The Binding Problem in Population Neurodynamics: A Network Model for Stimulus-Specific Coherent Oscillations

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Abstract. A hypothesis is presented that coherent oscillatory discharges of spatially distributed neuronal groups (the supposed binding mechanism) are the result of the convergence of stimulus-dependent activity in modality-specific afferent pathways with oscillatory activity generated in unspecific sensory systems This view is supported by simulation experiments on model networks

Key words: Neuronal groups — Coherent oscillations — Binding mechanism — Model networks

Introduction

Sensory recognition systems obviously take advantage both of serial and parallel computing mechanisms in the form of distributed hierarchical processing

Serial processing requires hierarchically organized neural networks A stimulus evoked neuronal activity entering the brain via parallel pathways with divergent projections integrates at higher levels by convergence on common neuronal pools Such morphological and functional concepts create "intelligent" neuron (grandmother cell object specific neuron, cardinal cell, pontifical cell, gnostic cell, key neuron) capable of discriminating and identifying very complex and specific stimuli

Parallel processing in divergent systems is much more difficult to define In this case the neural image of a stimulus is not represented by an "intelligent" neuron but by simultaneous neuronal activity in several specialized brain areas at different levels These frequently remote regions must be functionally coupled (the binding problem) to provide global percept and memory formation (Edelman 1987)

In addition to binding built in by genes (anatomical architecture) or built

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up by experience (activity induced, plastic changes of functional connectivity) a third type of binding performed by synchronization of oscillatory responses of the relevant neurons is suggested (Livanov 1965, 1973, Crick and Koch 1990)

In response to various stimuli subsets of neurons in involved cortical areas display coherent rhythmic firing in the gamma frequency band (30 to 70 Hz) (Eckhorn et al 1988, Gray and Singer 1989 Murphy and Fetz 1992, Laurent 1996) occurring even across the two hemispheres (Engel et al 1991) Rhythmic bursts have usually a duration of about 100 ms with 4 6 cycles of oscillation at typical frequencies at 10 60 Hz (Freeman 1994) A general theoretical problem of considerable interest is the design of efficient systems that can quickly set up coherent, phase-locked oscillations with zero phase-lag

The presented model is based on neurophysiological concept suggesting duality of function in the somatic afferent system which is composed of specific and unspecific subsystems (Mountcastle 1967) The specific subsystem is represented by nerve cells preserving information from different classes of receptors (modality, temporal characteristics and topographic arrangement between receptor regions and cortical projection areas) Such groups of cells (called feature extractors) give rise to spatially separated cortical activity The unspecific subsystem transmits via diffuse pathways projecting to vast brain stem and cortical regions convergent (multimodal heterotopic) information

It is suggested that binding between neuronal assemblies distributed over many cortical areas (formation of a "horizontal assembly") is brought about by the integration (convergence) of activity mediated by both mentioned "vertical" afferent subsystems (Fig 1 4)

Materials and Methods

Our model neuron (neuroid) JASTAP (Janco et al 1994) obeys the principles which govern the physiology of a biologically realistic neuron with chemical transmission of information

The basic element of the network is a neuroid It is described by

1) Instantaneous membrane potential (Mp) Mp is a dimensionless quantity within the $\langle -1\ 1\rangle$ range

2) Membrane potential determined as the sum of postsynaptic potentials (Psp) limited by the nonlinear function

$$Mp(t) = (2/\pi)\,\mathrm{arctg}\left(\sum Psp(t)\right) \tag{1}$$

3) A threshold (Th) from the interval $\langle 0, 1\rangle$

4) The frequency of spikes (Sp) is restricted by the absolute refractory period This is managed by setting minimum (Imn) and maximum (Imx) interspike

intervals The actual interspike interval (Ia) is determined as

$$Ia = Imn + (Imx - Imn)\,(2/\pi)\,\mathrm{arctg}\,((Mp - Th)/(1 - Mp)) \qquad (2)$$

The standard value for Imn was 1 ms, and Imx ranged from 2 ms to 10 ms The Th values used in simulations were close to 0 5

5) Behavior in terms of phasicity or tonicity (these functions have not been activated in the present work)

Every neuroid can have 8 synaptic inputs but a single output The program treats the synapse as a part of the neuroid The output can be connected to one or several synapses in the network of neuroids A synapse is characterized by

a) Input connected to it

b) Shape of a Psp prototype which is evoked by Sp arriving at this synapse (particular waveform is selected from a set of prototype Psp shapes stored in a buffer of Psp waveforms) The Psp prototype is described by

$$Psp(t) = k\,(1 - \exp(-t/t_1))^2 \exp(-2t/t_2) \qquad (3)$$

The waveform simulates whether the synapse in question is located on the soma or on the dendritic tree (the time-course and the attenuation of its amplitude) In this presentation the same Psp time-courses were used for excitatory and inhibitory Psp ($t_1 = 0\ 3$ and $t_2 = 2\ 7$ ms) with a lower amplitude for inhibitory Psp

c) Latency (time delay) of the synaptic transmission and/or axonal conduction

d) Synaptic weight (Sw) has its value from the interval $\langle -1, 1\rangle$ Sw simulates effectiveness of a synaptic input (a synchronously activated set of axons of the same type or a cluster of the terminal branches of an axon)

e) Developmental changes which determine instantaneous, effective Sw These mechanisms representing plastic activity-induced alterations were not activated in the presented simulations

The computer program JASTAP has been written in C^{++} language for IBM compatible personal computers, it runs under Windows 3 11 The program can define a network by simple command language and simulate its activity in discrete time intervals (0 5 ms steps) Results can be displayed in the form of intracellular recording with a microelectrode (Figs 3, 4), or as a raster map of the spike potentials (Fig 1B D) or saved to disk files

Results

Morphological and functional principles incorporated in presented model networks

a) Each modality of sensation depends upon information transmitted along one or more (parallel) sensory pathways with projection to one specialized (primary) projection cortical area or to more (nonprimary) cortical areas b) Each modality

