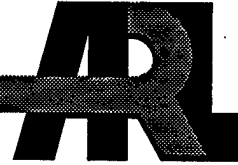


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Cortical Oscillations in the Visual Cortex

by Adrienne Raglin

ARL-TR-1558

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Cortical Oscillations in the Visual Cortex

Adrienne Raglin

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Abstract

Computer vision deals with algorithms that allow machines to detect, segment, feature extract, and recognize objects in an image. There are numerous applications in medicine, manufacturing, and security for this technology. By studying the visual processes of biological systems, enhancements can be achieved in the development of computer vision algorithms. One biological function of interest involves the oscillatory pulses generated in the primary visual cortex engaged in stimulus-specific oscillatory responses. As a result of these experiments, it can be concluded that these tightly correlated, stimulus-induced oscillations may play a role in the recognition of images. Therefore, these cortical oscillations have been modeled to investigate their ability to segment objects in a visual field. This report briefly discusses the visual system and the internally stimulus-dependent oscillations that may lead to identification of images. Emphasis will be on the models that attempt to reproduce this biological phenomena, their computational and behavioral aspects, as well as simulation performance. Detail will be given to their computational and behavioral aspects since it is in these areas that possible improvements can be achieved through more detailed modeling.

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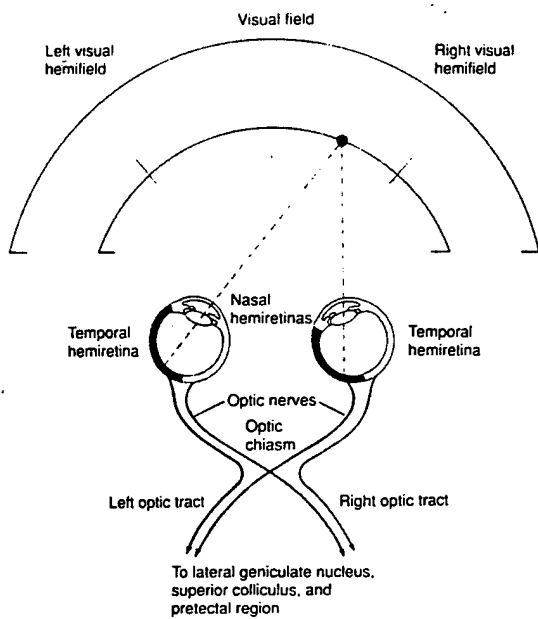
1. Introduction

Computer vision deals with algorithms that allow machines to detect, segment, feature extract, and recognize objects in an image. The algorithms can then be implemented and transferred to integrated circuits and allow machines to "see." There are numerous applications in medicine, manufacturing, and security for this technology. Studying the visual processes of biological systems can greatly enhance the development of computer vision algorithms, by incorporating biological functions that perform tasks so efficiently. Recently, oscillations in the brains of cats due to visual stimuli have led to the development of several models that mimic the interacting neurons. Results of this work may lead to a computer model that segments objects in an image. This paper presents how biological exploration may be leading to tools that can improve advancements in the area of computer vision.

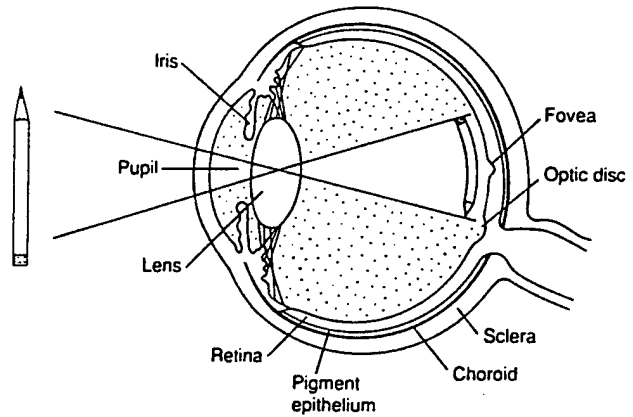
The primary visual cortex produces oscillatory pulses in response to specific stimulus. These responses are thought to be produced at the primary visual cortex and not induced by other sections of the visual pathway (see Figure 1) (Kandell, Schwartz, and Jessell 1991; Zeki 1990). Therefore, these cortical oscillations have been modeled to investigate their ability to recognize objects in a visual field. There are three models that can be used to reproduce these oscillations, and they will be discussed in the following sections.

In areas 17 (A17) and 18 (A18) of the cat visual cortex (see Figure 2) (Zeki 1993), the firing of neurons in response to the presentation of optimally aligned light bars within their receptive field oscillates with a peak frequency near 40 Hz. Thus, neuronal firing pattern is tightly correlated with the phase and amplitude of an oscillatory local field potential (LFP) recorded from the same electrode. Experiments have demonstrated that local neuronal populations in the visual cortex engaged in stimulus specific synchronous oscillations resulting from an intracortical mechanism.

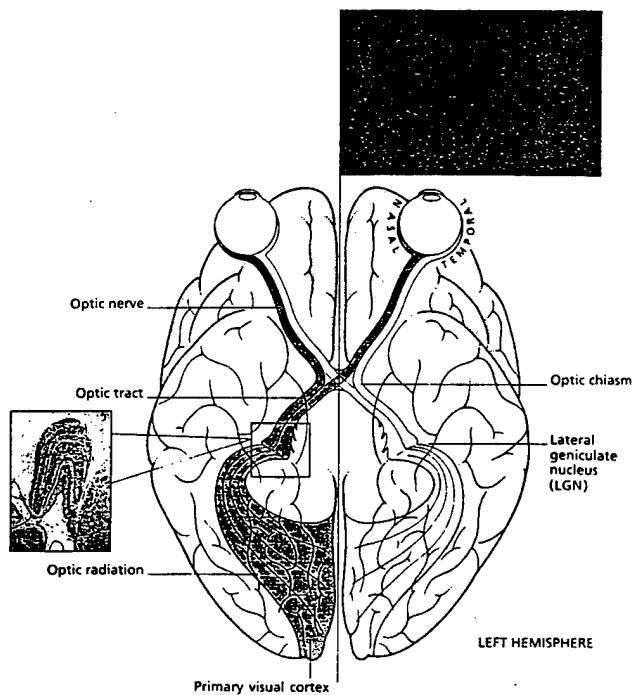
Moreover, observations in A17 of alert cats seem to indicate that the neuronal response recorded during periods of attention exhibits a rhythmic firing pattern that is tightly correlated with an oscillatory LFP having a frequency near 40 Hz. Furthermore, observations and reports show that



(a)



(b)



(c)

Figure 1. The Visual Field (a), the Lens of the Eye (b), and the Visual Pathway (c).

