The dynamic spatio-temporal behavior of visual responses in thalamus and cortex

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Abstract: Due to eye and object movements the visual world changes on a rather fast time scale and the neuronal network of the primary visual pathway has to immediately react to these changes. Accordingly the neuronal activity patterns in the visual thalamus and cortex show a pronounced dynamic behavior which reenters the circuitry such that the actual cell responses are also guided by the activation history of the network. Thus, spatial and temporal aspects of visual receptive fields change not only by means of the actual visual stimulation but also as a consequence of the state of the network. In this short review we will try to summarize the different aspects which can influence the *temporal* firing patterns of cells in the visual thalamus (lateral geniculate nucleus, LGN) mainly by demonstrating how their inter-spike interval distributions will change. We will then show, how these firing patterns are able to change the *spatial* shape of receptive fields in the visual cortex (see Fig.12 for a summary diagram). Finally, by means of a biophysical model, we will argue that the observed changes could serve to adjust the temporal and spatial resolution within the primary visual pathway to the different demands for information processing in an attentive as compared to a non-attentive state.

Introduction

The primary visual pathway consists of three substructures: retina, lateral geniculate nucleus, which is a part of the thalamus, and the visual cortex with its many different areas. All these structures are connected by afferent fibers and the hierarchical arrangement, which dominates the first levels, is given up at the level of the cortex, where it is replaced by a widely branching parallel connectivity. Apart from the retina, rich feedback connections exist between cortex and thalamus as well as lateral connections between the different cortical areas. These anatomical observations, for which strong evidence accumulated not later than around 1970 [6,57,61,72], indicate that visual information processing must be a process utilizing recurrent loops and involving massive dynamic interactions. It is thus puzzling that visual cortical receptive fields were for a very long time regarded as rather static entities entirely governed by spatial influences [27]. Accordingly, receptive fields were initially described only by their spatial substructure. Cortical fields, for example, are usually elongated and consist (in simple cells) of several adjacent subfields with antagonistic ON- and OFF-characteristic. It was not before 1980 that very slowly a shift of paradigm was introduced in visual neurophysiology which led to the realization that visual responses and receptive fields at all levels are highly dynamic processes [see refs. 2,11]. Dynamic changes of the spatial structure of receptive fields have been described in different contexts: for plastic changes after peripheral or cortical lesions [15,22], for repetitive co-stimulation of border regions of the receptive field [14] and for the fast state-dependent changes [76] described below in more detail. These processes occur at very different time scales. They include normal and pathological aspects of cortical physiology it is possible that they share similar or identical mechanisms. The brain is not static, not even in adulthood, and lesion-induced and learning-related dynamic processes appear to be very similar.

Another important impetus for investigating temporal dynamics came from observations that the firing of a cell needs in many cases a better descriptor than just the mean impulse rate [12,24,69]. The intrinsic temporal structure of the firing pattern, together with the mean firing rate, is required to describe the behavior of such cells more accurately [11]. Thus, receptive fields have not only a spatial but also a temporal substructure. In addition, processes were observed, some of which will be described to a greater detail in this review, which showed that the (temporal and spatial) receptive field structure can change strongly on quite different time scales. On the one hand, it became evident that the activation history of the network from the last 100 milliseconds or so can exert a strong influence on the momentarily expected response [2,41]. Furthermore, it was demonstrated that long term processes (long-term-potentiation, plasticity, etc.) can influence a receptive field quite strongly [10,14,22,55].

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In this article we will show examples of temporal response characteristics of visual neurons which cannot be described merely by using the mean impulse rate and we will discuss their possible influence on visual information processing. Then, we will show that the response characteristics of thalamic and cortical cells can change rather strongly in a way correlated with the global state of the brain as measured by electroencephalographic activity (EEG). Here, we will compare changes of the temporal response pattern of thalamic cells with those of the mean impulse rate and also with spatial changes of cortical response fields (receptive fields). We will then continue with a model that can explain the *spatial* restructuring of cortical receptive fields on the basis of changes of the *temporal* characteristics of thalamic responses. Finally, we will discuss the possible functional importance of these fast changes in the temporal and spatial aspects of visual activity in the context of bottom-up (pre-)attentive processes. The main conclusions are: i) Depending on the state of arousal (or attention) visual information is passed through the thalamus in different ways: detailed information about the spatial distribution of light contrast is transmitted by a temporally structured tonic (single spike) activity in the aroused state. In a less aroused state, tonic inhibition of LGN relay cells by PGN cells eliminates the information about steady contrast distribution included in tonic firing and allows only for strong responses elicited by sudden changes in contrast. The generation of spike bursts by the so called low-threshold calcium spike in the LGN reinforces this process. ii) The changed temporal pattern of LGN spike activity has further consequences for cortical processing: cortex cells receive convergent input from many LGN cells and the efficiency of a single input in eliciting a cortical spike seems to be small. However, the high frequency spike bursts of LGN cells may be strong enough even at a single synapse located peripherally within the receptive field or dendritic tree of the cortex cell. The result is a large receptive field, sensitive for small but sudden changes in contrast. On the other hand, tonic single spike activity has to sum up with other coincident inputs to become supra-threshold, a way to make the receptive field highly specific for spatial combinations of temporally structured inputs.
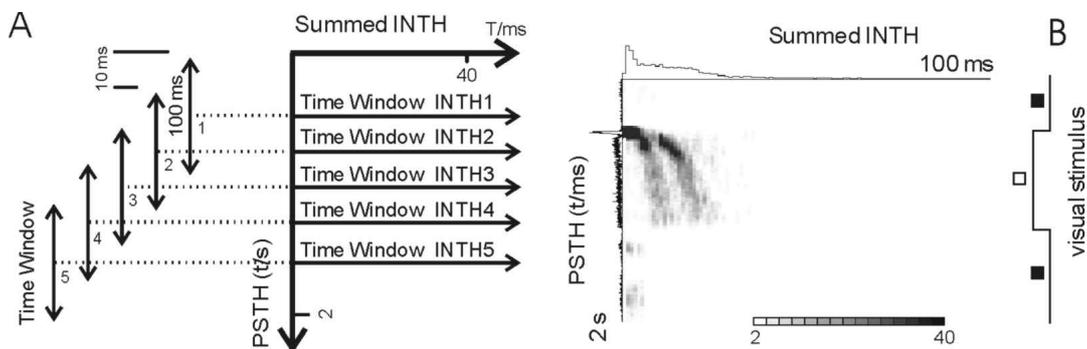
Fig.1 Procedure to generate an intervalogram. (A) A PSTH is plotted downward and a regular summed INTH on top of the diagram. A time window of 100 ms size is moved in 10 ms steps along the time axis and the interval distribution is determined for every step and plotted - using a gray-scaling for the number of intervals - to the right in the diagram. This way a temporally resolved INTH – an intervalogram – is created. (B) Example of an intervalogram for an LGN X-On cell response to a spot of light (1 deg visual angle in diameter, 90% contrast) flashed within the center of its receptive field. The stimulus protocol is shown to the right. Two bands of preferred inter-spike intervals are clearly visible, which cannot be seen in the summed INTH on top. Modified from [20].