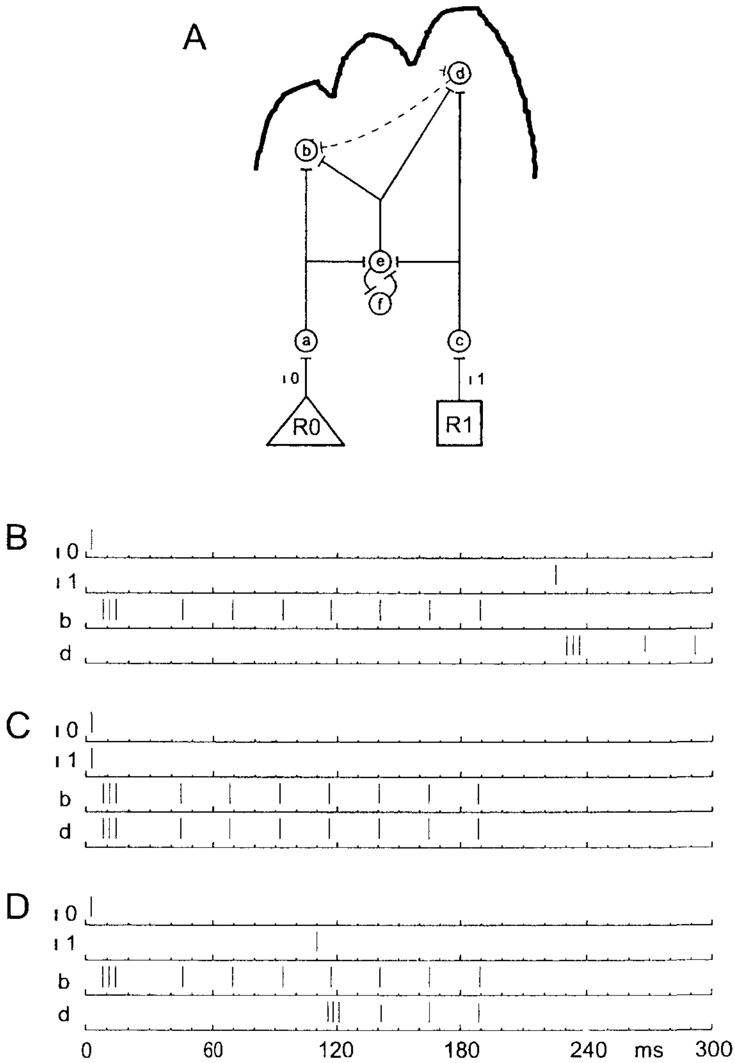

Figure 1. Coherent oscillations in spatially distributed neuronal groups. *A* Simplified schematic illustration of the sensory systems. R0 and R1 stand for two different receptor groups with their inputs (i0 and i1) into the central nervous system. The full lines connecting *a*–*b* and *c*–*d* represent afferent pathways belonging to the specific sensory systems which transmit activity to the neurons in the cortical primary projection areas (*b*, *d*). The dashed and dotted lines illustrate intracortical and transcallosal connections. Neurons in subcortical unspecific structures indicated by *e* and *f* receive convergent (heteromodal and/or heterotopic) afferent information (*a*–*e* and *c*–*e*); they have reciprocal connections (*e*–*f* and *f*–*e*) and divergent projections to neurons in cortical areas (*e*–*b* and *e*–*d*).

reaches the cerebral cortex via 1) A specific afferent system, transmitting information from one kind of sensory receptor (monomodal projection) (Fig 1 4, *RO a b* and *R1 c d*) or from a few kinds of sensory receptors (a compound tract with subdivisions for different modalities) 2) Unspecific afferent systems with convergent inputs from different kinds of receptors (multimodal and heterotopic projection) (Fig 1 4, units e and f) such systems project diffusively to subcortical brain structures and cortical areas (Fig 1 4, *RO a c b* and *d R1 c e b* and *d*) c) Specific as well as unspecific projection to cerebral cortex is not direct line, it is represented by oligosynaptic or polysynaptic pathway It can represent the site of divergent *and/or convergent projections (anatomic substrate of hierarchic organization)* and serve as a delay line (timing of sensory stimuli)

Two substantial constraints were taken into account in the proposed model network 1) Synchronizing connections (Fig 1 unit e) must not excite with suprathreshold intensity model neurons (neuroids) in the "target column" (Fig 1 4 units *b* and *d*) because in that case, coupled neuroids would respond with discharge activity to multiple spatially distributed receptive fields 2) They have to allow for quickly occurring synchronization with zero phase-lag

The approach to problem solution

The response of neuroids in projection areas (Fig 1 4 units b and d) evoked by activity in modality-specific afferent pathways (Fig 1 4 *R0 a b* and *R1 c d*) consists of an early discharge followed by sustained (above one hundred ms) subthreshold excitation The period of subthreshold excitation represents the time during which binding between projection areas can be established

The signal entering an unspecific system evokes activity in chains of neuroids organized in closed loops (Fig 1 4 units *c* and *f*) Spiking activity reverberating in loops generates rhythmic, oscillatory discharges transmitted to all projection areas (Fig 1 4 *c b* and *c d*) Oscillatory discharges excite neuroids in projection areas with subthreshold intensity

Temporal and spatial summation of the sustained excitatory influence evoked by the activity in the modality-specific pathway with rhythmic excitatory volleys generated by unspecific systems occurs and transient coalition of neuroids in different projection areas is established in the form of coherent oscillatory responses with none or minimal phase-lag The minimal phase-lag could be the result of un-

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B A raster display of the spike potentials (vertical bars) arriving via inputs i0, i1 and generated in units *b* and *d* In this case, the activation of i0 (i1) was set 2 5 ms (225 ms) after the start of the simulation *C* Simulataneous activation of i0 and i1 2 5 ms after the start of the simulation *D* Activation of i0 (i1) 2 5 ms (110 ms) after the start of the simulation See text for details even distances between oscillatory center and projection areas (Fig. 14, compare *a–b* with *c–d*).

During the binding period, the inputs to neuronal throngs involved (modality specific – Fig. 14 *a–c* and convergent – Fig. 14 *e*, *f*) are 'closed'.

The network generates regulatory commands which determine the timing of the binding period as well as the duration of other concomitant processes (e.g. inputs protection against disturbing signals). Fast oscillations and slow (regulatory) commands have the same substrate (Pavlasek 1997).