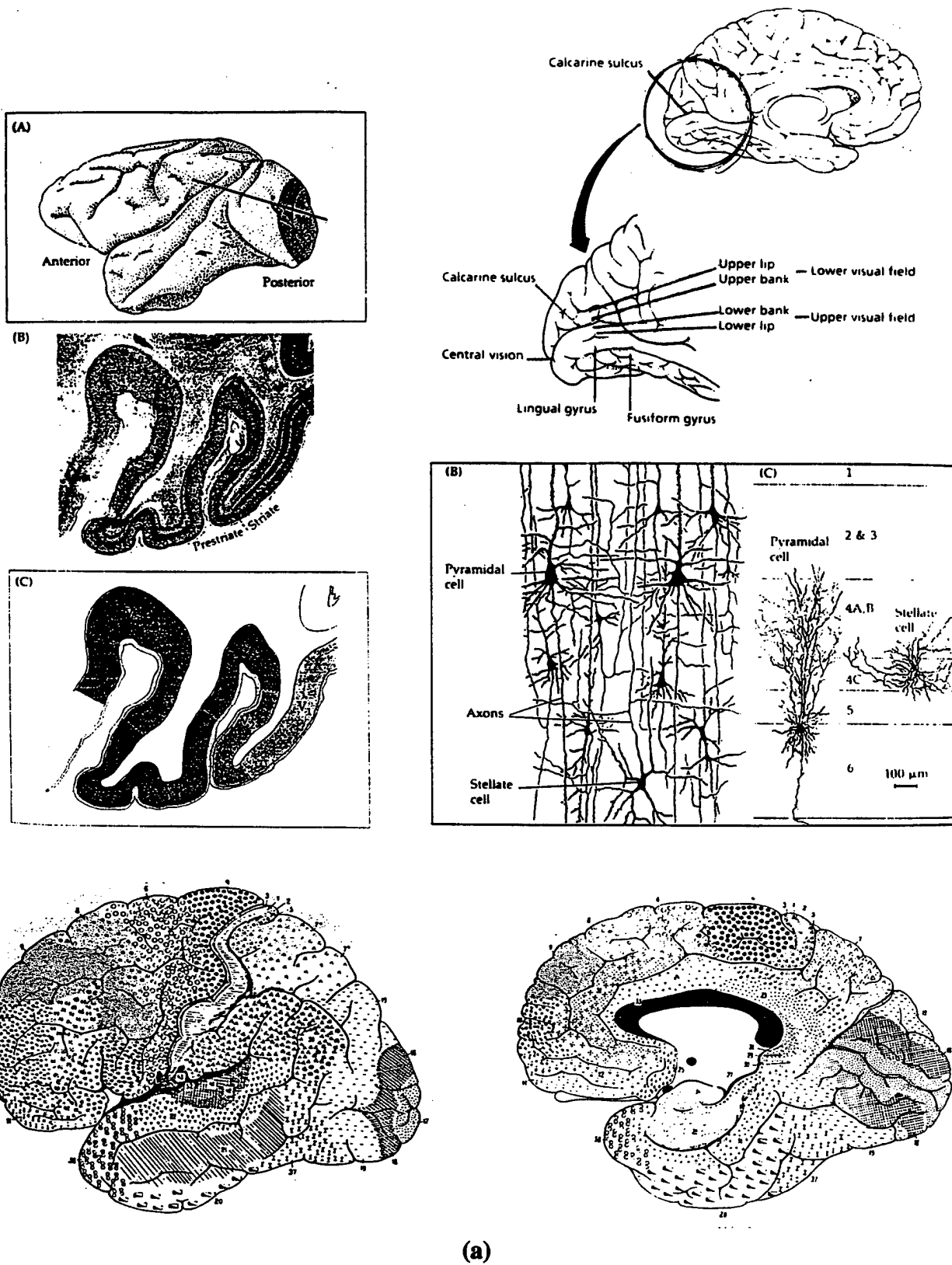


Figure 2. The Primary Visual Cortex and Brodmann's Cytoarchitectonic Chart (a) and the On-Center, Off-Surround and the Linear Receptive Fields (b).

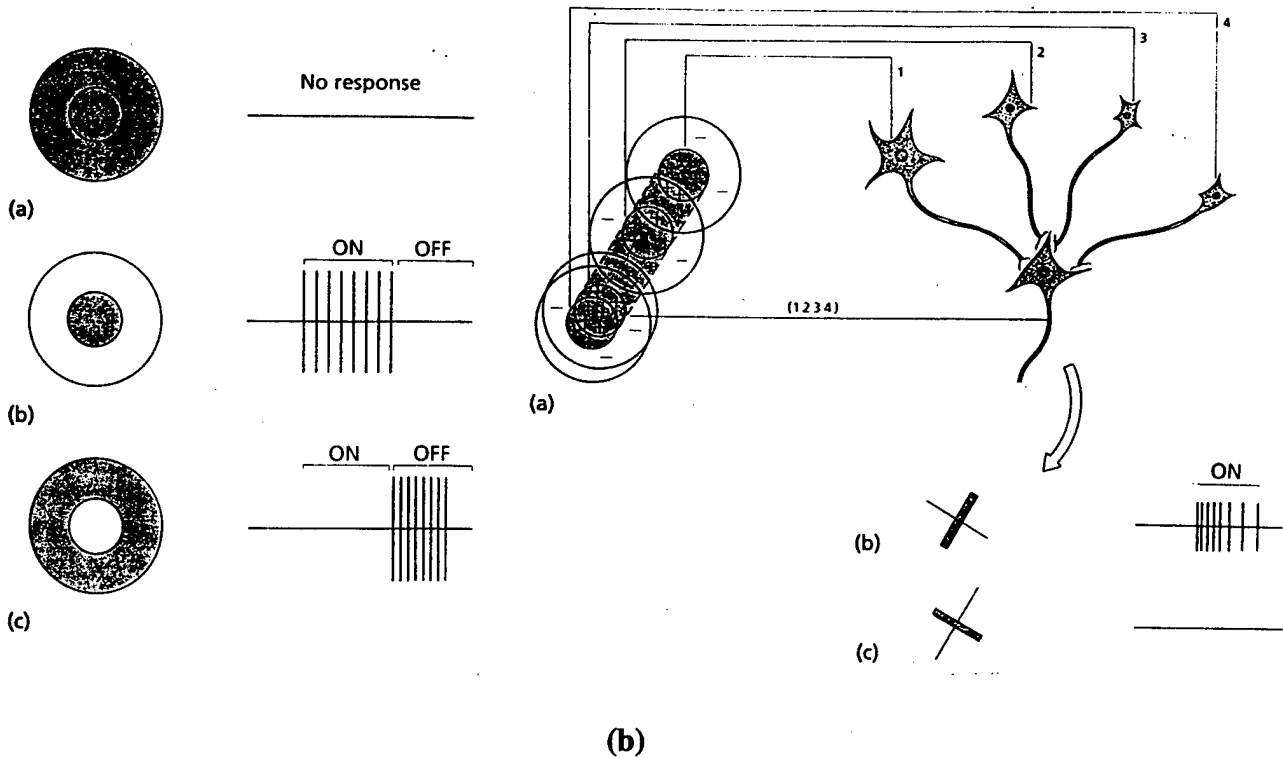


Figure 2. The Primary Visual Cortex and Brodmann's Cytoarchitectonic Chart (a) and the On-Center, Off-Surround and the Linear Receptive Fields (b) (continued).

local groups of neurons within functional columns of the visual cortex engaged in stimulus-specific oscillatory responses having a frequency near 40 Hz. These results suggest that the temporal patterns of the oscillatory response can be used to synchronize the activity of neuronal populations in spatially separate regions of the cortex.

As a result of the experimentation described, it can be concluded that these tightly correlated stimulus-induced oscillations may play a role in recognition of images (stimuli). Specifically, if these 40-Hz oscillations are specific in reference to the applied stimulus, then a "map" is formed between the stimulus and the subsequent neurons that fire. Therefore, the observation of the firing pattern of the neurons may be used to "identify" the stimulus that was applied to the visual field.

A brief look at the visual system will be presented in this report. Emphasis is on those areas leading to where these internally stimulus-dependent oscillations occur, the biological testing done

to observe the oscillations, and the models that attempt to reproduce this biological phenomena. This information is used as the basis for a model that attempts to utilize these neuronal responses for segmentation of objects in an image.