Time structure in thalamic relay cell responses.
Cells in the lateral geniculate nucleus (LGN) of the anesthetized cat can be very efficiently stimulated with flashing bright or dark spots on a medium gray background. After the visual latency (in LGN around 40 ms) the cells will first respond with a strong phasic peak with a duration of 50-100 ms and with an amplitude which is largely independent of contrast [57]. Subsequently, they will settle to tonic firing of up to 200 Hz impulse rate. The actual firing rate of the tonic component is strongly contrast dependent and in most cells slow adaptational processes will reduce the initial tonic rate by a significant amount during the first second of stimulation. In a peri-stimulus-time histogram (PSTH) the tonic response therefore shows up as a gradually downward sloping straight line. This has led to the misleading interpretation that (apart from a gradual adaptation) the tonic LGN component

represents a *constant* firing rate. This interpretation was supported by the observation that LGN inter-spike interval histograms (INTH) resemble a Poisson distribution. Fig. 1 shows that this is actually not the case for many cells. Here, we plot a time-resolved diagram of the spike interval distribution of an LGN X-On cell [19]. Gray-scaled pixel lines represent single INTHs obtained from inside a temporal window of 100 ms length. The window was shifted in 10 ms steps along the time-axis such that adjacent pixel lines represent two snapshot-INTHs with 90% overlap of their input data. This way a sliding window INTH is constructed reminiscent of a sonogram known from auditory signal analysis. There, frequency distributions are plotted in a time-resolved way, whereas here we plot interval distributions. Accordingly, we call this type of diagram an "intervalogram". The multiple bands in the shown intervalogram indicate that the apparently constant firing rate of this LGN cell, observed during its tonic response, consists indeed of an accumulation of preferred intervals. These preferred intervals are included in every single response but, due to some jitter in latency [74], they are not visible in the summed tonic response of the PSTH. A closer look reveals that higher order intervals, represented by those bands towards the right side of the diagram, are integer multiples of the leftmost interval band, which we call the fundamental interval. This behavior is mainly found in On-cells and can be explained by inhibitory and/or disfacilatory processes at the level of the LGN because retinal responses do not contain multiple bands [19]. Fig.2A shows that increasing surround inhibition, when using increasingly larger stimuli, will lead to more pronounced higher order bands supporting the notion that inhibition is involved in this process.
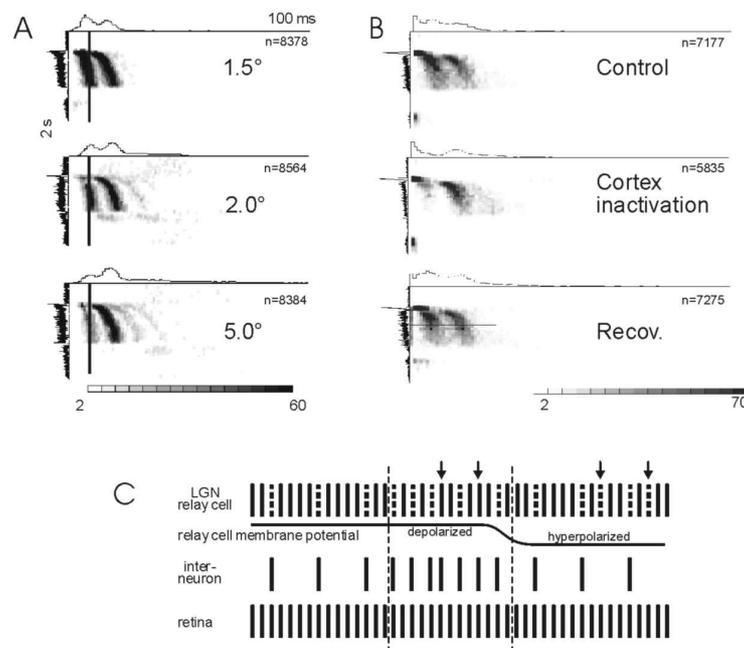
Fig.2 (A) Intervalograms of X-On cell responses to flashing spot stimuli of different size but same contrast (90%). (B) Intervalograms of the response of a different X-On cell to a spot of 1.5 deg (90% contrast) before, during and after inactivation of the corticofugal feedback. Blockade of the corticogeniculate feedback was achieved by continuous micro-iontophoretic application of GABA to topographically related parts of layer 6 of area 17/18. The total number of intervals is given by n. In both situations the higher order bands become more prominent. Stimulus protocols as in figure 1B. (C) Schematic diagram showing how intra-geniculate inhibition and a changing relay cell membrane potential affects the composition of the LGN output spike train by fundamental and higher order intervals. Activity of an inhibitory interneuron is low in the left part preventing only a few retinal input spikes from being transmitted by the LGN relay cell (dashed lines). In the middle part, inhibitory activity is higher, punching more holes into the output spike train. Due to a depolarized level of the LGN membrane potential, inhibition may sometimes be ineffective (spikes labeled by arrows). The LGN relay cell is hyperpolarized in the right part. Now, transmission of retinal inputs is not only reduced by inhibitory activity but also because some retinal EPSPs may remain subthreshold (dashed lines labeled by arrows). Recompiled from [19] and [75].

Fig. 2B, on the other hand, demonstrates that reversible removal of the corticofugal feedback leads to a similar effect. This can be interpreted as a removal of excitatory cortical influences at the LGN cell[2]. Thus, an increase of inhibition or a decrease of excitation both lead to a similar effect concerning the interval distribution of LGN responses. It is known that LGN cells respond under optimal circumstances in an almost one-to-one manner to the afferent input, thereby faithfully producing one output spike for each single retinal input spike [32,42]. The influence of intra-LGN inhibitory or corticofugal excitatory signals on the momentarily observed interval distribution indicates that gradual shifts of the LGN membrane potential with respect to the firing threshold can strongly affect the signal transmission rate. Fig.2C shows this in a schematic way. During active cortex and without inhibition an 1:1 transmission occurs. As soon as inhibition sets in, not every retinal EPSP will reach spiking threshold: "holes are punched" in the LGN spike train. A hyperpolarization of the LGN membrane potential due to a removal of the corticofugal excitatory action will lead to the same effect. What are the possible functional implications of this multi-interval pattern? We hypothesize that the temporal structure of the spike train can be helpful to resolve ambiguous stimulus situations which could evoke almost the same mean rate of spike discharge at the LGN level.
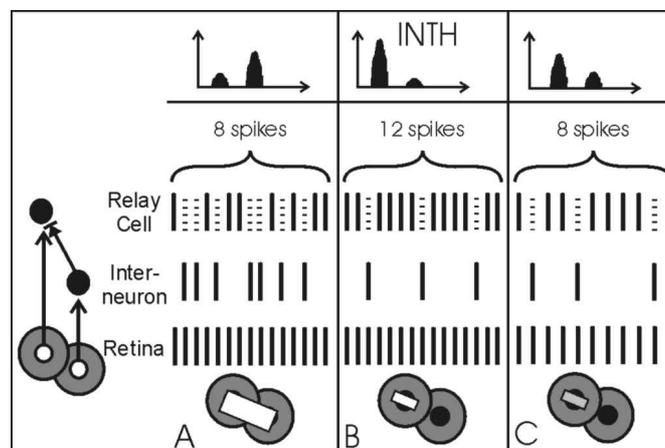
Fig.3 *Schematic diagram depicting the retinal and geniculate spike patterns that result from three different stimulus situations (A,B,C). The small bright stimulus in B optimally stimulates the center of the retinal ganglion cell that excites the LGN cell without causing much inhibitory activity. The result of this stimulus is an LGN output of 12 spikes within the shown time window. Two processes can now reduce the mean LGN spike rate such that in our example the same number of spikes occurs in the time window (8 spikes): A larger stimulus which also excites neighboring ganglion cells (A) elicits about the same firing rate in the ganglion cell feeding excitation to the LGN cell, but at the same time the neighboring ganglion cell drives an LGN interneuron. The inhibitory input to the LGN relay cell prevents the transmission of some retinal input spikes and leads to a higher number of double-sized spike intervals at the LGN output. Reducing the brightness but not changing the size of the stimulus (C) results in a reduced firing rate of the ganglion cell without affecting the inhibitory activity. The ratio of fundamental and double-sized intervals is similar to case B but the absolute length of the intervals is now changed. Modified from [20].*

