correspond to a mixed contribution made up of excitatory and inhibitory elements.

The excitatory term can be written as

\[
I_{ex} = \sum_n \left\{ \frac{f_{ex}^0(x, y)_n}{\sigma_n^2} \right\} \frac{(x_n - x_0)(y_n - y_0)}{(\sigma_{ex})^2} - \frac{2(x_n - x_0)(y_n - y_0)}{(\sigma_{ex})^2 d(n)\gamma} + \frac{(x_n - x_0)(y_n - y_0)e^{-d(n)/(\gamma n)}}{(\sigma_{ex})^2 d(n)\gamma}
\]

(16)

and the mixed term,

\[
I_{in-ex} = \sum_n \left\{ \frac{f_{in}^0(x, y)_n}{\sigma_n^2} \right\} \frac{(x_n - x_0)(y_n - y_0)}{(\sigma_{ex})^2} - \frac{2(x_n - x_0)(y_n - y_0)}{(\sigma_{ex})^2 d(n)\gamma} + \frac{(x_n - x_0)(y_n - y_0)e^{-d(n)/(\gamma n)}}{(\sigma_{ex})^2 d(n)\gamma}
\]

(17)

This allows us to represent the Fisher topographic information as function of the plasticity \( \gamma \), with respect to the distance to the lesion \( d(n) \) and the size of the lesion which is implicitly included in the number of removal sites \((x_i, y_j)\).

III. RESULTS

A. Enlargement of receptive field

We initially present simulation results which show the effect of the post-lesion plasticity on the receptive fields, before following with analytical results which have been numerically solved for precisely the same situation as that simulated.

The results in this section all stem from the same population, the number of neurons is 225 and it spans 16 degrees of the visual field in both the x- and the y directions. Once a lesion has been induced, GABA levels in the cortex drop, the plasticity parameter \( \gamma \) is increased and dis-inhibition takes place in the intact surrounding neurons, in the direction of the damage. The drop in the GABA levels, and therefore the increase in plasticity, is dependent on the size of the lesion and on \( \gamma \).

Figure 2 shows a contour plot of a receptive field of a single neuron in the neural population. The peak of the receptive field, \((x_0, y_0)\), is dependent of the position of the neuron
within the cortex because its receptive field are mapped across it. In each trial, a white noise is applied to the population and the response of each neuron is recorded and used to calculate the points of the receptive field.

The amount of overlap in the receptive field is important in terms of information transfer. Figure 3 shows how receptive fields of neurons in a population of neurons are topographically mapped across the cortex. Only a few of the neurons present in the population are shown in order to make the figure more understandable. Each receptive field is represented by its peak point, \((x_0, y_0)\), and a curve indicating the half width of the field. The amount of overlap of receptive fields is determined by the number of neurons in the population, the size of the visual field being represented and the width of the receptive fields \(\sigma\).

Figure 4 shows how the size of a damage influences the enlargement of receptive field. The damage in Figure 4B covers a larger area of neurons than the damage in Figure 4A and therefore influences a greater drop in inhibition. The plasticity levels are increased, dependent on the drop in GABA and for these simulations, \(\gamma = 0.02\) refers to the plasticity level in the cortex following a small lesion shown in Figure 4A and \(\gamma = 0.1\) is the plasticity value induced by the larger lesion shown in Figure 4B.

The distance between neurons and the damage is a significant parameter. Neurons that lie closer to the lesion in Figure 4 exhibit even larger changes in their receptive fields due to damages of the same sizes as presented in the figure. On the other hand, neurons lying further away from the lesion experience less enlargement in their receptive field. Figure 5 shows the relationship between the expansion of receptive fields and the distance of neurons to the lesion. The expansion of each receptive field is represented as the deviation, in
Expansion of RFs [%] after Inf. / Inf. before with γ A and B respectively. The value of the values induced by the two damages shown in Figure 4 and each cell within the damaged area.

The two lowest plasticity values presented in Figure 5 are the values induced by the two damages shown in Figure 4 A and B respectively. The value of γ = 0.02 only affects neurons closer to the damage than d = 10. It is evident that γ = 0.81 causes substantial enlargement in all neurons in the population, even the ones at the greatest, d. However, if the population were larger than the one presented here, the effects would decrease further away from the damage in the same manner and with the same relationship as for plasticity values γ = 0.02 and γ = 0.1.

**B. Fisher Topographic information**

Equations (17), (18) and (19) were implemented in order to explore the effects of different values of γ on the Fisher Information I. Figure 6 shows that the information transfer post stroke is dependent on the plasticity, γ. Following a stroke-like damage, the information transfer drops from its original value because neurons within the damaged area do not convey information about the applied stimuli. When the plasticity level is raised, the neurons surrounding the damage start to expand their receptive fields in order to take up lost functions, and the information transfer increases. However, when γ is raised even further and the expansion increases, overlap of the receptive fields becomes too much and information is lost again. This is due to the fact that infinitely large receptive field do not provide any information about applied stimuli because every stimulus pattern is weighted in the same way and no discrimination is accomplished. Large overlapping receptive fields are therefore not specific enough to give information about the input. There is therefore a certain value, γ 0 which optimises the performance of the cortex. Figure 6 shows the Fisher Information for three different sizes of damages. Bigger lesion led to more information loss and less recovery with increasing γ.

Note that although we have presented results here using the analytical formulae derived, we have obtained very similar results using a purely numerical implementation of the Fisher information calculation. The analytical formulation has particular advantages, in that it allows the effects to be broken down into excitatory, inhibitory, and interaction terms. The purely computational approach of course may be applied to a wider range of models.

**IV. CONCLUSION**

The current model describes how intact neurons surrounding a stroke-like cortical lesion adapt to the damage and to the resulting functional impairment. The neurons experience a disinhibition in their receptive fields and therefore their total receptive field expands in the direction of the lesion. The amount of enlargement apparent in each neuron is determined by two factors: the distance of the neuron in question from the damage and the size of the damage. Neurons close to to the damage expand their receptive field more than neurons further away. Our findings suggest that by tuning the receptive field plasticity levels to certain value, the information transfer through the cortex post stroke can be optimized. This may be of interest both for understanding the effects of potential therapeutic interventions in stroke, as well as for developing engineering principles for incorporating brain-like redundancy into machines.

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REFERENCES