This report is organized as follows: (1) a general overview of the anatomy of the visual system; (2) a discussion of the biological test conducted to observe the cortical oscillations; (3) an explanation of the models developed to simulate the cortical oscillations found in the visual system; and (4) a discussion of questions and areas of further research.

2. General Anatomy of the Visual System

In order to determine how the visual system represents images in the visual field and what responses are initiated in the primary visual cortex, a general overview of the visual system is presented in succeeding text. The parts of the visual system discussed are the visual field, the visual pathway, and the primary visual cortex.

The visual field is the view seen by two eyes without movement of the head. The left half of the visual field projects to the nasal hemiretina of the left eye and on the temporal hemiretina of the right eye, and similarly for the right half of the visual field (see Figure 1) (Kandell, Schwartz, and Jessell 1991). In addition, the lens of the eye inverts the visual image that is projected onto the back of the retina.

The optic disc is located in the back of the retina below the fovea. It can be pinpointed as the region where the ganglion cells leave the retina. These ganglion cells form the optic nerve. The optic nerve from one eye joins at the optic chiasm with that of the other, which allows the nerves to continue traversing the brain. Fibers from both eyes enter the optic tract and project to the lateral geniculate nucleus (LGN). The LGN is a section of the cerebral hemisphere. Specifically, the fibers from the nasal half of each retina cross to opposite sides of the brain and axons from the temporal half project to the same side of the brain to the LGN. Therefore, the left optic tract is the section of the pathway that carries a complete representation of the right half of the visual field to the LGN and

similarly for the left. The LGN has six layers of cell bodies. The ventral layers contain large cell bodies and, therefore, is called magnocellular (M) layers. The dorsal layers contain the small cell bodies and is called parvocellular (P) layers. The M cells and the P cells project to the primary visual cortex. The primary visual cortex is hidden within the calcarine sulcus area on the medial surface of the hemisphere (see Figure 2a). The primary visual cortex represents the visual field by transforming receptive fields into linear segments and boundaries. The visual cortex is subdivided anatomically into five areas, labeled V1–V5. The primary visual cortex is considered V1 and V2, also known as Areas 17 and 18 (Brodmann's cytoarchitectonic chart of the brain).

The cells found in the primary visual cortex are pyramidal and stellate cells. Pyramidal cells are large and have long spiny dendrites. They project to other areas of the brain. On the other hand, the stellate cells have spiny and smooth dendrites. They are found solely in the primary visual cortex. The pyramidal and spiny stellate cells are excitatory and use glutamate or aspartate as their neurotransmitter. The smooth stellate cells are inhibitory cells and use GABA as its neurotransmitter. Once nerves from LGN enter the primary visual cortex, information flows from one cortical layer to another, starting with spiny stellate cells. The spiny stellate cells project to the pyramidal cells to form the excitatory path. The pyramidal cells excite inhibitory, smooth stellate cells, modulating the firing of the excitatory cells.

Several stimuli could be used to mediate the firing of these cells. However, experiments indicate that a small spot of light is the most effective stimulus for the retina, the LGN, and the input layer of the cortex. Neurons in the LGN and retina are known to have an on-center, off-surround receptive field (see Figure 2b). For an on-center, off-surround receptive field application of diffused light initiates no response; if light is shown in the center, a response is initiated. If no light is shown in the surround area (outside the smaller circle but inside the larger circle), a response is initiated. These responses are the firing of the cells in the visual cortex.

Cells in the primary visual cortex do not have circular receptive fields, and they respond best to stimulus that is linear. These cells are divided into simple and complex cells. Simple cells and complex cells have receptive fields that have a specific axis of orientation. A cell can have a

rectangular receptive field where one area is excitatory and the other area is inhibitory. Complex cells respond most effectively to a moving bar of light. Thus, a stimulus must excite a segment of the retina and have a specific axis of orientation. A bar of light with a particular orientation or a spot of light initiates a response by exciting several neurons. When the orientation of the bar of light or the location of the spot of light is changed, then the response is diminished by the exciting inhibitory neurons.

The overview of the anatomy is needed as background to understand how the visual system functions. Now that the general structure of the visual system is presented, our attention can be focused on the primary visual cortex where the oscillations occur. The biological test and subsequent models focus on the response observed in the primary visual cortex. The models are empirical in nature and do not model the visual cortex itself.

3. Biological Testing

The biological tests were experiments performed on cats. The cats were prepared for surgery by injecting short-acting anesthesia that was maintained during recordings. Recordings were taken 3–4 hrs after the initial anesthesia to ensure any negative affects had sufficiently worn off. The neuronal activity and voltage readings were recorded from 25- μ m-diameter electrodes (see Figure 3a) (Gray and Singer 1989; Leibovic 1990).

This section examines the biological tests that were performed in A17 and A18. The general test was conducted by placing electrodes in the primary visual cortex. A horizontal bar of light moving vertically down was used as the stimulus. For this stimulus, an increase in voltage was measured and an excitatory response occurred. When the bar of light was moved vertically up, an excitatory response was measured also. For that orientation of the stimulus, a positive voltage and an excitatory response occurred. When a vertical bar of light moving horizontally to the right was used, a negative voltage was measured and an inhibitory response was elicited. When the bar of light was moved horizontally to the left, an inhibitory potential was measured again. For this particular orientation, a negative voltage and an inhibitory potential were measured. If the stimulus is removed, the