In Fig.3 three stimulus situations are shown which elicit different temporal patterns of tonic LGN activity: (A) a large, bright stimulus, (B) a small, bright stimulus and (C) a small, less bright stimulus. The schematic drawing is based on LGN data obtained with different contrast and size of visual stimuli [19,20]. The basic firing frequency is contrast dependent. Accordingly, the retinal cells in cases A and B will fire more strongly than in case C. In addition, we assume that surround inhibition at the level of the LGN is much larger than in the retina [27,59], where we – for simplicity - set it to zero. Therefore, identical retinal spike trains occur in A and B. The large stimulus in A drives not only the retinal input that excites the LGN relay cell but stimulates also neighboring ganglion cells which provide input to the antagonistic surround of the relay cell via intra-geniculate inhibitory interneurons. The result is a stronger surround inhibition than in cases B and C. The example is constructed in such

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[2] All corticofugal synapses are excitatory. They terminate at LGN relay cells and inhibitory interneurons (47,71) but in most cases inactivation of the corticofugal feedback leads to a reduction of the net excitatory input at the target LGN cells also in other studies (3,16,31,44).

a way that due to these processes the same number of spikes is observed in the LGN for the large bright (A) and the small, less bright (C) stimulus. The mean firing rate in this window is therefore identical and these two stimulus situations cannot be distinguished by it. The interval pattern, however, differs. The stronger surround inhibition in A leads to more "holes" and, accordingly, the higher order intervals dominate here. In the situation depicted in C, a lower retinal impulse rate is met by little intra-LGN inhibition. Only very few "holes" are punched and the fundamental interval dominates. Thus, the interval distribution would allow to disambiguate the two situations (A) and (C) even though the mean firing rates are identical. Certainly, the same could be achieved by the cooperative action of more than one cell in the visual neural network, but this example was supposed to demonstrate that, in principle, already a single LGN cell suffices.

Simultaneous recordings of LGN spikes and retinal pre-potentials [19] have shown that multi-modal spike interval distributions appear first at the level of the LGN. The tonic retinal activity consists of only one spike interval, resembling the fundamental interval, corresponding to the leftmost band in LGN spike interval distributions. The occurrence of a single interval in tonic retinal firing points to the involvement of an oscillatory process. Indeed, oscillatory activity in a frequency range (60-200 Hz) corresponding to the observed spike intervals has been described for ganglion cells stimulated with a large uniform field [36] or a moving grating [51]. It can be assumed that the preferred retinal and geniculate spike intervals are closely related to the oscillatory pattern of activity. Direct correlation was less frequent when LGN cells were stimulated with small spots of light [74], however, large coherent stimuli seem to promote both the occurrence of oscillations and the synchronization of activity in the population of stimulated retinal and geniculate cells [51]. Studies of geniculate visual activity during reversible inactivation [18] or removal [60] of the primary visual cortex further indicate that the cortico-geniculate feedback promotes synchronization of visual activity in the LGN.
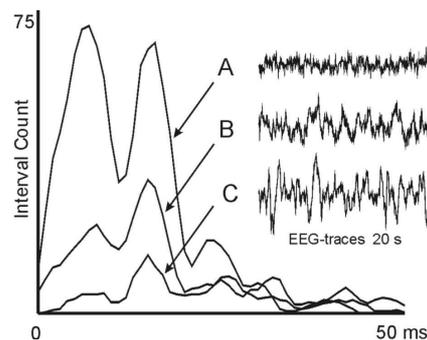
Fig.4 *Regular interval histograms (INTHs) obtained from cross-sections through intervalograms for an X-On cell recorded during spontaneous EEG transitions. (A) Non-synchronized EEG, (B) intermediate state and (C) synchronized EEG. With increasing EEG synchronization, the responsiveness of the cell decays, fundamental intervals (left peak) are strongly reduced and higher order intervals start to dominate. Modified from [75].*

The influence of the EEG-state on thalamic cell responses
In the last section we had demonstrated that increasing inhibition and/or decreasing excitation can lead to very similar effects concerning the interval band-structure of LGN responses. It is known that the level of excitation at an LGN cell is also reduced during sleep [16,57,63]. Excitatory brain stem and corticofugal influences are diminished during an EEG state which is dominated by δ-waves [16,61]. This state is in a non-anesthetized situation usually associated with deep sleep. In an anesthetized preparation still strong spontaneous transitions between a δ-wave dominated (so called *synchronized EEG*) and an EEG of reduced δ-wave activity (so called *non-synchronized EEG*) can be observed. Therefore, we expected that during synchronized EEG net excitation at the LGN cells should be reduced and the interval structure of the LGN responses should be affected in a similar way as during removal of corticofugal feedback: The higher order intervals should become more pronounced. This is exemplified in Fig.4. The EEG traces reflect a continuous transition from a non-synchronized to a strongly synchronized EEG. Regular INTHs that have been obtained by averaging several pixel lines from a cross-section of the corresponding intervalograms of the activity of an LGN X-On cell are

shown for each EEG state. It is clearly evident that the fundamental interval decreases during increasingly synchronized EEG at the cost of relatively larger higher order bands.