The circuitry

Figure 2 shows a model network consisting of 30 neuroids (0–29). There are two inputs (i0 and i1) representing primary afferents entering the network and conveying information from two kinds of receptors (e.g. different modalities and/or heterotopic areas). Each input has oligosynaptic projection (disynaptic in this case) via the specific afferent system (neuroids 0 and 6) to its projection area (neuroids 1

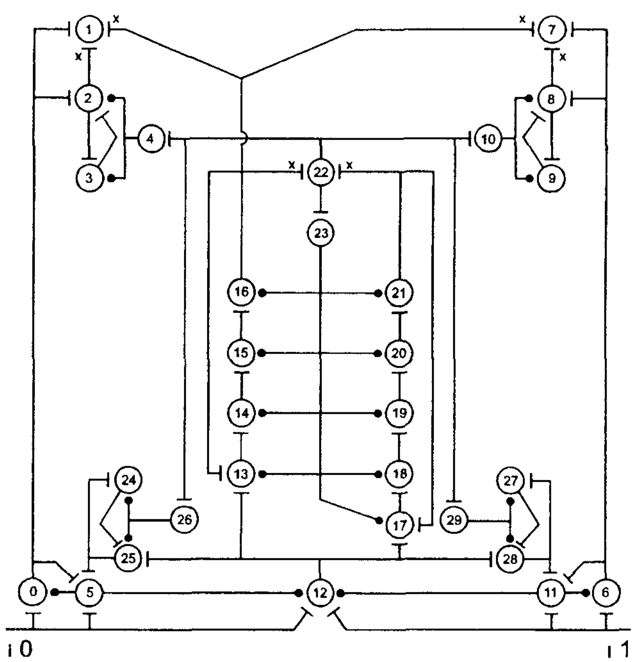


Figure 2. A model network consisting of 30 model neurons (neuroids 0–29) with two inputs (i0 and i1). Connections marked by the bars (dots) are excitatory (inhibitory); the crosses indicate subthreshold excitatory influence. Neuroids 0–6 and 1, 7 represent units *a–c* and *b–d* from Fig. 14. See text for details.

and 7) Moreover both inputs converge on neuroid 12 representing the first unit in the unspecific (multimodal, heterotopic) polysynaptic afferent system (chain of neuroids 13 16) with five synapses in this simulation which has diffuse projections to specific projection areas (neuroids 12–13 14 15 16 1 and 12 13 14 15 16 7) Two two neuroid loops (2 3 and 8 9) represent dynamic memory units maintaining information about activity in the specific afferent systems (neuroids 0 and 6) in the form of a continual train of reverberating spikes Two closed loops of distinct complexity (neuroids 13 14 15 16 13 and 17 18 19 20 21 17) are activated from the same source (neuroid 12) and then outputs are coupled by convergence on a common neuroid (22) Coupled loops represent a system producing time delay between the input (neuroid 12) and output (neuroid 22) signal (Pavlasek 1997) This system also serves as a source of oscillatory activity (generated in the loop 13 14 15 16 13) oscillatory activity is transmitted to specific projection areas (via pathways 16 1 and 16 7) The network is provided with mechanisms preventing the arrival of disturbing signals (inhibitory neuroids 5 and 11 excited by reverberating activity in two neuroid loops 24 25 and 27 28) and is equipped with inhibitory units for loop resetting (neuroids 4, 10 23 26 and 29)

Timing of activity flow

Two signals arrive simultaneously in the network via two inputs (i0 and i1) 2 5 ms after the start of simulation (Fig 1*C*) They evoke monosynaptic suprathreshold excitatory postsynaptic potentials (EPSPs) in neuroids 0 (Fig 3) and 6 simulating primary neurons in two specific (monomodal) afferent pathways Both signals entering the network set up at the same time monosynaptic suprathreshold EPSP at the level of neuroid 12 (Fig 3) simulating an input unit of an unspecific (convergent) system The decay phase of monosynaptic EPSPs in neuroids 0 and 6 is shortened by the feedforward disynaptic (i0-5 0 and i1 11 6) and feedback (recurrent) trisynaptic (i0 0 5 0 and i1 6 11 6) inhibitory influence of neuroids 5 (Fig 3) and 11 upon neuroids 0 (Fig 3) and 6 In the case of the convergent neuroid 12 the feedforward disynaptic inhibition (i0 5 12, i1 11 12) and feedback oligosynaptic inhibition (i0 12 25 5 12, i1 12 28 11 12) become effective (Fig 3) The inhibition of neuroids 0, 6 and 12 is restored and maintained by disynaptic activation (i0 12 25 and i1 12 28) of two short two-neuroid loops 24 25 (Fig 4) and 27 28 with reverberating activity exciting inhibitory neuroids 5 (Fig 3) and 11 In such a manner the inputs to specific projection areas as well as to unspecific system are 'closed' and the network is protected against disturbing afferent signals However supposing a higher intensity of stimulus (heterotopic, heteromodal) with arrival shortly delayed after oscillation has already started in some regions or considering lower effectiveness of the inhibition of the inputs to specific projection areas (Fig 2, i0 0 compared with i0 12), oscillation coherent with oscillations induced slightly earlier in other projection areas could be generated (Fig 1*D*, *b*, *d*) These timing mechanisms could codetermine whether a pattern of receptors activation is centrally processed as one complex stimulus (many inputs activated at a time) (Fig 1C b, d) or as two different stimuli (close sequence of stimuli separated by inhibitory periods) (Fig 1B, b, d)