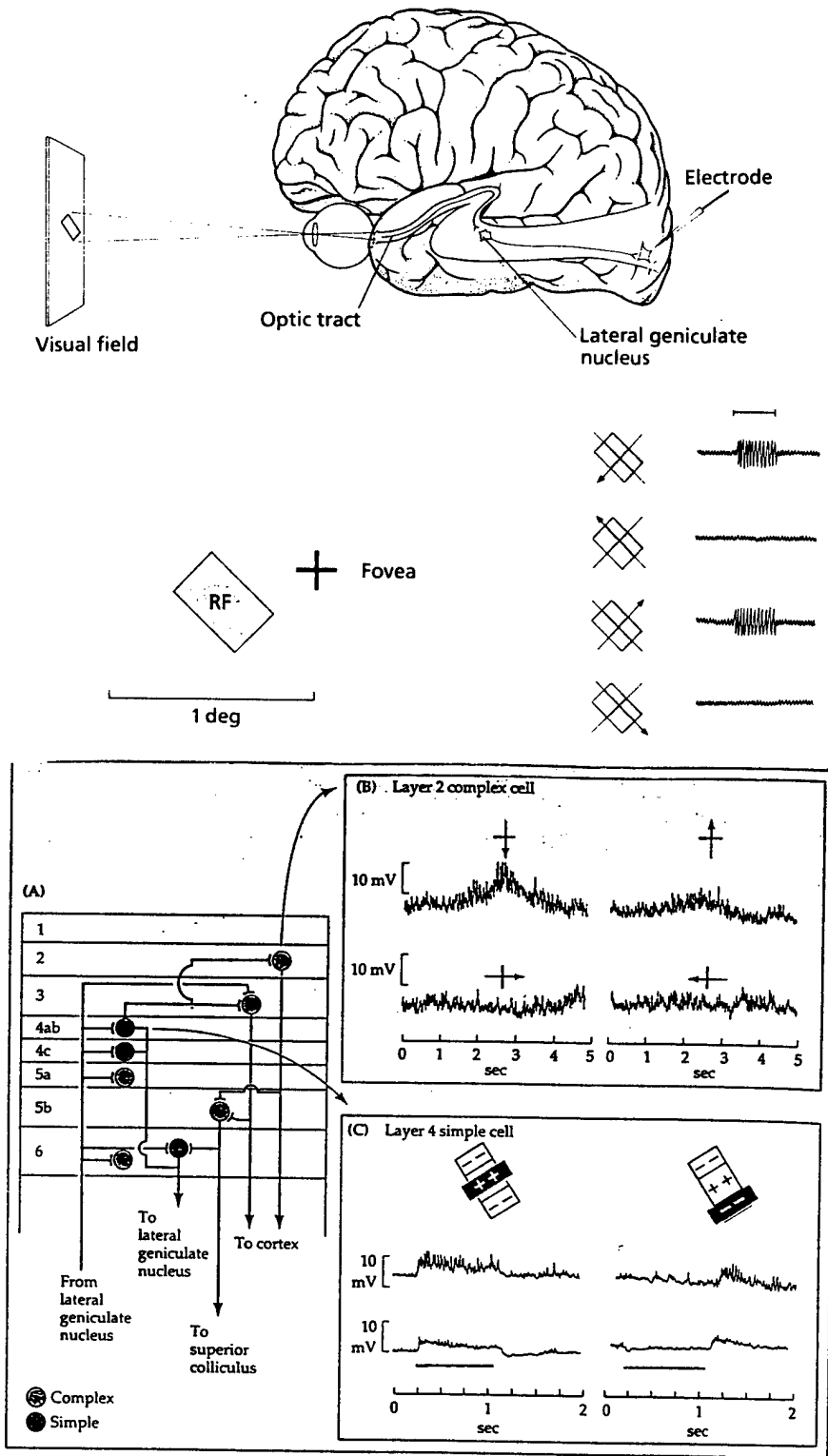
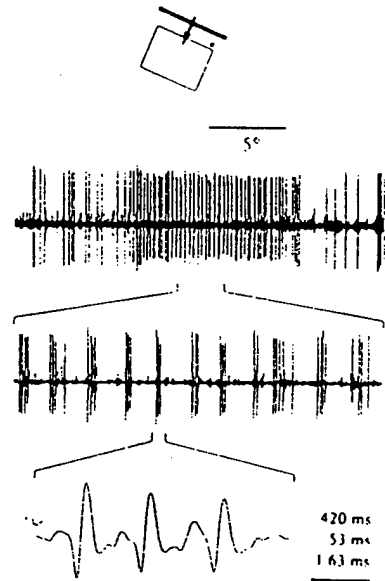
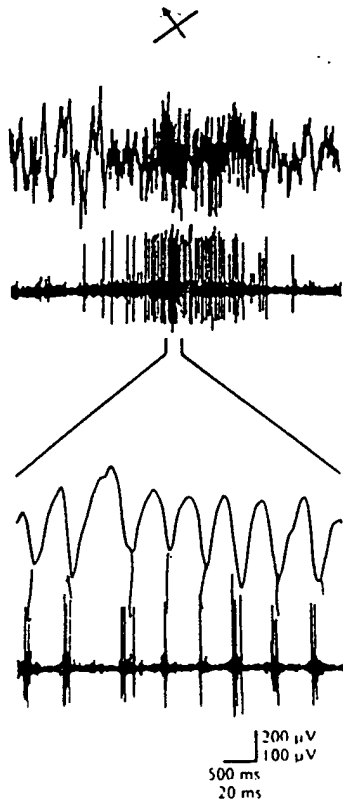


Figure 3. Electrodes in the Primary Visual Cortex and the Responses to Specific Stimuli (a) and Multiunit Activity (MUA) and LFP (b).



(b)

Figure 3. Electrodes in the Primary Visual Cortex and the Responses to Specific Stimuli (a) and Multiunit Activity (MUA) and LFP (b) (continued).

response, whether excitatory or inhibitory, is removed. Hence, a spot of light or a moving bar of light can be used as a stimulus to elicit firing of neurons in the primary visual cortex. Therefore, the presence of an effective stimulus results in the firing of the corresponding neurons.

The experiments performed by Gray and Singer also involved placing electrodes in the visual cortex of a cat. A stimulus was applied in the visual field, and recordings of neuronal responses in the visual cortex were taken. The receptive field properties of the neurons firing were recorded from each electrode with a bar of light projected on a screen in front of the cat's eye plane. The measurements taken from the electrode include the LFP, which is the average voltage, and the MUA, which indicates the neurons firing (see Figure 3b).

The recordings from areas 17 and 18 in an adult cat when a light bar of optimal orientation was passed through the receptive field show a rhythmic firing pattern. This neuronal spike train was associated with a high-amplitude oscillation of the LFP. The spike occurred during the negative phase of the LFP oscillations, indicating periodicity in the firing of neurons. It is noted that the highest frequency of the LFP directly correlates with the maximum number of neurons firing in the MUA. Computation of the power spectra of the LFP was used to support that the oscillatory activities of the neurons were stimulus-dependent.

The average peak frequency recorded of the LFP was between 35 and 49 Hz. It was observed that the amplitude and latency of the response are similar for the LFP and the MUA. It was found that oscillatory responses occurred much more frequently in the complex cells as opposed to the simple cells of the primary visual cortex.

The results of these experiments suggest that during responses to light stimulus, adjacent neurons have a strong tendency to act simultaneously and synchronously. These oscillations result from intracortical mechanisms and not by oscillatory inputs from the LGN. In addition, results suggest that the synchronous oscillation of ensemble neurons in the frequency range of 35–50 Hz is an integral part of the neuronal response in the visual cortex.

In addition, the field potential has revealed the oscillatory behavior of the neuronal response, and because of its close correlation to the MUA, this field potential seems to reflect the synchronous activity of a population of cells.

Although the mechanism of the oscillations was unknown, it suggested that the interaction among a population of synaptically coupled neurons, both excitation and recurrent inhibition, was sufficient for generating oscillations. Experiments also show that when activated appropriately, groups of adjacent cortical neurons change in cooperative interactions. These interactions led to coherent and periodic patterns of activity.

4. Modeling of Neuron

4.1 General Model. A variety of models for the cortical oscillations found in the visual system have been proposed. These include a general model and models developed by H. J. Eckhorn and J. L. Johnson. The general model of an artificial neural net (ANN) is an artificial neuron (AN) that receives inputs from a number of other ANs or from an external stimulus. A weighted sum of these inputs constitutes the argument to a function. The resulting value of the function is the output of the AN. The output mimics the firing of a biological neuron. This output gets distributed along weighted connections to other ANs. Therefore, an artificial neuron anatomically models a biological neuron.

Looking briefly at a biological neuron, we see that it is composed of dendrites, a cell body, an axon, and synaptic buttons (see Figure 4) (Vermuri 1992). This is a very elementary view of a real neuron. The branching structure are dendrites. Dendrites are where the neuron picks up signals from other neurons. The cell body, or soma, is where the axon hillock acts as a threshold function. The axon hillock determines if a neuron fires (generates an action potential) based on a comparison of the membrane potential and the threshold value. The long transmission line-like structure would be the axon, and the action potential propagates down the myelinated axon to the presynaptic terminal. The brushlike structures at the end of the axon are synaptic buttons. Synaptic buttons have vesicles which release neurotransmitter (NT) from the presynaptic terminal.