We can summarize all these observations in the following way: Higher order interval bands dominate over lower order intervals as soon as the membrane potential of LGN cells is increasingly hyperpolarized regardless of the responsible underlying process, be it increased intra-LGN inhibition, reduced corticofugal excitation, a reduced facilitation by brain stem inputs (synchronized EEG), or any other process which lowers the LGN membrane potential (e.g., also by experimental pharmacological interference, see e.g., Fig. 6 in ref. 75). Changes in the level of brain stem controlled arousal have been shown to affect not only thalamic activity but also the temporal pattern of cortical activity. Transient desynchronization of the EEG caused by electrical stimulation of the brain stem arousal centers increases the power of γ-activity. Highly synchronized oscillations in the γ-range (30-90 Hz) specifically occur during visual stimulation with coherent stimuli [12,24,51]. Then, γ-oscillations are also visible in local field potentials. However, the degree to which neurons can synchronize within this frequency range critically depends on the state of arousal. For example, electrical stimulation of the brain stem increases synchronicity of visually induced γ-oscillations [48,62].
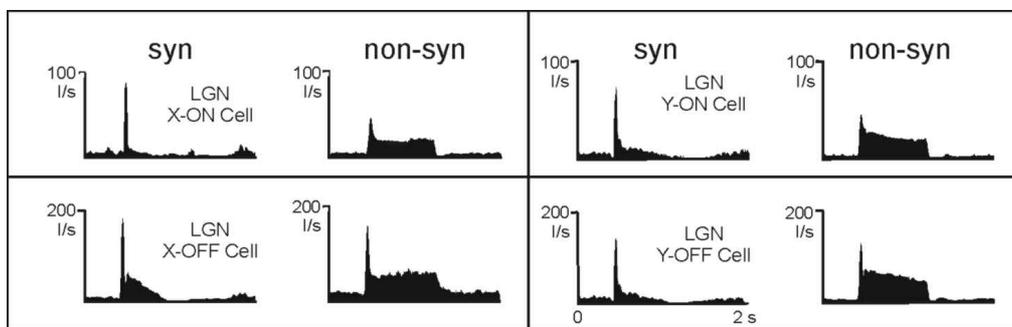
Fig. 5 PSTHs from four different LGN cells recorded during different EEG states. During synchronized EEG strong phasic bursts are observed at onset of the stimulus but tonic visual activity is strongly reduced. During non-synchronized EEG the initial bursts are often reduced in size and are followed by a pronounced tonic response. Modified from [76].

Fig.4 is not very well suited to demonstrate the tremendous impact that an EEG change can exert on the thalamic cell behavior. Therefore, in Fig.5 we show a few PSTHs of LGN cells recorded during synchronized and non-synchronized EEG. The same cell changes its firing characteristic completely when such an EEG transition happens. During a non-synchronized EEG, the initial phasic response is followed by a pronounced tonic response component which is almost missing during synchronized EEG. Such transitions can occur on a rather short time scale. Figs. 6A,B give an example where two EEG-transitions (from non-syn. to syn. and back) occur within 200 seconds of recording time. The EEG traces on top show that δ-waves are more pronounced between stimulus sweep 25 and 75 when tonic LGN light responses disappeared (Fig.6B). In Figs.6C,D we plot the mean LGN firing rate (thin line) against the relative spectral power of the δ-range in the EEG (1-4 Hz) for a different cell. Both traces were averaged and filtered prior to plotting. Two different filter settings were used. In 6C a strong low pass filter was applied which cancels all high frequency oscillations and emphasizes the gradual increase in EEG δ-activity which is accompanied by a correlated decrease in mean LGN firing rate. A correlation of this type was found whenever the EEG showed slow changes in δ-activity [38]. In 6D a band-pass filter was used to remove the DC but leave higher frequencies intact. Here, we see distinct oscillations with a period of about 5-10 s. These oscillations are very generically found in almost all LGN cells and seem to reflect a very slow intrinsic rhythm [64]. Many times during these oscillations mean firing rate and EEG δ-power are also inversely correlated but this relation is less significant than during the slow EEG changes described before (e.g., Figs.6A,C, and ref. 38).

The LGN-PGN antagonism
The primary visual thalamus consists of the LGN and an accessory structure, the perigeniculate nucleus (PGN) which is a thin cell layer covering the LGN at its dorsolateral border.
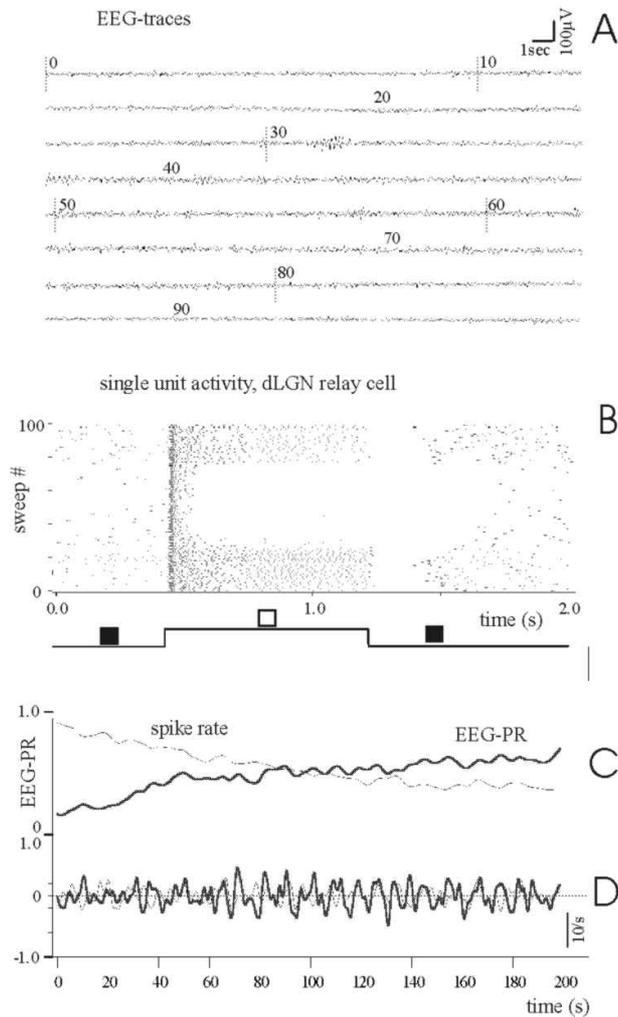
Fig. 6 *Time course of the EEG-correlated change in LGN firing behavior of an X-On cell. (A) EEG-trace for 200 s of recording duration. (B) Dot raster diagram of the LGN cell recorded simultaneously with the EEG. Note the absence of the tonic visual response during the period of increased slow waves in the EEG. (C,D) Results of another LGN cell. (C) Slow changes of the EEG power ratio (EEG-PR, thick line) and mean LGN firing rate (thin line). The EEG-PR measures the δ-power in the range 1-4 Hz relative to the power in the β-range (20-30 Hz). (D) Fast oscillatory changes of the EEG-PR accompanied by a similar oscillation of the mean LGN firing rate. Note, the oscillations have a period of about 5-10 s. For a change in the spectral composition of the EEG this is rather fast. Recompiled from [38].*

