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Neurocomputing 52–54 (2003) 919–924

NEUROCOMPUTING

www.elsevier.com/locate/neucom

The impact of thalamo-cortical projections on activity spread in cortex

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Abstract

To what extent does thalamo-cortical connectivity influence global patterns of macroscopical activity spread in cat cortex? We address this question by simulation of activity flow in a network model representing cortical areas and thalamic nuclei as nodes with all or none activity. The connectivity schemes employed are based on data from anatomical tracer studies. For two networks, with and without thalamic nuclei, we investigate how well physiological experiments from strychnine neuronography can be reproduced. Our results indicate that thalamic interaction does influence cortical activity spread. However, while the relation between physiological activity spread and anatomy of the cortico–cortical connectivity is significant, the relation between activity spread and the anatomy of cortico–thalamic connections is not.

Keywords: Activity spread; Cortex; Thalamus

1. Introduction

The systematic collation of data from in vivo tracer techniques [3] reveals networks between cortical areas as well as between cortical areas and thalamic nuclei of the cat cerebral hemisphere. In an earlier study we used information about the cortico–cortical networks in a simulation study to reveal mechanisms of cortical activity spread [1]. Here we extend our earlier approach by including thalamic nuclei. Our question is how the addition of the thalamo–cortical loops influences the cortical spread of

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0925-2312/03/$ - see front matter
doi:10.1016/S0925-2312(02)00837-8

activity. To this end we examined the spread of activity by simulation experiments in two different networks. One network contained cortical areas and thalamic nuclei, the other contained just the cortical areas. We compared simulation outcomes to electrophysiologically ascertain topographic activation patterns that were induced by stimulation of the corresponding cortical regions.

2. Empirical data

2.1. The connectivity data

We used a collation of 823 connections between 53 cortical areas (called C) and 651 connections between the cortical areas and 42 thalamic nuclei (called C–T) [3]. The connection strengths were graded. Connections reported as dense or strong were given a weighting of 3. Connections of intermediate strength or for which no strength information was available were weighted as 2. Connections reported as weak or sparse were weighted as 1. Connections to thalamic nuclei were assumed to be reciprocal and of symmetric weight.

2.2. The electrophysiological data

To assess effects of connectivity on the global activity spread we compared the simulation results with relevant physiological findings. We used data from strychnine sulfate neuronographic analysis in cat [2]. Saturated strychnine sulfate was applied to small patches of cortex and induced stable and reproducible patterns of cortical activity. These patterns were recorded with bipolar electrodes in a total of 18 anesthetized cats. The activity resulting from stimulation of a single cortical area was mapped topographically marking active and silent areas. The remainder of the areas had not been explored or showed variable activity. In [2] 15 different areas have been strychninized. Due to difficulties with transfering areas in the parcellation of [3] we used 11 experiments out of 15.

3. The simulation model

We devised a minimal model for describing activation of cortical areas and thalamic nuclei. Each model unit describes the activity of one area or nucleus as "active" or "silent" by a binary variable. Thus, activity was represented by a binary pattern $x \in \{0,1\}^n$ with the number of areas and nuclei $n = 53$ or 95 depending on which connectivity model was used. A connection from area i to area j is denoted s_{ij}. The model prescribed that an area or nucleus was active in the next time step $(t+1)$ if the sum of its activating inputs exceeded a certain threshold Θ.

$$x(t+1)_j = H\left[\sum_{i=1}^{n} w_{ij} x(t)_i - \Theta\right], \tag{1}$$

where Θ is the activation threshold of a unit and $H[x]$ is the Heaviside function, w_{ij} denotes the coupling strength from unit i to j and $x(t)_i$ is the activity of the respective afferent unit. Simulation started with an initial activity pattern $x(0)$ containing the strychninized area of the corresponding experiment described in [2] as a single active unit. The coupling strength was aligned to the anatomical connection strength:

$$w_{ij} = (s_{ij})^{\gamma}, \tag{2}$$

γ and the values $w_{ii} = w_D$ for excitatory self-feedback were adjusted for the best results on average over all experiments. The threshold was controlled in a way that activity in the network spread as fast as possible from the single stimulated area to a set of a activated areas/nuclei, where a is chosen according to the individual experiment, i.e.

$$\Theta(t+1) = \max\{\Theta\colon\ \min\{|x(t+1) - a|\}\}. \tag{3}$$

After activity a is reached the iteration is continued until a fixed point or cycle is reached. Possible error types are "miss" errors if the simulation results in silent areas that have been reported to be active, and "add" errors if activity is found in areas reported to be silent. Areas with unknown or variable activity were ignored in the error statistics.

