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
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 (Livianov 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, Grav 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 ($M_p$) $M_p$ is a dimensionless quantity within the $(-1, 1)$ range.

2) Membrane potential determined as the sum of postsynaptic potentials ($P_{sp}$) limited by the nonlinear function

$$M_p(t) = \frac{2}{\pi} \arctg \left( \sum P_{sp}(t) \right)$$

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

4) The frequency of spikes ($Sp$) is restricted by the absolute refractory period. This is managed by setting minimum ($Imm$) and maximum ($Imx$) interspike
intervals. The actual interspike interval ($I_a$) is determined as

$$I_a = I_m + (I_{mx} - I_m) (2/\pi) \arctg((M_p - Th)/(1 - M_p))$$

The standard value for $I_m$ was 1 ms, and $I_{mx}$ 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 neuron can have 8 synaptic inputs but a single output. The program treats the synapse as a part of the neuron. The output can be connected to one or several synapses in the network of neurons. 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

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

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 ($S_w$) has its value from the interval $(-1,1)$. $S_w$ 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 $S_w$. 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. RO 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, c, and e, c) they have reciprocal connections (e, f, and f, c) and divergent projections to neurons in cortical areas (c, 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 14, RO a b and R1 e 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 14, units e and f) such systems project diffusively to subcortical brain structures and cortical areas (Fig 14, RO a c b and d R1 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 hierarchical 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 (neurond) in the ‘target column’ (Fig 14 units b and d) because in that case, coupled neurons 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 neuronds in projection areas (Fig 14 units b and d) evoked by activity in modality-specific afferent pathways (Fig 14 RO 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 neuronds organized in closed loops (Fig 14 units e and f) Spiking activity reverberating in loops generates rhythmic oscillatory discharges transmitted to all projection areas (Fig 14 c b and e d) Oscillatory discharges excite neuronds 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 neuronds 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-

B A raster display of the spike potentials (vertical bars) arriving via inputs iO, 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 Simultaneous 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 e, 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 neurons (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 (neurons 0 and 6) to its projection area (neurons 1

![Figure 2](image-url) - 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. Neurons 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 neuron 12 representing the first unit in the unspecific (multimodal, heterotopic) polysynaptic afferent system (chain of neurons 13-16) with five synapses in this simulation which has diffuse projections to specific projection areas (neurons 12-13 14 15 16 1 and 12 13 14 15 16 7). Two two-neruroid loops (2 3 and 8 9) represent dynamic memory units maintaining information about activity in the specific afferent systems (neurons 0 and 6) in the form of a continual train of reverberating spikes. Two closed loops of distinct complexity (neurons 13 14 15 16 13 and 17 18 19 20 21 17) are activated from the same source (neuron 12) and their outputs are coupled by convergence on a common neuron (22). Coupled loops represent a system producing time delay between the input (neuron 12) and output (neuron 22) signal (Pavlošek 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 neurons 5 and 11 excited by reverberating activity in two neuron loops 24 25 and 27 28) and is equipped with inhibitory units for loop resetting (neurons 4, 10, 23, 26, 29).