Anatomically the PGN is part of the thalamic reticular formation and functionally it is involved in the generation of EEG sleep-spindles [4,34,46,56,70] and – together with the corticofugal loop – also of δ-waves. LGN and PGN form a recurrent excitatory-inhibitory loop: LGN cells excite PGN cells and these in turn inhibit the LGN cells. Fig. 7 shows that this leads to a pronounced LGN-PGN antagonism. The LGN cell firing rate is reduced significantly as soon as the PGN cell fires strongly. The figure furthermore demonstrates that this antagonism is also correlated with the state of the EEG (Fig.7C). We have previously shown that increasing δ-activity of the EEG is associated with reduced LGN firing [38]. With simultaneous recordings of the activity of topographically matched PGN and LGN cells we could also demonstrate that PGN firing is usually increased during high EEG δ-activity
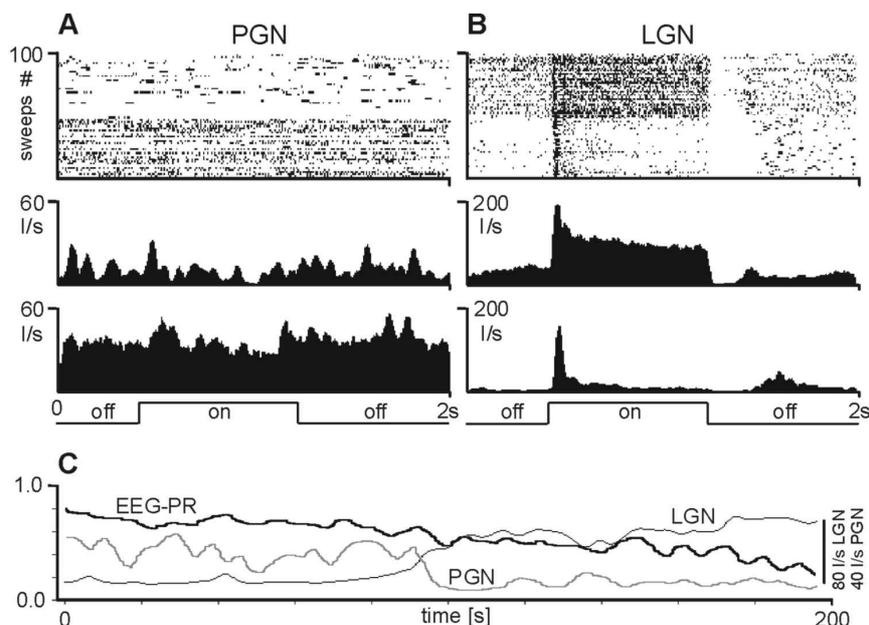
Fig. 7 LGN-PGN antagonism and its relation to the EEG. (A) PGN dot raster diagram, which shows a spontaneous transition from high tonic to burst firing around stimulus sweep 45. Accordingly, two PSTHs below show the summed activity for sweeps 1-45 and 46-100, respectively. (B) Same for an LGN cell recorded simultaneously. (C) Mean firing rate of PGN and LGN cell and the EEG-PR. The antagonistic behavior of LGN and PGN cells is clearly visible. The stimulus was a small (1 deg) spot of light switched on and off within the overlapping receptive fields of the LGN and PGN cell. The PGN cell shows only a tiny visual response because the stimulus was too small to efficiently stimulate the receptive field of the PGN cell.

[21]. So far, this was found for the majority (17/18) of PGN-LGN double recordings that included a spontaneous change of the EEG pattern. Thus, the correlation between thalamic (LGN and PGN) cell behavior and EEG state is very pronounced and opposite changes in activity occur almost simultaneously. The inverse correlation between LGN and PGN activity indicates that the PGN is strongly involved in the control of the retino-cortical transmission of visual information [21].

The following (simplified) picture emerges [16,21]: With high PGN activity during certain sleep states the LGN cells will fire only phasically and a faithful transmission of stimulus properties (e.g., spatial contrast) to the cortex is largely prevented. Stimulus dependent cortical input consists mainly of brief but strong bursts of LGN activity, which cannot be used to generate a reliable visual perception but are very well suited to act as a wake-up signal [16,25,57]. During wakefulness, PGN activity is lower and the LGN cells will fire tonically. Stimulus properties can be encoded in the tonic firing rate and a perceptional analysis of the visual scene becomes possible starting at the level of the primary visual cortex. It should, however, be noted that the PGN is certainly not the driving force of this process. Brain stem influences from the ascending reticular arousal system which terminate in PGN and LGN are more likely candidates for this task [1,17,21,45,50] and the complete dynamic interplay of all these structures underlies the observed activity changes.

Dynamics of cortical receptive fields and the correlation of EEG-state with cortical responses
The strong temporal changes in the firing characteristic of LGN cells make it seem likely that their cortical targets should also display an EEG-correlated behavior. Early indications arose from the studies of Ikeda and Wright 1974 [29] who found that cortical cells respond more phasically during synchronized EEG. This is probably a direct reflection of the LGN cell properties. Around 1982 a series of studies was published by a Russian group which showed that the receptive fields of cortical cells change as a reaction to an air puff (arousal stimulus) was applied to the closed eye of a paralyzed but unanesthetized animal. The stimulus applied during sleep induced a state change to a non-synchronized EEG and the cortical receptive fields, recorded while stimulating the other eye, also changed their shape [58,66,67,68]. The authors attribute those receptive field changes to arousal effects mainly arising from the brain stem and also to an intracortical restructuring mechanism. The

time-scale for the effects which they found was much longer (approx. 20 min) than the rather fast changes observed by us. Furthermore, a different procedure of receptive field mapping was used by these authors.

In 1982 very little was known about the EEG-dependency of the thalamic behavior [8,26,43]. Only after a more complete picture had emerged a clear prediction concerning the correlation between EEG and cortical cell behavior could be made by our models (see below). Intriguingly, the model predicted that the receptive fields of cortical cells should show a more pronounced spatial change as compared to a relatively weak temporal change in their firing behavior. In a set of experiments we tested this hypothesis.
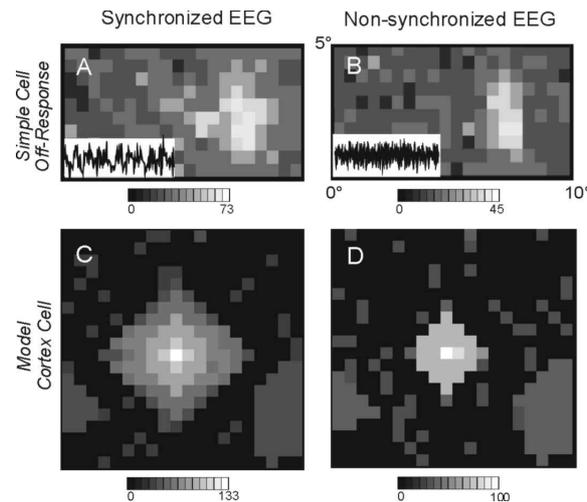
Fig. 8 State-dependent changes of receptive fields of real cortical cells (A,B) and model cells (C,D). Insets in A and B show EEG traces. The receptive Off-subfield of a simple cell measured during the initial part of the visual response (first 20 ms) was wider during the synchronized EEG state than during the non-synchronized state. In addition, the response was stronger as can be seen by the higher spike counts. Gray scales indicate the number of spikes counted at each grid position for a total of 30 stimulus repetitions at every position. Stimuli were short bright or dark bars (1x0.5 deg) flashed with optimal orientation in a grid of 20x10 positions corresponding to 10x5 deg of visual angle. (C,D) Results for a model cell with simulation of a state change by changing PGN inhibition onto the LGN cells. The grid has arbitrary units, orientation selectivity has not been implemented in the model and, therefore, the responses are isotropic. As in the experiment, also the model cell shows a larger responsive area during the synchronized state (C) and a somewhat stronger early visual response. Recompiled from [76].