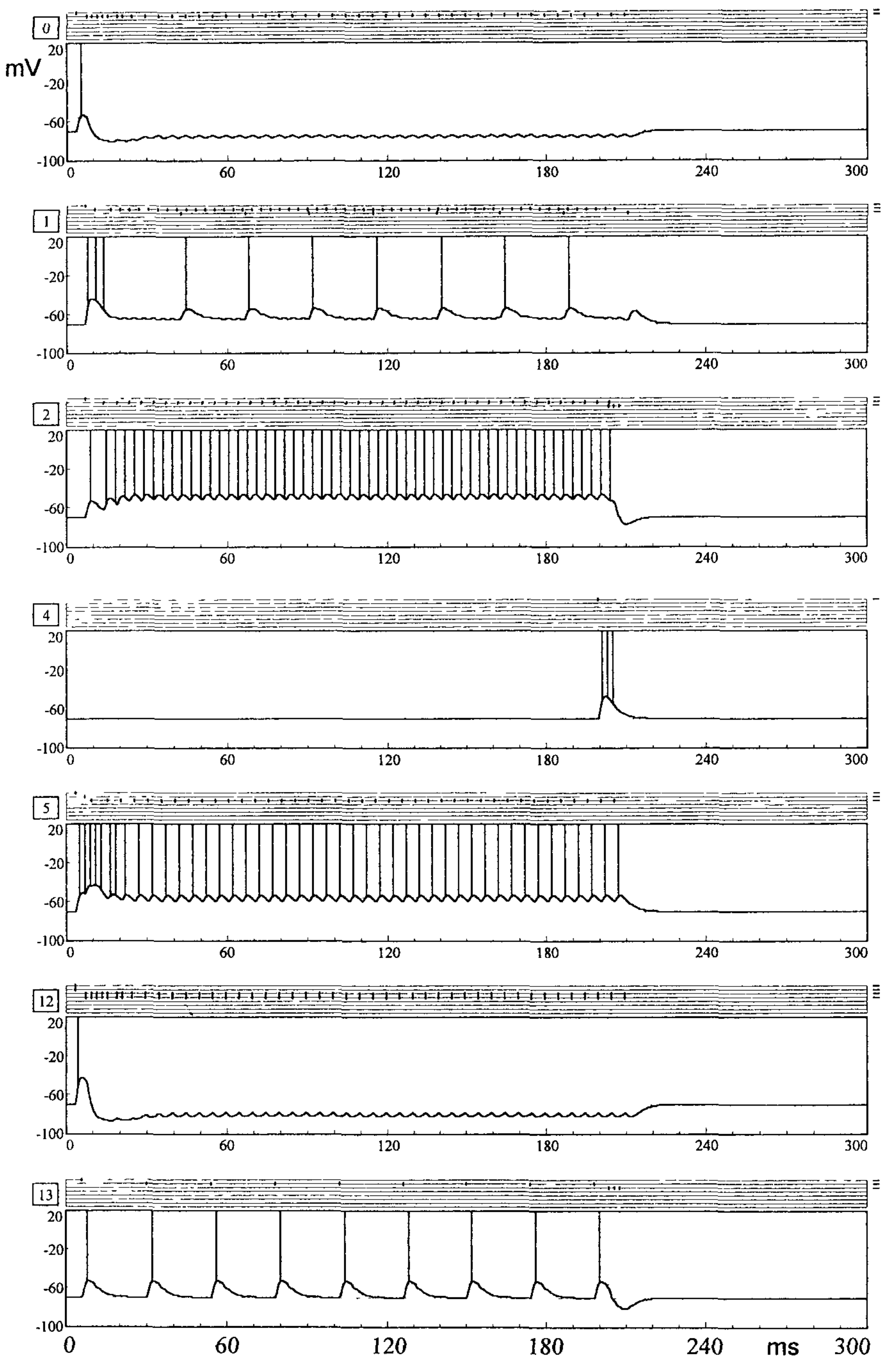
Spikes outgoing neuroids 0 and 6 evoke early monosynaptic responses of neuroids 1 (Fig 3) and 7 simulating neurons in specific (primary) projection cortical areas (Fig 1A, units b and d are represented by neuroids 1 and 7 in Fig 2)

At the same time the spike propagated in axon collateral of neuroid 0 initiates (via monosynaptic connection 0 2) reverberating activity in two neuroid loop 2 3 (Fig 3) which exerts sustained subthreshold excitatory influence on neuroid 1 (Fig 3) (synaptic connection 2 1) The spike in the axon branch of neuroid 6 evokes (via monosynaptic connection 6 8) reverberating activity in loop 8 9 which initiates sustained subthreshold excitation in neuroid 7 (synaptic connection 8 7) The aforementioned processes could have representation in early components of the evoked responses of cortical neurons which ought to be composed of an early discharge followed by subthreshold transmembrane depolarization lasting about 100 ms (Fig 3)

The activity evoked in the unspecific (convergent) afferent pathway (i0 12 and i1 12) is mediated through a polysynaptic chain (neuroids 12 13 14 15) to neuroid (16) having diffuse projections (16 1 and 16 7) to specific projection areas (neuroids 1 and 7) (Fig 2) It means that late (multisynaptic) components in the responses of neuroids 1 and 7 have a common generator The unspecific ascending system excites neuroids in specific projection areas (1 and 7) with subthreshold intensity

A characteristic structural feature of the complex neuropil of an unspecific system are recurrent or reciprocal synaptic connections (closed loops) They are simulated in the presented model network by two loops consisting of four (13 16)