**Tuning of activity flow**

Two signals arrive simultaneously in the network via two inputs (10 and 11) 2.5 ms after the start of simulation (Fig 1C). They evoke monosynaptic suprathreshold excitatory postsynaptic potentials (EPSPs) in neurons 0 (Fig 3) and 6 simulating primary neurons in two specific (monomodal) afferent pathways. Both signals enter the network set up at the same time monosynaptic suprathreshold EPSP at the level of neuron 12 (Fig 3) simulating an input unit of an unspecific (convergent) system. The decay phase of monosynaptic EPSPs in neurons 0 and 6 is shortened by the feedforward disynaptic (10-5 0 and 11 11 6) and feedback (recurrent) triphasic (10 0 5 0 and 11 6 11 6) inhibitory influence of neurons 5 (Fig 3) and 11 upon neurons 0 (Fig 3) and 6. In the case of the convergent neuron 12 the feedforward disynaptic inhibition (10 5 12, 11 11 12) and feedback oligosynaptic inhibition (10 12 25 5 12, 11 12 28 11 12) become effective (Fig 3). The inhibition of neurons 0, 6 and 12 is restored and maintained by disynaptic activation (10 12 25 and 11 12 28) of two short two-neruroid loops 24 25 (Fig 4) and 27 28 with reverberating activity exciting inhibitory neurons 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 slightly 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, 10 0 compared with 10 12), oscillation coherent with oscillations induced slightly earlier in other projection areas could be generated (Fig 1D, b, d). These
timing mechanisms could co-determine 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 neurords 0 and 6 evoke early monosynaptic responses of neuronds 1 (Fig 3) and 7 simulating neurons in specific (primary) projection cortical areas (Fig. 1A, units b and d are represented by neuronds 1 and 7 in Fig. 2)

At the same time, the spike propagated in axon collateral of neurond 0 initiates (via monosynaptic connection 0 2) reverberating activity in two neurond loop 2-3 (Fig 3) which exerts sustained subthreshold excitatory influence on neurond 1 (Fig 3) (synaptic connection 2 1). The spike in the axon branch of neurond 6 evokes (via monosynaptic connection 6 8) reverberating activity in loop 8-9 which initiates sustained subthreshold excitation in neurond 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 (10 12 and 11 12) is mediated through a polysynaptic chain (neuronds 12 13 14 15) to neurond (16) having diffuse projections (16 1 and 16 7) to specific projection areas (neuronds 1 and 7) (Fig 2). It means that late (multisynaptic) components in the responses of neuronds 1 and 7 have a common generator. The unspecific ascending system excites neuronds 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 neuronds. The results simulating intracellularly recorded postsynaptic potentials in neuronds 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.
and five (17-21) neurords (Fig. 2). The signal entering the network via neurord 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. 1B - 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 neurord 16 (neurord 16 in Fig. 2 represents unit e from Fig. 1C) monosynaptically excite neurords 1 and 7 (neurords 1, 7 in Fig. 2 correspond to units b, d in Fig. 1C) with subthreshold intensity. In the simulated case signals in inputs i0 and i1 evoked reverberating activity in two neurord loops 2-3 and 8-9 (Fig. 2) exciting sustained subthreshold excitatory influence on neurords 1 (Fig. 3) and 7. The temporal summation of subthreshold excitatory influences from both sources occurs (Fig. 3 neurord 1) and synchronous oscillatory discharges (frequency 42 Hz) without phase lag are generated in neurords 1 and 7 (Fig. 1C b,d Fig. 3).

The outputs of two loops (neurords 16 and 21) are coupled by convergence on a common neurord 22 (Fig. 2). Each of them excites neurord 22 with subthreshold intensity. The propagated response (spike) in neurord 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 neurord 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 neurords 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 (neurords 0, 6 and 12) are “closed” by postsynaptic inhibition.

Neurord 22 excites monosynaptically with suprathreshold intensity inhibitory neurords 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 neurords 1 and 7 (Fig. 2) are extinguished and the hyperpolarizing shift of the membrane potential in input neurords 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 neurords II. The results simulating intracellularly recorded postsynaptic potentials in neurords 16, 17, 21, 22, 23, 25 and 26 (Fig. 2). Other symbols as in Fig. 3. See text for details.
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 (Amanzian 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 fre-
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Quently 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 Pilyavskin 1981, Pavlasek and Petrovicky 1994):

- a) Autoshutdown 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 (Wernicke and Whitsell 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. Neurons 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 (Kong 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, Contreas et al. 1996). Transection 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 (Contreas 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 combinational 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.
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Just questions

The rhythms in the brain and especially coherent oscillations as a temporal code could underlie 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 Kung 1994) binding mechanisms, global stimulus perception, selection of functionally coherent neuronal ensembles, long-term modifications of synaptic efficacy and reordering of functional connectivity, structuring and tuning 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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