Fig. 8 (top) demonstrates that cortical receptive fields change their size in an EEG-related way. These receptive field plots were measured by the „reverse correlation technique". This method determines how often any given stimulus of the applied set could have been responsible for eliciting a spike response from the cell under study [11,13]. As stimuli we used small bright and dark dots or bars flashed with a duration of 300 ms on a medium gray background within a grid of 20x10 locations which covered the receptive field completely. Furthermore, several temporal windows were predefined starting at zero delay and going back in time with equidistant steps of 10 ms. For any given spike we looked back in time and determined which stimulus location was active in the different windows. The corresponding count is raised by one and this way a 2-dimensional stimulus-response occurrence histogram is created in every time-delayed window. If, after some latency, a certain stimulus was able to elicit a spike with above-chance probability, then the corresponding bin in the latency-matched occurrence histogram stands out above noise level. This way multiple temporally staggered receptive field maps could be created which represent snapshots of the different sites and states of excitability in the region covered by the stimulus set.

The basic observation is that cortical receptive fields are larger during a synchronized EEG than during a non-synchronized EEG. This is particularly pronounced during the initial part of the response (first 20 ms, see Fig.8) which was taken to construct the receptive field map. In a set of 63 cells we

observed an average 27% increase in receptive field size that was correlated with a 2.5 fold increase in the δ-power range of the EEG [76]. In control experiments we checked if the receptive fields of LGN cells also changed during a change in EEG pattern. We found, however, that they remain almost the same regardless of the EEG state. Thus, receptive field restructuring seem to be a cortical phenomenon. At the same time we observed that the cortical receptive fields also shrink with increasing time during stimulation (Fig. 9). About fifty milliseconds after stimulation with a flashing bar the first cortical spikes can be observed. At that time responses can be elicited from a rather wide area extending into regions far laterally from the receptive field center. During the next 50 ms the receptive field shrinks and spikes cannot anymore be elicited far away from the center. The shrinkage of the receptive field can be expected to enhance the spatial resolution of the receptive field. This could in principle be accompanied with an increase of the specificity of the receptive field for certain aspects of the stimulus. However, this seems to be not the case for orientation selectivity since orientation tuning does not sharpen during the course of the visual response [7]. Other studies could not demonstrate a shrinkage of receptive field subunits because of the very short stimulus duration (40 ms) used for the mapping procedure but have shown other aspects of receptive field restructuring. DeAngelis and co-workers [11] have demonstrated that the sensitivity of receptive field On- and Off-subregions can change in polarity during the course of the response. Also in this case the temporal pattern of the response could contribute to the observed effect. For example, the spatio-temporal change in receptive subfields is considerably changed when intra-cortical inhibition is partially blocked by the GABA$_A$-receptor antagonist bicuculline [54].
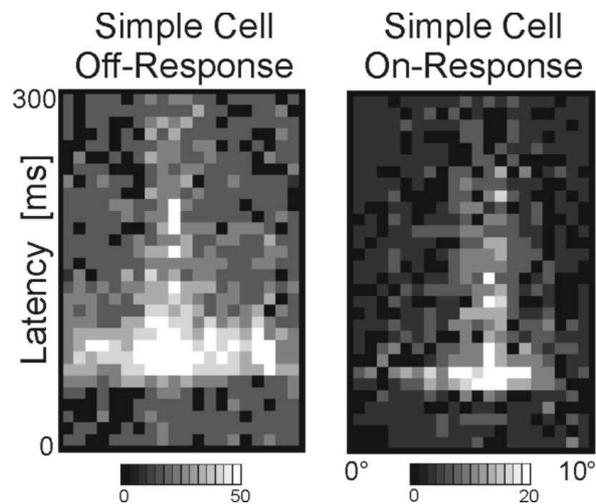
Fig. 9 Temporal changes of the size of two cortical receptive fields. The receptive fields are wide during the initial part of the visual response but considerably shrink with time when visual activity changes to a tonic response. In these diagrams only the x-dimension of the spatial receptive field plot is shown on the abscissa. Therefore, spike activity was summed up along the Y-axis of the receptive field (i.e. along the ordinate of the diagrams in figure 8). Time is plotted along the ordinate. Stimulus duration for each grid location was 300 ms. Recompiled from [76].

Modeling the state-dependent spatial receptive field restructuring of cortical cells
One could expect that the strong changes of the thalamic firing pattern should also influence the cortical cell behavior. A mechanistic explanation for the observed spatial receptive field restructuring, however, requires a more quantitative approach. Therefore, we designed a biophysically realistic computer model of parts of the primary visual pathway including retina, LGN-PGN and primary visual cortex. Cells were modeled as improved integrate and fire units [73] and the network contained about 2000 cells in total. The mechanism which we thought might principally underlie most of the observed thalamic and cortical behavior, roots in the temporal characteristic of LGN activity. It can switch between the tonic firing mode and the phasic burst mode [25,57]. Regular tonic firing occurs if LGN cells are in a depolarized state. If they are hyperpolarized, low threshold Ca^{2+} channels become de-inactivated and even small depolarizing inputs may be able to elicit a Ca^{2+} spike which has a duration of several tens of milliseconds [30,37,39]. The Ca^{2+} spike itself leads to a further depolarization such that the regular firing threshold of Na$^+$ spikes is reached. Since the depolarization

is rather strong and long lasting, several Na$^+$ spikes are elicited with a maximum firing frequency of 200 to 700 Hz: a so called burst has occurred. The burst is followed by a strong repolarization that brings the cell back to the hyperpolarized level.
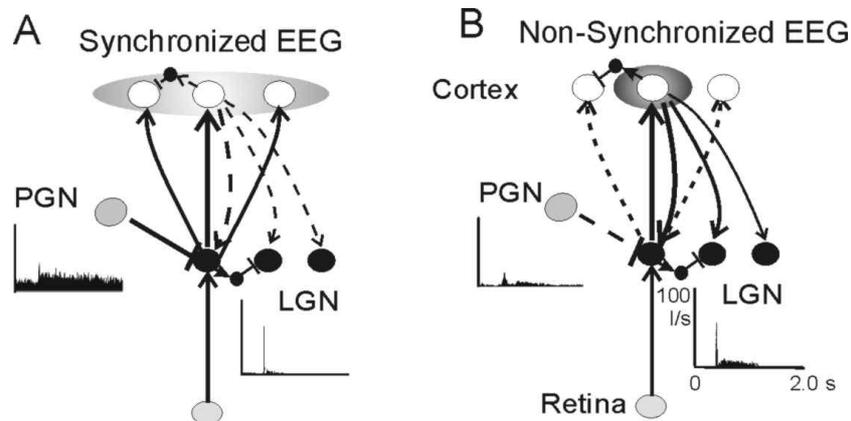
Fig. 10 *Schematic diagram of our model. Obviously the same anatomical connectivity exists during both EEG states, but the effective connectivity changes. Thick lines indicate efficient connections which – based on the burst activity in the synchronized state – span a larger cortical area than in the desynchronized state when tonic activity prevails. For further explanations see text. Recompiled from [76].*