Figure 3 The activity flow in the model network giving rise to coherent oscillations in spatially distributed neuroids I The results simulating intracellularly recorded postsynaptic potentials in neuroids 0 1 2 4 5 12 and 13 (Fig 2) The eight horizontal lines above the simulated recordings represent possible synaptic inputs and the small vertical bars superimposed on them indicate spikes arriving in the synaptic ending (active inputs are marked by short horizontal bars on the right hand side) The dotted horizontal lines are the threshold levels for spike (SP) generation (vertical bars on the simulated recordings) The dash-dot dot horizontal lines represent resting transmembrane potential upward (downward) deflections simulate excitatory (inhibitory) postsynaptic potentials (PSP) Abscissa simulation time in milliseconds ordinate simulation of the transmembrane potential in millivolts providing an approximate range of PSP and SP amplitudes in a biologically realistic neuron See text for details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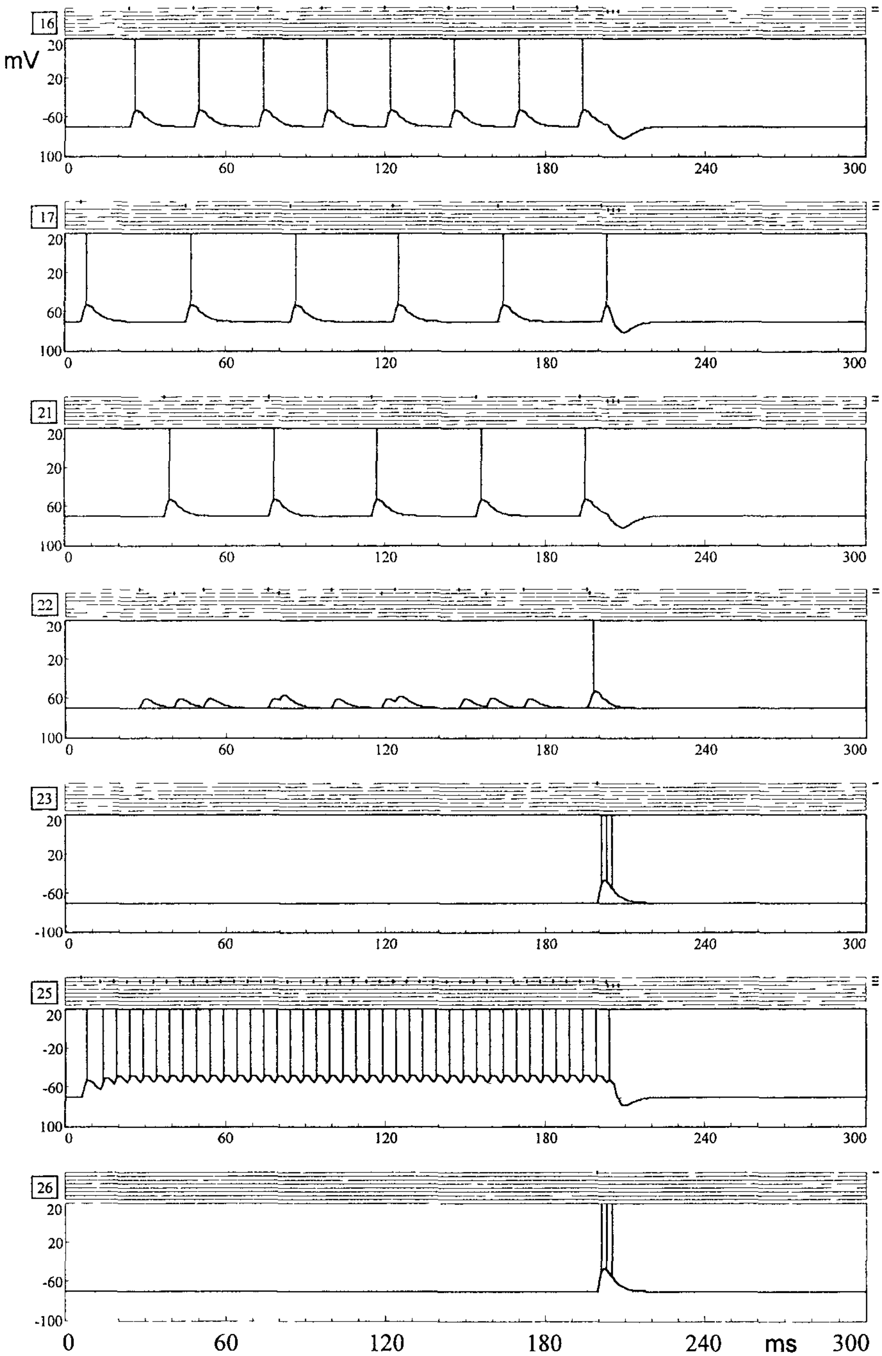
and five (17–21) neuroids (Fig. 2). The signal entering the network via neuroid 12 and activating the unspecific system evokes reverberatory activity in both loops. Their distinct complexity results in different frequencies of reverberatory spiking: simulated interspike interval of 24 ms for loop 13–16 corresponds to approximately 42 Hz (Fig. 1$B-D$ b,d; Fig. 3), the interval of 39 ms for loop 17–21 (Fig. 4) corresponds to about 26 Hz. The 24 (39) ms interval simulates synaptic transmission in a relatively short chain consisting of approximately 8 (13) biologically realistic neurons (Pavlasek and Petrovicky 1994).

Rhythmic discharges of neuroid 16 (neuroid 16 in Fig. 2 represents unit *e* from Fig. 1A) monosynaptically excite neuroids 1 and 7 (neuroids 1, 7 in Fig. 2 correspond to units *b*, *d* in Fig. 1A) with subthreshold intensity. In the simulated case signals in inputs i0 and i1 evoked reverberating activity in two neuroid loops 2–3 and 8–9 (Fig. 2) exerting sustained subthreshold excitatory influence on neuroids 1 (Fig. 3) and 7. The temporal summation of subthreshold excitatory influences from both sources occurs (Fig. 3, neuroid 1) and synchronous oscillatory discharges (frequency 42 Hz) without phase lag are generated in neuroids 1 and 7 (Fig. 1C' *b*, *d*; Fig. 3).

The outputs of two loops (neuroids 16 and 21) are coupled by convergence on a common neuroid 22 (Fig. 2). Each of them excites neuroid 22 with subthreshold intensity: the propagated response (spike) in neuroid 22 is set up only when maximal temporal summation of the EPSPs evoked by both of them occurs (Fig. 4). The input signals in i0 and i1 arrive simultaneously 2.5 ms after the start of the simulation (Fig. 1C) and propagated spike in neuroid 22 is generated 195 ms later (Fig. 4). Thus, in the presented simulation the interval of 195 ms represents the time period in which: a) Stimulus induced oscillations at the level of projection areas (Fig. 2, neuroids 1 and 7) are in progress (Fig. 3) and binding is established between them by coherent oscillation without a phase-lag (Fig. 1C, *b*, *d*); b) The inputs of the network (neuroids 0, 6 and 12) are "closed" by postsynaptic inhibition.