To judge the influence of a systematic connectivity structure like traced connectivity on the experimental outcome, we need to compare simulations based on the systematic connectivity with those based on random connectivity with same density and grading distributions. Therefore, we studied random connectivity models, for each comparison we run simulations with 20 random matrices. In the following the random model corresponding to the cortex is denoted rnd(C), and the random model corresponding to the cortico–thalamic network rnd(C–T). In a third random model, called C-rnd(T), we studied the influence of a randomization of thalamic connections only. To this end 20 matrices where generated with experimentally observed cortico–cortical connections but random connections to thalamic nuclei.

4. Results

The parameters γ and w_D in our model were adjusted for smallest errors on average over the 11 simulated experiments corresponding to different stimulation sites. Table 1 shows the optimized values and the resulting average errors.

The error percentages achieved by the two connectivity models for each stimulated area is shown in Fig. 1(a).

Table 1
Average errors of models with experimentally traced connections and optimized parameters

Model	ω_{ii}	γ	add %	miss %	error %
C	25	2.9	10.59	14.49	12.54
C–T	23	2.9	10.71	8.71	9.71

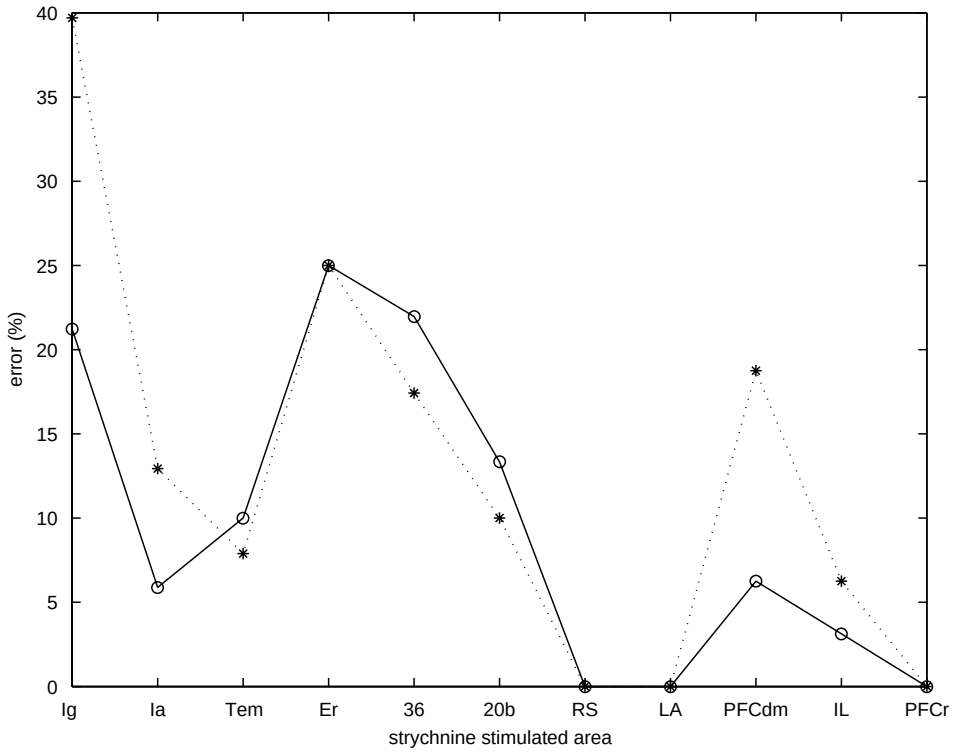

Fig. 1. (a) -○- C–T; · · ∗ · · C, (b) -○- C–T; -∗- C-rnd(T); · · + · · s.d. Errors obtained in simulations of activity spread using different connectivity models (s.d. = ±1 standard deviation).

In four experiments the error percentages were lower with thalamic nuclei and in three without connections to the thalamus. However, on average across all experiments the model including cortico–thalamic connections performed better. We controlled simulations for chance results. The random models rnd(C) and rnd(C–T) produced larger mean errors in replicating the electrophysical experiments than the "real" topographies, but had smaller standard deviations. Some "real" model simulation results were within the standard deviation range of "random" model results. Simple counting shows that the model without connections from and to thalamus has three such stimulated areas whereas the model with cortico–thalamic connections has only one result in standard deviation range (see Fig. 2a and b).

The C-rnd(T) "real/random" model produced mean errors of an order comparable to the "real" models (see Fig. 1b). We assessed mean errors obtained with "real" and "random" topographies by t-tests for equal or unequal variances depending on the outcome of an f-test. Each corresponding pair of "real" and "random" topographies showed significantly different mean errors, both "real" models compared to the mixed "real/random" C-rnd(T) model did not show these differences (Tables 2 and 3).