We implemented this mechanism implicitly in our model LGN cells. In addition, we assumed that the PGN acts as the central controller for the state in which LGN cells momentarily are. As soon as PGN inhibition is strong - which is the case during synchronized EEG - LGN cells are hyperpolarized and fire only in phasic burst mode. If PGN inhibition ceases with desynchronization of the EEG, the LGN cells become depolarized and switch to tonic transmission mode [17,21]. In the model the PGN got this central role, because in the real brain it controls geniculate activity by integrating corticofugal and brain stem influences and by contributing to the generation of spindles and δ-waves [4,56,70].

From anatomy it is known that many (30-100) LGN cells converge onto every single cortical target cell [27]. At the same time the lateral spread of the afferent convergence is quite large [28]. Thus the activity of every LGN cell reaches many cortical units, resulting also in a rather large anatomical divergence. The model diagram (Fig. 10) depicts this situation in a schematic way. A large anatomical divergence, however, is not necessarily related to also a large point spread of the activity. The actual synaptic strength determines to a large degree how far the activity of a small stimulus will spread out in the cortical network. Of additional - if not more – importance, however is the immediate activation history at every synapse. If many spikes arrive in a rather short temporal interval, temporal summation takes place and also weaker synapses can lead to spiking. In our case strong temporal summation occurs as soon as the LGN cells are in phasic burst mode. In this mode 2-10 spikes arrive at the cortical targets within only 50 ms. This leads to spiking also at targets located lateral to the central thalamocortical projection column. A wide point spread is observed and, accordingly, also wide cortical receptive fields. If the LGN cells are in tonic transmission mode less temporal summation occurs, the point spread is reduced and the receptive fields are smaller. In addition, the longer lasting activity can drive lateral inhibitory cells which in turn suppress their neighbors' firing. This adds to the effect of keeping the receptive fields small (based e.g. on the iceberg effect).

It is, however, mainly the temporal firing characteristic of LGN cells that determines in our model the spatial dimensions of cortical receptive fields. The corticofugal feedback plays a minor role in this scenario. During synchronized EEG, LGN bursts are too short to drive the corticofugal loop efficiently and phasic corticofugal activity will fall into the period of hyperpolarization that follows the burst in LGN cells. During non-synchronized EEG, the feedback will help a little to keep the LGN cells in the central projection columns depolarized in order to maintain tonic transmission mode. If the feedback is removed, the LGN cells fall back into phasic burst mode.
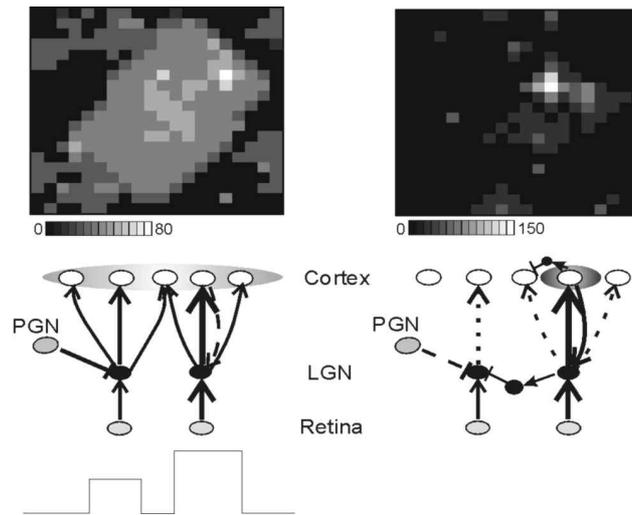
Fig. 11 *Implementing a bottom-up (pre-)attentional control mechanism. The left side of the figure depicts bursting behavior at two stimulus locations as it occurs at the onset of a sudden change in contrast. The right side shows the cortical responses during a later state of tonic visual activity after the attention mechanism "has selected" one stimulus. For further explanations see text.*

Receptive field restructuring as a mechanism for information flow control: A hypothesis

In this review we have tried to summarize the different results which describe how thalamic and cortical cells change their spatio-temporal firing behavior in relation to the state of the EEG [38,76]. Table 1 summarizes these results. Still it is not clear why a collection of such mechanisms should be implemented in the visual system. If we extrapolate the cellular behavior observed in the experimental preparation to a non-anesthetized situation, we can speculate about possible functional implications. A synchronized EEG is normally observed during deep sleep, dominated by δ-waves, or during drowsiness, dominated by α-waves. A non-synchronized EEG, which is dominated by β- and γ-waves, on the other hand, occurs during attentive wakefulness. If the observations can be transferred, then we would expect that during drowsiness wide and unspecific cortical receptive fields exist, whereas during attentive wakefulness they should be small and specific. In the first situation almost all stimuli – if strong enough – could elicit LGN bursts and due to the wide point spread many cortical cells will be driven. In addition, these bursts lead to fast spiking due to efficient temporal summation. Taken together this could act as a fast but broad and unspecific wake-up signal alerting the drowsy animal as soon as anything of importance (a sudden change in the visual environment) happened. The visual activity signals only that something has happened and were it approximately has occurred but the spatial resolution is low. Only after this wake-up call has been received by the system, it might switch to a more specific mode and initiates a more detailed analysis of the incoming signals. In order to do this, tonic LGN activity, which faithfully encodes stimulus properties (like spatial and temporal contrast distribution), and small receptive fields of cortical cells are required.

Table 1) Comparison of different EEG states.

Global State	**drowsy**	**alert**
Information processing	wake-up signal	transmission mode
EEG state	synchronized	non-synchronized
EEG waves	α/δ	β
Neural temporal behavior (LGN)	Phasic firing (burst)	tonic firing
Neural temporal behavior (PGN)	tonic firing	phasic firing (burst)
Neural spatial behavior (Ctx)	wide receptive field	narrow receptive field
Effective connectivity (LGN→Ctx)	high	low
Synaptic weights	unchanged	unchanged

This functional hypothesis can be taken even further because it is only consequent to discuss possible effects of shifting attention within the same context. After all, a switch between drowsiness and

wakefulness should in principle be related to a shift between being inattentive or attentive, only the level of changing overall neural activity is certainly too small to detect such an attention shift in the EEG. From an engineering point of view it would be very useful if only those signals are exactly analyzed on which the focus of attention currently rests. For all other signals a coarser level of analysis would probably suffice. This argument becomes even stronger if one considers the vast amount of information which arrives at the opened eyes at any one point in time. In technical information processing systems such a large information inflow must be restricted otherwise an input overflow catastrophe occurs. The incessantly changing focus of attention is probably one of the central flow-control mechanisms in the brain [35,40,65]. Good evidence for such a process has recently been presented by the results of a functional MRI study [33] which demonstrates that the cortical representations corresponding to multiple salient objects shown in a visual scene compete in an inhibitory fashion.