Neuroid 22 excites monosynaptically with suprathreshold intensity inhibitory neuroids 4, 10, 23, 26 and 29 (Figs. 2, 3, 4) and resetting of all loops occurs (Figs. 3, 4). In this way the subthreshold excitatory influences on neuroids 1 and 7 (Fig. 2) are extinguished and the hyperpolarizing shift of the membrane potential in input neuroids 0, 6 and 12 (Fig. 2) is terminated (Fig. 3), the inputs "open" for the subsequent spikes arriving in the network.

Figure 4. The activity flow in the model network giving rise to coherent oscillations in spatially distributed neuroids II. The results simulating intracellularly recorded postsynaptic potentials in neuroids 16, 17, 21, 22, 23, 25 and 26 (Fig. 2). Other symbols as in Fig. 3. See text for details.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Discussion

Physiological plausibility of the presented model

The submitted hypothesis, supported by the simulations provided with model networks suggests that a) Oscillatory cortical activity is primarily generated at the level of unspecific subcortical systems with diffuse ascending projection to primary sensory cortical areas b) Coherent oscillations with zero phase-lag in spatially separated projection areas can be quickly set up by stimulus-evoked activity transmitted in specific and unspecific systems and converging at the level of neurons in the projection areas involved In the next sections, this hypothesis will be supported by results of neurophysiological experiments

In the deep structures multimodal and heterotopic convergence was observed in the reticular formation (Scheibel et al 1955) and at the thalamic level (Albe-Fessard and Besson 1973) The reticulo-thalamic tract (running from the medial system of the reticular formation into the posterior intralaminar, and medial nucleus of the thalamus) is a part of the ascending activating system (Moruzzi and Magoun 1949, Kinomura et al 1996) Multiple cortical regions having convergent properties have been described (Amassian 1954, Albe-Fessard and Besson 1973), these can be activated by thalamo-cortical fibers originating in non-specific thalamus (reticulo-thalamo-cortical system)

The earlier components of the somatosensory evoked potentials (SEPs) observed in human brain electrophysiology possibly reflect activity in the receptor-specific and site-specific afferents of the lemniscal portion of the thalamo-cortical pathway and represent early cortical postsynaptic activity (Werner and Whitsel 1973) The late components of the SEPs (with latencies longer than about 70 ms) (Regan 1989) obviously have a common denominator as their time-courses are similar (Ciganek 1991) These "associative" responses evoked by brief somatic, visual, or auditory stimuli are vulnerable to barbiturates, frequent stimulation, and they are substantially modified by the state of wakefulness (the depressant effect of "arousal") (Segundo and Galeano 1960) All these facts indicate that the convergent cortical inflow is mediated through non-specific reticular or thalamic zones (Albe-Fessard and Besson 1973)

As was shown by intracellular recordings from cortical neurons, electric stimulation of the specific thalamic nucleus (VL) evoked in pyramidal and nonpyramidal tract neurons short-latency discharge followed by an additional synaptic depolarization of the membrane potential lasting more than 50 ms (Purpura et al 1964, Purpura 1967)

The EPSPs elicited in neocortical neurons by stimulation of nonspecific thalamocortical projections (Creutzfeldt and Lux 1964) are mediated by axodendritic synaptic contacts localized much further away from the soma of the convergent cortical cells than specific afferents (Nacimiento et al 1964) Such EPSPs are frequently of subthreshold intensity for spike generation If the subthreshold EPSP of specific origin falls on the EPSP of non-specific origin, summation of depolarization and cell firing occurs

The origin of oscillations

Cellular pacemakers (Llinas 1990) as well as emergent functional properties of the networks can be the source of oscillatory rhythms With all probability, both mechanisms coexist in the bulbar reticular formation (Segundo et al 1967) as well as in the thalamus (Steriade and Llinas 1988) The regular spontaneous discharge activity observed in thalamic neurons (Purpura and Shofer 1963) may become synchronized with the cortical neurons in a closely correlated fast rhythm (Steriade et al 1991 Barth and MacDonald 1996) The presence of oscillatory activity in the thalamus after decortication indicates that local synchrony is still maintained by intrathalamic connectivity (Contreras et al 1996) This effect could be the consequence of extensive communication between thalamo cortical and thalamic reticular cells (Crick 1984)

The morphological substrate for rhythmic firing can be sequential activity propagation in networks with a ring geometry as well as in networks with recurrent connections and/or reciprocal links (closed loops) The sensitivity of the system generating oscillatory activity in closed loops to disturbing afferent signals should be stressed (Tsutsumi and Matsumoto 1984) There exist experimental results indicating that at various levels of the unspecific sensory systems inhibitory mechanisms operate which could "guard" the inputs to the involved structures while the information processing is in progress The following has been confirmed in the ponto medullary reticular formation (Pavlasek and Pilyavskii 1981, Pavlasek and Petrovicky 1994) a) Autoinhibition of the stimulated input channel (depression of the response to the second stimulus in a twin-stimulus experimental regimen) b) A blocking interaction (in the experiments with conditioning-testing stimulus protocol) among sensory channels The mentioned inhibition (complete or partial) can last for hundreds of ms The depression of the response to the second stimulus occurs at the thalamic relay station as well as at the cortical level (Werner and Whitsel 1973) An inhibition of responses rapidly develops in the reticular formation (Pavlasek and Petrovicky 1994) and unspecific thalamus (Albe Fessard and Besson 1973) when the repetition rate of the stimuli applied to the same peripheral region is higher than 3 Hz

Direct repetitive stimulation of the reticular formation can be considered as a barrage of disturbing signals bypassing the inputs protecting mechanisms and abolishing spontaneous oscillatory activity in the unspecific subcortical structures As observed in cortical neurons, such stimulation caused disappearance of the phasic discharges which were replaced by the whole range of activation patterns rarely reaching the firing level (Skrebitsky et al 1980 Steriade et al 1980)

There are intracortical mechanisms which might play an important role in establishing local neural synchronization. A biophysically distinct subset of cortical neurons termed "chattering" cells has been reported (Gray and McCormick 1996). In response to sensory stimulus they intrinsically generate 20 to 70 Hz repetitive burst firing and thus participate in the recruitment of large populations of cells into synchronously firing assemblies. Neuroids of this type have not been included in the present model.