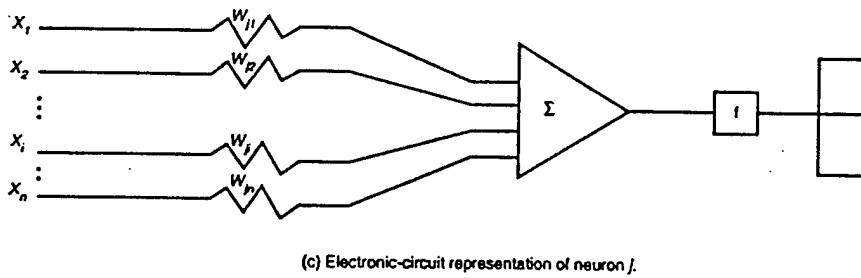
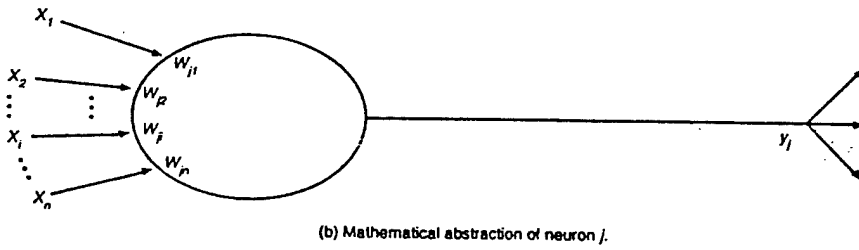
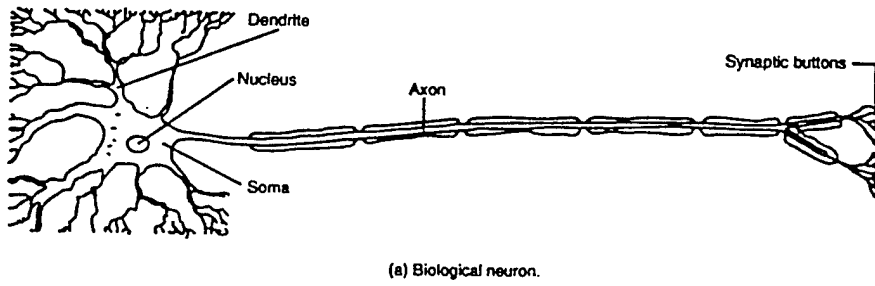
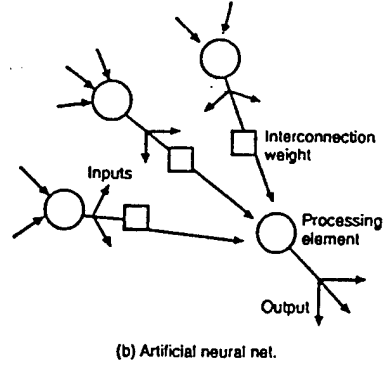
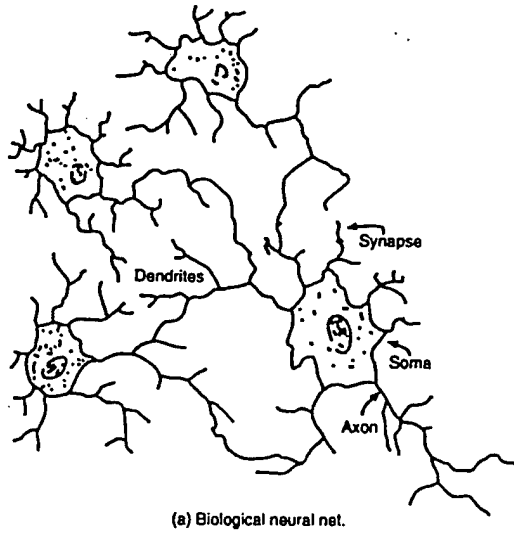


Figure 4. General Model of Neuron and Neural Net.

In a mathematical abstraction of a neuron, X_1-X_n represents the inputs received by the j^{th} neuron, W_{ji} represents the synaptic strengths, and Y_j is the output of the j^{th} neuron. A neuron receives signals from its neighbors via the synapses and performs a weighted algebraic summation on the inputs. It computes a thresholding function and produces an output based on this sum.

A diagram of a neural net shows the points where neurons come close together. These points form the synapses. It is at this point of contact that the synaptic transmission takes place. The neurons influence each other electrochemically. When a signal arrives at a synapse, it elicits the release of an NT. The NT changes the potential of the neuron. When that potential exceeds a certain threshold, an action potential is elicited in the receiving cell.

A biological net would include multiple neurons with the dendrites making multiple connections between neurons. An artificial neural net would then include multiple copies of the modeled neuron with interconnecting weights and the output that is determined by a processing element.

This is the basis for modeling of the neuron and neural net. However, each component of the model requires detail. In Eckhorn's model, the processing elements are developed. He discusses the type of inputs required, the subdividing of the cell body into compartments that perform certain functions, and the parameters that generate an output.

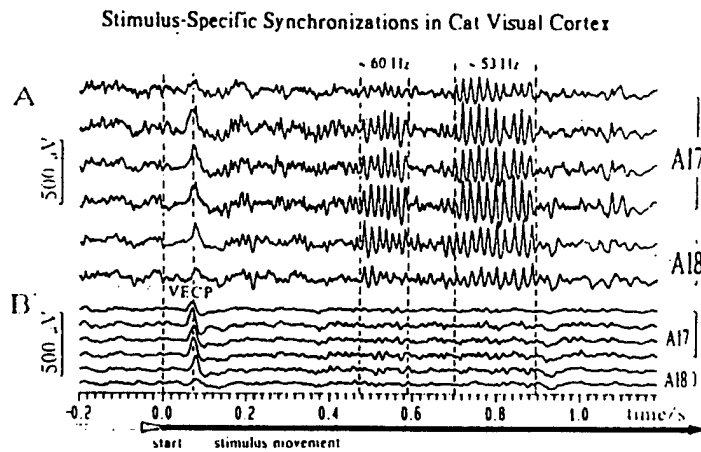
Of interest are two models that recreate the neuronal response seen in the studies by Gray and Singer. The first model is by Eckhorn and closely recreates the responses of the biological system. The second model is by Johnson. Johnson's model takes Eckhorn's model and attempts to modify it to use the patterns of the neuronal response to segment images in a visual field.

4.2 Eckhorn's Model. Eckhorn discovered that stimulus-induced oscillatory activities (35–80 Hz) in cells of the visual cortex synchronize the response of the cell if a common stimulus drives the cells. He proposed that synchronization is a general principle for coding sensory systems and that there are at least two types of synchronizations. These synchronizations are stimulus-forced (event-locked) synchronizations and stimulus-induced (oscillatory) synchronizations.