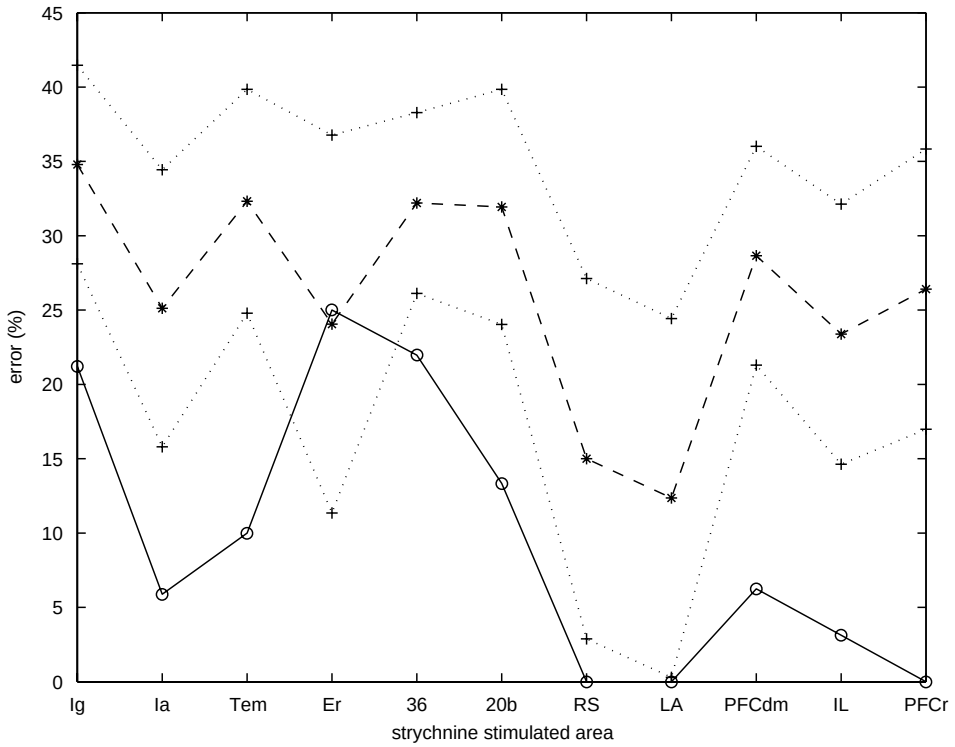

Fig. 2. (a) -○- C; -∗- rnd(C); · · + · · s. d. (b) -○- C–T; -∗- rnd(C–T); · · + · · s.d. Errors obtained in simulations of activity spread using the experimentally traced connectivity models and 20 models with random connectivities of the same density and grading (s.d. $= \pm 1$ standard deviation).

Table 2
Mean percentages and standard deviation of errors averaged across 11 experiments using different connectivity topologies

Model	Mean	s.d.
C	12.54	12.23
C–T	9.71	9.39
rnd(C)	28.08	9.32
rnd(C–T)	26.02	7.19
C-rnd(T)	12.44	10.41

Table 3
Significance level for different mean errors between the "real" and "random" topographies. In column 2 the first row was tested against rnd(C), the second against rnd(C–T)

significance level p	rnd(C)/rnd(C–T)	C-rnd(T)
C	0.003	0.984
C–T	0.000	0.526

5. Summary

We presented a simulation study to investigate the influence of thalamo–cortical projections on global pattern of activity spread in cortex. The simulation model was based on cortico–cortical and cortico–thalamic connectivity data from neuranatomical studies. We compared two connectivity models, a cortex network and a cortico–thalamic network. Both network models reproduced neuronographic experiments with good overall mean errors, the isolated cortex network with 12.5%, the cortico–thalamic network even better, with 9.7%. Systematic cortical connections always performed significantly better than random topologies with real densities, leading to errors of roughly 27%. The thalamo–cortical connections, however, do not act nearly as specifically: Networks with "real" cortico–cortical connections but random projections to thalamus perform almost as well as with the cortico–thalamic projections taken from the neuronanatomical database. Thus, our simulation experiments suggest that thalamic interactions only mildly influence the results of neuronography. The experimentally observed activity patterns are mostly determined by the cortico–cortical networks. The specific anatomy of thalamo–cortical connections has only a weak impact on activity propagation. In agreement with Scannell's analysis thalamo–cortical connections do not seem to modify the cortico–cortical organisation when tested in a simulated activity propagation paradigm.

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