There are experimental observations on split-brain (Engel et al. 1991) and strabismic kittens (Lowel and Singer 1992) indicating a cortico-cortical mechanism of synchronization. Simulation studies demonstrate that intracortical mechanisms might generate coherent oscillations over large distances without phase lag despite variable conduction delays in the synchronizing intracortical connections (Konig and Schillen 1991; Traub et al. 1996). According to these results, synchronization with zero phase-lag can be achieved without common input.

Other authors tend to suppose that the interareal synchrony is not attained within cortical circuits (cortico-cortical connections); such an opinion is supported by the fact that a deep cut through the cortex does not extinguish coherent oscillations recorded with cortical electrodes placed on the opposite sites of the lesion (Livanov 1989; Contreras et al. 1996). Transsection of subcortico-cortical afferents abolishes coherent oscillations in the involved cortical region (Livanov 1989); this result points to the crucial role of subcortical structures in generating synchronized activity of cortical cells. Moreover, results of theoretical works support the view that long-distance synchronization with zero phase-lag is indicative of common input (Gerstein and Perkel 1972).

Therefore the oscillatory activity in unspecific subcortical center(s) (brainstem reticular formation and thalamus) seems to be a proper candidate for setting up coherent cortical oscillations. The generation, stabilization, selection and focusing of synchronous thalamo-cortical oscillations depend on feedback projections from cortical regions to nearly all thalamic nuclei and on mechanism of lateral inhibition (Contreras et al. 1996; Kral and Majernik 1996). Supposing subthreshold excitatory influence of unspecific thalamic cells on cortical neurons, such a stream of activity could be instrumental for putting together all neuronal groups (feature extractors) simultaneously responding to specific afferentation.

The suggested mechanism has the following characteristics: a) It enables rapid establishing of transitory (Freeman 1988; Gray et al. 1992) functional relationships between cell groups lacking direct reciprocal connections. b) Its substantial attribute is a combinatorial flexibility. c) It can reflect the feature constellations of the stimulus. d) The topographic projections or preservation of metric proportionality is not an imperative condition.

Just questions

The rhythms in the brain and especially coherent oscillations as a temporal code could under the fundamental brain functions connected with information processing and consciousness. The following ideas are hotly debated (Crick 1984; Gray et al. 1989; Crick and Koch 1990; Singer et al. 1990; Singer 1993; Schillen and König 1994): binding mechanisms, global stimulus perception, selection of functionally coherent neuronal ensembles, long term modifications of synaptic efficacy and re-ordering of functional connectivity, structuring and timing of the activity flow, working memory, non-local information storage in the anatomical space, the basis for dynamic processes in nonlinear systems, visual awareness.

The presented speculative hypothesis suggests a specific role for unspecific systems in the mechanisms enabling different groups of oscillating neurons to fall into step across large distances.

All of these concepts, plausible though they may be, must be regarded as speculative until supported by much stronger neurophysiological experimental evidence and assessed at the behavioral level. The reduced but not oversimplified neural network models could have a major impact on them thereby providing a logical framework and suggesting solutions.

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References

Albe-Fessard D., Besson J. M. (1973): Convergent thalamic and cortical projections – the nonspecific system. In: Handbook of Sensory Physiology, Vol. II, Somatosensory System (Ed. A. Iggo), pp. 489—560, Springer Verlag, Berlin, Heidelberg, New York

Amassian V. E. (1954): Studies on organization of a somesthetic association area including a single unit analysis. J. Neurophysiol. **17**, 39—58

Barth D. S., MacDonald K. D. (1996): Thalamic modulation of high frequency oscillating potentials in auditory cortex. Nature **383**, 78—81

Ciganek L. (1991): Evoked Potentials and Their Application in Clinical Praxis. Osveta, Martin (in Slovak)

Contreras D., Destexhe A., Terrence J., Sejnowski J., Steriade M. (1996): Control of spatiotemporal coherence of a thalamic oscillation by corticothalamic feedback. Science **274**, 771—774

Creutzfeldt O. D., Lux H. D. (1964): Zur Unterscheidung von specifischen und unspecifischen Synapsen an corticalen Nervenzellen. Naturwiss. **51**, 89—90

Crick F. (1984): Function of the thalamic reticular complex: The searchlight hypothesis. Proc. Natl. Acad. Sci. USA **81**, 4586—4590

Crick F., Koch C. (1990): Some reflections on visual awareness. In: The Brain. Cold Spring Harbor Symposia on Quantitative Biology, Vol. LV, pp. 953—962, Cold Spring Harbor Laboratory Press, New York

Eckhorn R., Bauer R., Jordan W., Brosch M., Kruse W., Munk M., Reitboeck H. J. (1988) Coherent oscillations: A mechanism of feature linking in the visual cortex? Biol. Cybern. **60,** 121—130

Edelman G. M. (1987). Neural Darwinism. The Theory of Neuronal Group Selection Basis Books, New York

Engel A. K., König P., Kreiter A., Singer W. (1991): Interhemispheric synchronization of oscillatory neuronal responses in cat visual cortex Science **252,** 1177—1179

Freeman W. J. (1988): Nonlinear neural dynamics in olfaction as a model for cognition In: Dynamics of Sensory and Cognitive Processing by the Brain (Ed. E. Basar) pp 19—29, Springer, Berlin

Freeman W. J. (1994): Qualitative overview of population neurodynamics In: Neural Modeling and Neural Networks (Ed. F. Ventriglia) pp. 185—215, Pergamon Press, Oxford

Gerstein G. L., Perkel D. H. (1972). Mutual temporal relationship among neuronal spike trains Statistical techniques for display and analysis. Biophys. J. **12,** 453—473

Gray C. M., McCormick D. A. (1996) Chattering cells Superficial pyramidal neurons contributing to the generation of synchronous oscillations in the visual cortex Science **274,** 109—113

Gray C. M., Singer W. (1989). Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. Proc. Natl. Acad. Sci. USA **86,** 1698—1702

Gray C. M., Konig P., Engel A. K., Singer W. (1989) Oscillatory responses in cat visual cortex exhibit inter-columnar synchronization which reflects global stimulus properties Nature **338,** 334—337