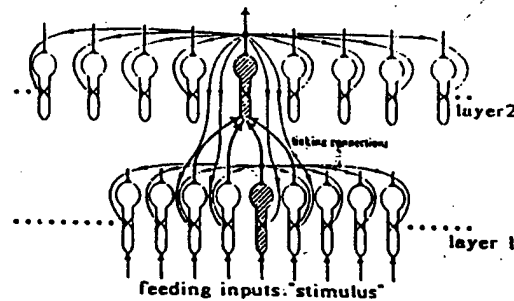
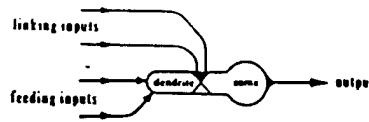
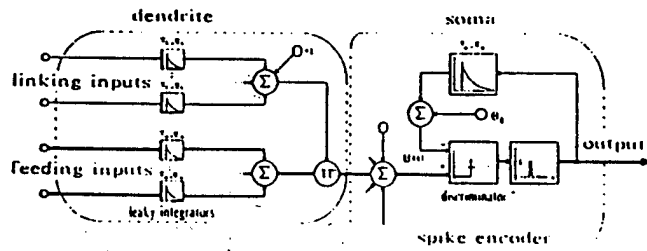
Eckhorn recorded large oscillation amplitudes of LFPs and MUAs that were induced by sustained binocular stimuli. Stimulus-induced oscillations of LFP and MUA appeared as oscillation spindles of 80–250 ms long, separated by intervals of stochastic activity, and their response latencies were longer than stimulus-locked, visually evoked cortical potentials (VECP). Stimulus-forced synchronizations (VECPs) are driven by a stimulus that is rapidly passed across the field of view and are usually not oscillatory. The stimulus-specific response is a series of oscillating voltages at approximately 53 and 60 Hz (see Figure 5a) (Eckhorn et al. 1989). Stimulus-related oscillations of neural activities were discovered in the primary visual cortex of the cat through studies by Gray and Singer (1987) as mentioned earlier and by Eckhorn et al. (1988). Further studies by Gray and Viana Di Prisco (1993) indicated that stimulus-dependent oscillations of 30–60 Hz have been confirmed in single-electrode recordings in A17 of alert cats. These findings with studies by Grossberg (1983) and Reitboeck (1983) support the hypothesis that synchronization may be a mechanism for carrying out the linking of local visual features into impressions of objects.

Stimulus-specific synchronization was observed in different cortex areas if the neurons coded common visual features. As mentioned earlier with the work done by Gray and Singer, signal oscillations are generated in the cortex, and these oscillations can be synchronized by stimulation of neurons in their receptive fields. On the other hand, stimulus-induced synchronizations are recorded as oscillatory mass activities and are assumed to be produced by an internal mechanism. It is thought that this internal mechanism is the process achieved from stimulus-driven local oscillators that are mutually connected. Simulations have been done to support this assumption. These stimulus-specific oscillations are the responses modeled by Eckhorn.

The stimulus-induced synchronous oscillatory potential found to take place in the visual cortex were observed using computer simulations of neural network models. Eckhorn's model has two types of synapses: (1) the feeding synapses, which are connected directly to the stimulus that drives the neuron and (2) the linking synapses which receive auxiliary signals that modulate the input from the feeding synapses (see Figure 5b) (Eckhorn et al. 1989). The linking inputs are considered the synchronizing signals.



(a)



(b)

Figure 5. Stimulus-Specific Synchronizations in Cat Visual Cortex (a) and Eckhorn's Model of Neuron and Neural Net (b).

The model neuron has dynamic synapses represented by leaky integrators. During a synaptic input pulse, the integrator is charged and its output amplitude rises steeply. This is followed by an exponential decay determined by what is called the leak time constant. The spike encoder is represented by a leaky integrator. The spike encoder includes a differential amplitude discriminator and a spike former. The amplitude discriminator triggers the spike former when the input exceeds a variable threshold. The output of the neuron immediately charges the leaky integrator and raises the value of the threshold. This new threshold value exceeds the input, preventing an output immediately following a previous output. The elevated threshold produces the refractory period in the spike generator. Another output is not generated until the threshold value decays below the membrane potential.

Eckhorn believes that the concept of a modulatory synapse is supported by modulation seen in real neurons. These modulations may be achieved by changing dendritic membrane potentials through voltage-dependent channels that affect synaptic efficacy. From the simulations, feeding signals with modulation cause the model to respond initially with irregular receptive discharges, but subsequently, the neurons mutually synchronize their activity through the linking connections. This supports the phase locking of stimulus-induced synchronizations in the simulation.

4.2.1 Computational Aspects. Eckhorn's neural network consist of two layers of neurons. The bottom layer (layer 1) consists of several neurons, and each layer 1 neuron receives inputs from the stimulus as feeding inputs and receives input from the four closest neighboring neurons as linking inputs. The top layer (layer 2) consists of several neurons, and each layer 2 neuron receives feeding inputs from the four closest neurons in layer 1 and receives four linking inputs from its four closest neighboring neuron in layer 2. Furthermore, each layer 2 neuron sends feeding inputs to the four closest layer 1 neurons. Eckhorn models his individual neuron as a dendrite and soma, with the output being a pulse and the input being the feeding and linking inputs. The linking inputs and feeding input are modeled as leaky integrators. Each neuron is modeled to have multiple feeding and linking inputs. The linking inputs are added to a constant value of one, then multiplied with the feeding input to form the input to the soma. The soma is modeled as a spike encoder. The spike encoder is where the output from the dendrite is compared with a preset threshold value. If the value

is greater than the threshold, the amplitude discriminator triggers the spike former to output a pulse. This output is fed back into the system to change the value of the threshold to prevent the generating of additional pulses.

The equations that follow describe the model parameters (see Figure 6). The output of the dendrite is the membrane voltage $U_{m,k}(t)$ for the K^{th} neuron:

$$U_{m,k}(t) = [F_k(t)] \cdot [1 + L_k(t)].$$

The feeding input:

$$F_k(t) = \sum_{i=1}^N [W_{ki}^f Y_i(t) + S_k(t) + N_k(t)] * I(v, \tau, t).$$

The linking input:

$$L_k(t) = \sum_{i=1}^N [W_{ki}^l Y_i(t) + N_k(t)] * I(v, \tau, t).$$

The threshold:

$$\Theta_k(t) = \Theta_0 + Y_k(t) * I(v, \tau, t).$$

The output of the spike encoder of the K^{th} neuron:

$$Y_k(t) = 1 \text{ if } U_{m,k}(t) \geq \Theta_k(t)$$

$$Y_k(t) = 0 \text{ elsewhere.}$$

The impulse response of the leaky integrator:

$$I(v, \tau, t) = V \cdot \exp(-t/\tau) \quad \text{if } t \geq 0$$

$$I(v, \tau, t) = 0 \quad \text{elsewhere,}$$

The membrane voltage $U_{m,k}(t)$ of the k th neuron is given by

$$U_{m,k}(t) = F_k(t) \cdot [1 + L_k(t)] \quad (\text{A.1})$$

where $F_k(t)$ is the contribution via the feeding-inputs and $L_k(t)$ is the contribution via the linking-inputs.

$F_k(t)$ is calculated from

$$F_k(t) = \sum_{i=1}^N [w_{ki}^f Y_i(t) + S_k(t) + N_k(t)] * I(V^a, \tau^a, t) \quad (\text{A.2})$$

$I(V^a, \tau^a, t)$: impulse response of leaky-integrators at the feeding inputs.

$$I(V, \tau, t) = \begin{cases} V \cdot \exp(-t/\tau) & \text{if } t \geq 0 \\ 0 & \text{else} \end{cases} \quad (\text{A.3})$$

The contribution via linking-inputs (index "1") is

$$L_k(t) = \sum_{i=1}^N [w_{ki}^1 Y_i(t) + N_k(t)] * I(V^1, \tau^1, t) \quad (\text{A.5})$$

The output of the spike-encoder of the k th neuron is given by:

$$Y_k(t) = \begin{cases} 1 & \text{if } U_{m,k}(t) \geq \theta_k(t) \\ 0 & \text{else} \end{cases} \quad (\text{A.6})$$

The threshold's time course is derived by

$$\theta_k(t) = \theta_0 + Y_k(t) * I(V^a, \tau^a, t) \quad (\text{A.7})$$

with a threshold-offset θ_0 .