In our approach state-dependent brain stem inputs firing (not explicitly modeled) have influenced PGN such that different overall levels of inhibition influenced the LGN cells. In the case of shifting attention we should assume that the animal is alert and inhibitory PGN input to the LGN should in general be small. Which mechanism however could then be responsible for adjusting the temporal firing pattern in the LGN and thus, the spatial receptive field resolution in the cortex? The goal must be to achieve a high resolution at the location were the focus of attention rests and a lower resolution everywhere else. In addition, this must be a rather localized mechanism, because the spatial extent of the focus of attention is rather small. We believe that this could be achieved by means of the corticofugal feedback. We have shown before that this feedback is involved in the control of signal transmission from LGN to cortex: A missing feedback leads to more "holes" in the LGN spike trains and the higher order intervals become dominant. Thus, LGN cells are more hyperpolarized, when the feedback is missing [18,20,75] and more depolarized when it is active. Furthermore, feedback projections are relatively localized and do not just exert a broad unspecific influence on their target region. If we assume that the corticofugal feedback has its major influence at that specific location where the focus of attention currently rests, then the receptive fields of the cortical cells would be small there and wider everywhere else. Taken together, the corticofugal feedback could thus control the membrane potential level in the LGN to a degree necessary for distinguishing between attentive and inattentive local retinotopic regions.

Commonly, models of visual attention require some kind of control mechanism by which the focus of attention is directed to the most salient stimuli. This is usually achieved by a so called "saliency map" which is a topographically arranged neuronal network [35]. Neurons in the saliency map respond most strongly to very salient stimulus features like the color red or a fast motion. Feedback from the saliency map to the ascending pathways will lead to a stronger drive where ever a salient feature resides. In addition, such models usually implement a winner-take-all (WTA) mechanism, by which only the most salient stimulus is selected while the others are suppressed [52,53,65]. The activity of the saliency map and its WTA is transient such that after some time the activation decays and the second most interesting feature can be analyzed.

All this requires some higher level control. After all, who decides what should be salient and what else not? It seems reasonable to assume that such a higher level control exists at least for higher cognitive processes. The question, however, arises if there would be a more generic way to achieve some kind of pre-attentive control in early vision (i.e., along the first stages of the primary visual pathway).

We think that the effects described here could lead to the desired behavior by means of a stimulus driven *bottom-up* mechanism in which the corticofugal feedback will then lead to symmetry breaking. This can be described in an algorithmical and schematic way as follows (Fig.11): Let us assume that the system is in an inattentive state. Corticofugal feedback is weak and the LGN cells are everywhere in phasic burst mode. Thus, if a group of stimuli occurs, all of them will lead to burst-firing in the LGN. After that the system is aroused. Subsequently, the brightest stimulus (which is most salient), will induce the strongest tonic component as compared to the other stimuli. This could be regarded as a stimulus driven symmetry breaking effect: The corticofugal feedback will be strongest at that particular location at which the brightest stimulus has occurred. As a consequence lateral inhibition

exerts suppressive control on all other locations implementing a transient winner-take-all mechanism. Only after the transient inhibition has faded, other "less interesting" stimuli can take over and the same process can start again.
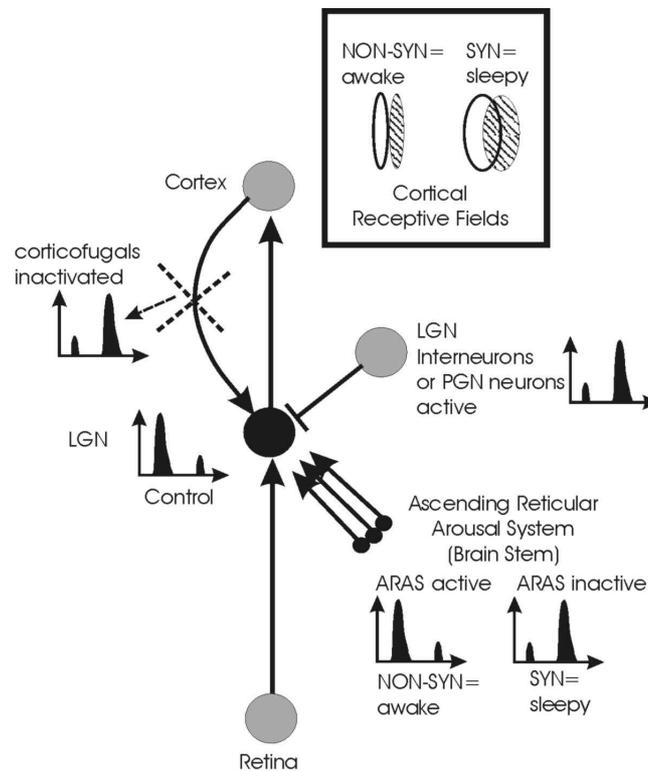
Fig. 12 Summary diagram of our main experimental findings. The little diagrams depict schematic inter-spike interval histograms (INTHs) of the LGN relay cell in the center. LGN interval distributions can change from the control case (1st peak is highest) to a situation where the second (or higher order) peak is highest by: 1) by increasing inhibitory inputs from LGN interneurons or from the PGN, 2) by removing corticofugal excitation, and 3) by a reduced ARAS activity. These changes in the temporal firing patterns of the LGN cells will induce a size-change in the visual cortical receptive fields possibly affecting the visual spatial resolution.

Conclusions:
Fig. 12 summarized in a schematic way our main findings. LGN interval distributions can change from the control case (1st peak is highest) to a situation where the second (or higher order) peak is highest in at least three ways: 1) by increasing inhibitory inputs from LGN interneurons or from the PGN, 2) by removing corticofugal excitation, and 3) by a reduced ARAS activity. These changes in the temporal firing patterns of the LGN cells will induce a size-change in the visual cortical receptive fields possibly affecting the visual spatial resolution. If our hypothesis hold, then such a change in resolution could be utilized to the changing requirements for visual information processing during changing visual attention. Several components of this last aspect have been already described by others [35,52,53,65] and, in some way, we have revived the famous search-light hypothesis of Crick [9] and tried to put it in the context of those facts which were still unknown in 1984. The central difference which distinguishes our ideas from other approaches is that the bottom-up aspect gets the strongest emphasis here. We believe that the primary visual pathway is still a low-level structure and thus only relatively unsophisticated mechanisms are probably implemented there in order to arrive at a good preprocessing of the visual scene. At the same time, it is vital that such a preprocessing is very fast. The mechanisms described here would serve both purposes. Obviously, in reality all this is a very dynamic process taking place in a highly interleaved way. Our current models are vastly oversimplified reflections of the complex reality, yet they demonstrate that the hypothesized

mechanisms in principle work. It will be a matter of ongoing research over the next years to support or falsify our ideas and we are looking forward to this.

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