Gray C. M. Engel A. K., König P., Singer W. (1992). Synchronization of oscillatory neuronal responses in cat striate cortex temporal properties. Visual Neurosci. **8,** 337—347

Jančo J., Stavrovský I., Pavlásek J. (1994): Modeling of neuronal functions A neuron-like element with the graded response. Comput. Artiff. Intell. **13,** 603—620

Kinomura S., Larsson J., Gulyás B., Roland P. E. (1996): Activation by attention of the human reticular formation and thalamic intralaminar nuclei. Science **271,** 512—515

König P., Schillen T. B. (1991): Stimulus-dependent assembly formation of oscillatory responses I. synchronization. Neural Comp. **3,** 155—166

Král A., Majerník V. (1996). On lateral inhibition in the auditory system Gen. Physiol. Biophys. **15,** 109—127

Laurent G. (1996). Dynamical representation of odors by oscillating and evolving neural assemblies Trends Neurosci. **19,** 489—496

Livanov M. N. (1965). The role of distant synchronization of cortical biopotentials in realization of temporary connections. Intern. Congr. Physiol. Sci., 23rd, Tokyo, p 600

Livanov M. N. (1973) Spatial Synchronization of Cerebral Biopotentials Nauka, Moscow (in Russian)

Livanov M. N. (1989): Spatio-Temporal Potential Organization and Systemic Brain Activity Nauka, Moscow (in Russian)

Llinás R. (1990): Intrinsic electrical properties of nerve cells and their role in network oscillation In. The Brain. Cold Spring Harbor Symposia on Quantitative Biology, Vol LV, pp. 933—938, Cold Spring Harbor Laboratory Press, New York

Lowel S Singer W (1992) Selection of intrinsic horizontal connections in the visual cortex by correlated neuronal activity Science **255,** 209—212

Moruzzi G , Magoun H W (1949) Brain stem reticular formation and activation of the EEG Electroencephalogr Clin Neurophysiol **1,** 445—473

Mountcastle V B (1967) The problem of sensing and the neural coding of sensory events In The Neurosciences (Eds G C Quarton T Melnechuk, F O Schmitt) pp 393—408, The Rockefeller University Press New York

Murphy V N , Fetz E E (1992) Coherent 25— to 35-Hz oscillations in the sensorimotor cortex of awake behaving monkeys Proc Natl Acad Sci USA **89,** 5670—5674

Nacimiento A C , Lux H D , Creutzfeld O D (1964) Postsynaptische Potenziale an Nervenzellen des motorischen Cortex nach elektrischer Reizung spezifischer und unspezifischer Thalamuskerne Pflugers Arch ges Physiol **281,** 152—169

Pavlasek J (1997) Timing of neural commands a model study with neuronal networks Biol Cybern **77,** 359—365

Pavlasek J Petrovicky P (1994) The Reticular Formation and the Reticulo-Spinal System Veda Publishing House of the Slovak Academy of Sciences Bratislava

Pavlasek J Pilyavskii A I (1981) The reduced responsiveness of reticulospinal neurons to paired stimuli in cats Physiol Bohemoslov **30,** 221 230

Purpura D P (1967) Comparative physiology of dendrites In The Neurosciences (Eds G C Quarton, T Melnechuk, F O Schmitt), pp 372 393, The Rockefeller University Press, New York

Purpura D P Shofer R J (1963) Intracellular recording from thalamic neurons during reticulocortical activation J Neurophysiol **26,** 494— 505

Purpura D P Shofer R J Musgrave F S (1964) Cortical intracellular potentials during augmenting and recruiting responses II Patterns of synaptic activities in pyramidal and nonpyramidal tract neurons J Neurophysiol **27,** 133 151

Regan D (1989) Human Brain Electrophysiology Elsevier, New York, Amsterdam, London

Scheibel M Scheibel A Mollica A , Moruzzi G (1955) Convergence and interaction of afferent impulses on single units of reticular formation J Neurophysiol **18,** 309 332

Schillen T B Konig P (1994) Binding by temporal structure in multiple feature domain of an oscillatory neuronal network Biol Cybern **70,** 397—405

Segundo J P , Galeano C (1960) Somatic functions of the nervous system Annu Rev Physiol **22,** 433—472

Segundo J P , Takenaka T , Encabo H (1967) Electrophysiology of bulbar reticular neurons J Neurophysiol **30,** 1194—1220

Singer W (1993) Synchronization of cortical activity and its putative role in information processing and learning Annu Rev Physiol **55,** 349—374

Singer W , Gray C , Engel A , Konig P , Artola A , Brocher S (1990) Formation of cortical cell assemblies In The Brain Cold Spring Harbor Symposia on Quantitative Biology, Vol LV pp 939—952, Cold Spring Harbor Laboratory Press, New York

Skrebitsky V G , Chepkova A N , Sharonova I N (1980) Reticular suppression of cortical inhibitory postsynaptic potentials In The Reticular Formation Revisited (Eds J A Hobson, M A B Brazier), pp 117—124, Raven Press, New York

Steriade M , Llinás R R (1988) The functional state of the thalamus and the associated neuronal interplay Physiol Rev **68,** 649—742

Steriade M Ropert N , Kitsikis A , Oakson G (1980) Ascending activating neuronal networks in midbrain reticular core and related rostral systems In The Reticular Formation Revisited (Eds J A Hobson M A B Brazier) pp 125 167 Raven Press New York

Steriade M , Curro-Dossi R Pareacute D Oakson G (1991) Fast oscillations (20 40 Hz) in thalamocortical systems and their potentiation by mesopontine cholinergic nuclei in the cat Proc Natl Acad Sci USA **88,** 4396– 4400

Traub R D Whittington M A , Stanford I A , Jefferys J G R (1996) A mechanism for generation of long-range synchronous fast oscillations in the cortex Nature **383,** 621 624

Tsutsumi K Matsumoto H (1984) Ring neural network qua a generator of rhythmic oscillations with period control mechanism Biol Cybern **51,** 181 –194

Werner G Whitsel B L (1973) Functional organization of the somatosensory cortex In Handbook of Sensory Physiology Vol II Somatosensory System (Ed A Iggo) pp 621 700 Springer-Verlag Berlin Heidelberg New York

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