Figure 6. Mathematical Description of the Model Neuron by Eckhorn.

where

N is the number of neurons;

W_{ki}^f is the synaptic weight of the feeding input from the i^{th} to the k^{th} neuron;

W_{ki}^1 is the synaptic weight of the linking input from the i^{th} to the k^{th} neuron;

$Y_i(t)$ is the analog stimulus input to the k^{th} neuron in layer 1;

$N_k(t)$ is the analog "noise" input to the k^{th} neuron, statistically independent in each neuron; and

Θ_0 is the threshold offset.

4.2.2 Simulations. Eckhorn performed several tests to show that synchronization can be produced with his model (see Figure 7). The first experiment is with two stimuli each individually applied to two patches of layer 1 neurons. The stimulus applied to the second patch was half the intensity of the first patch. The pulses generated in layer 1 and 2 are initially at different frequencies for each patch. When feedback (input from the linking neurons) is not included, these pulses do not lineup (synchronize), but with feedback included, we observe that over time the pulses do align, implying synchronization. Again, the experiment is done by applying stimulus to one patch prior to the other; pulses are again generated at different frequencies for the patches. Without feedback, they do not show synchronization, but when feedback is applied, they do synchronize. This is seen when cross correlation is monitored. When there is no feedback, there is no cross correlation recorded for the two patches. However, cross correlation is recorded when feedback is present. The experiments are repeated with stimulus intensities the same; the stimulus is applied to both patches, and the resulting pulses synchronize. Then the stimulus is removed from one patch. With pulses still generated from each patch, the resulting pulses synchronize over time. The pulses generated from the patch where the stimulus was removed is not as strong as the pulses generated from the patch where the stimulus remains. The reason why the pulse from the patch where the stimulus is removed still appears may be due to signals that are sent from the linking inputs corresponding to the four neighboring neurons.

4.2.3 Behavioral Aspects. Limitations of Eckhorn's model include the arbitrary setting of the linking weights and the globally setting of constants to experimentally determined values. The constants are set to allow the model simulations to closely resemble the cats' visual cortex experimental data. These constants are not calculated from biological values.

Eckhorn believes his model supports synchronization as a way to achieve perceptual feature linking. In order to bring neuronal mechanisms of feature linking into correspondence with perceptual functions, Eckhorn introduces the concept of linking field of a local neural assembly. The linking field of the local neuronal assembly is that area in the visual space where appropriate local stimulus features can initiate synchronizations in the activities of that assembly. If this concept can be supported, then we can interpret these synchronized patterns of neural response as corresponding

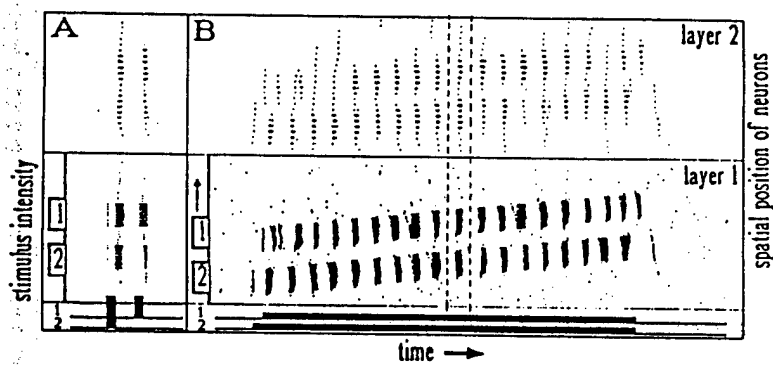
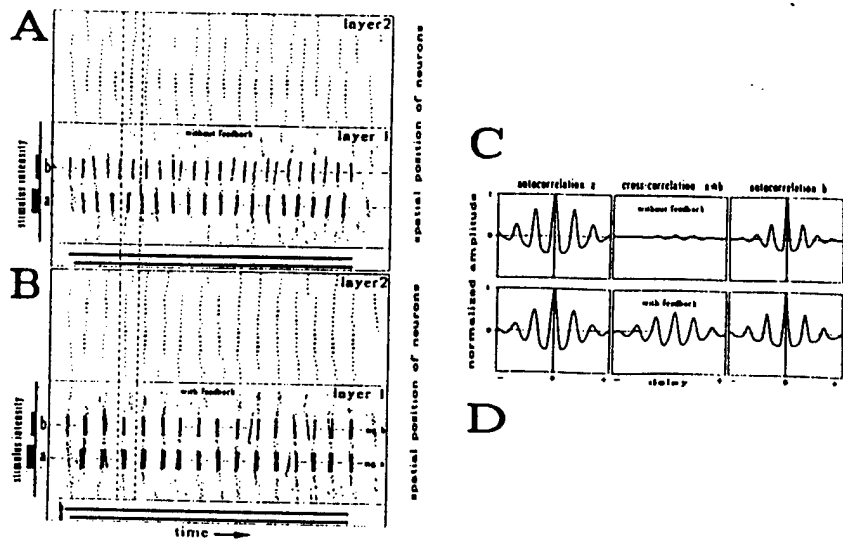


Figure 7. Neural Network Simulations.

to visual images. It has been seen that sensory systems that received well-timed signals might be dominated by stimulus-forced synchronization. Other systems may employ a mechanism of synchronized activity for oscillations and coding in the system.

4.3 Johnson's Model. The last model to be discussed is Johnson's model (see Figure 8a) (Johnson, Ranagath, and Caulfield 1994; Johnson 1994). Johnson's model does not correlate with the biological system as strongly as Eckhorn's model. Johnson's model is a modified version of Eckhorn's model. It only models a one-layer network but with multiple receptive fields. Johnson's model includes a dendritic-tree section, a linking section, and a pulse-generator section. The dendritic tree is similar to Eckhorn's dendrite. The dendritic tree includes the feeding inputs from other neurons, as well as the linking inputs from other neurons. The difference in this section is that the linking inputs are multiplied by a variable beta before the constant is added. Then this value is multiplied with the feeding input in the linking section. Moreover, in the linking section, linking inputs from other receptive fields are multiplied with the inputs from the current receptive fields. This value is then summed with inputs from other dendrites. The pulse-generator section includes a threshold discriminator which triggers the pulse former to output a pulse when the membrane potential exceeds the threshold value. The output is also feedback to change the value of the threshold as in Eckhorn's model.

4.3.1 Computational Aspects. It is helpful to look at an individual neuron from Johnson's model to describe what functions beta and tau-c perform (see Figure 8b). These variables are experimental and have not been shown to model biological parameters. Beta is called the weak-linking variable. Beta sets the strength of the linking inputs' effect on the feeding inputs. Tau-c is the time constant that defines the pulse-capture zone. This zone indicates a period of time where a linking input can influence the frequency of the output pulses. Although these constants are not biologically based, they do play an important role in the model.

In this model, when there is no feedback from the neighboring neurons (no linking input), beta (the weak linking variable) is set to zero. When there is feedback from the neighboring neurons, beta is greater than zero. To understand this concept, the output of a single neuron with beta equal to zero

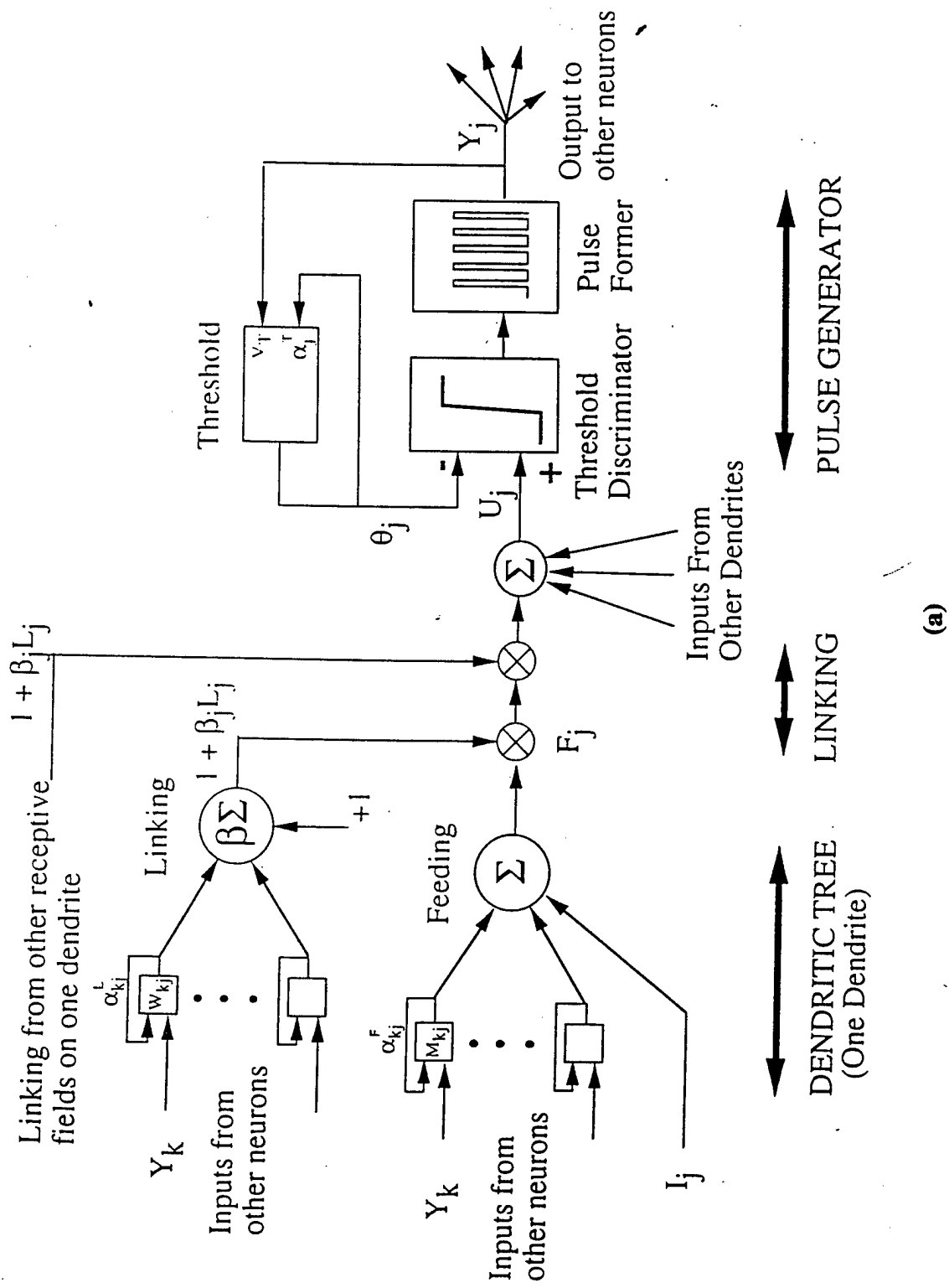


Figure 8. Johnson's Model of the Neuron and the Neural Net (a) and Neuron Function (b).

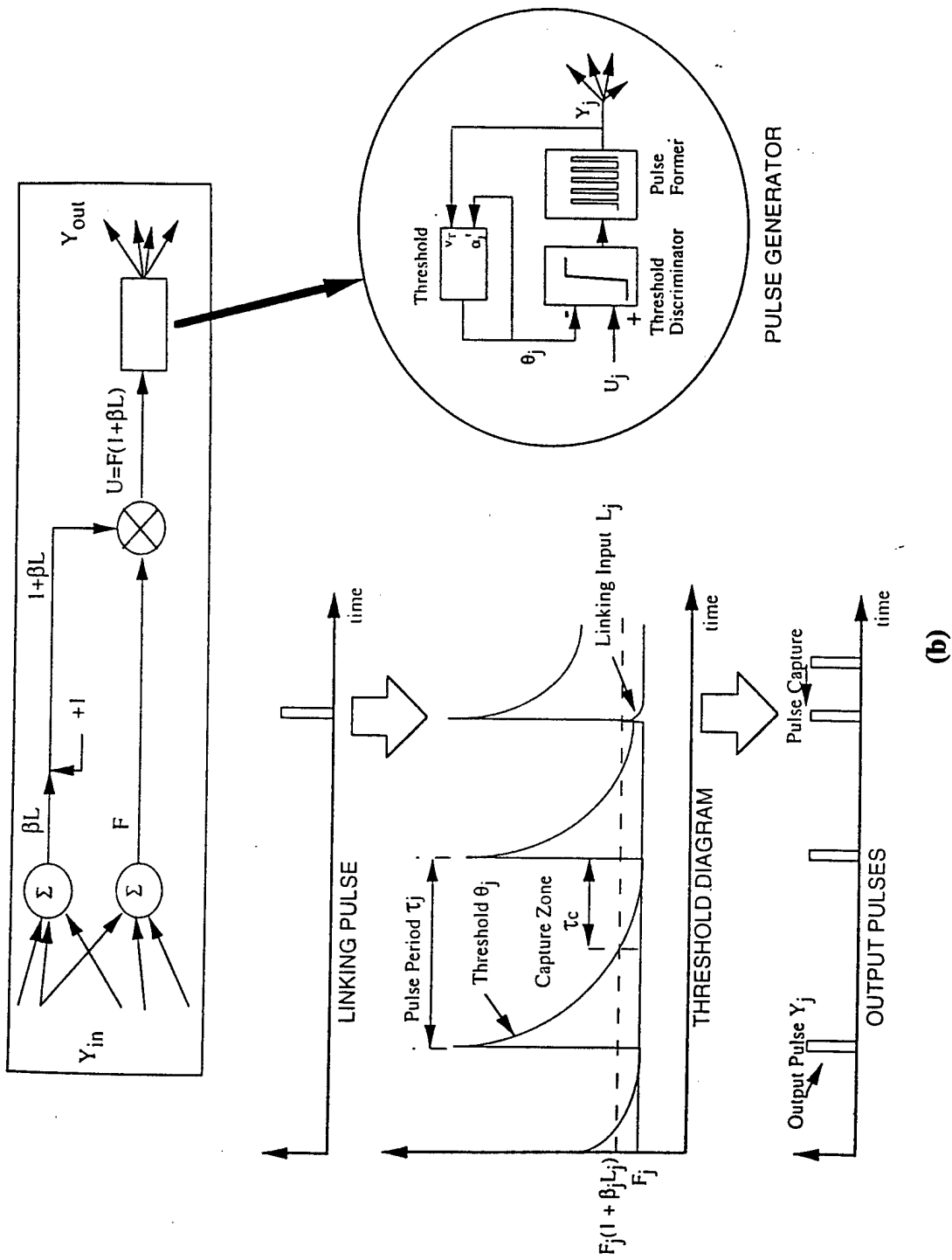
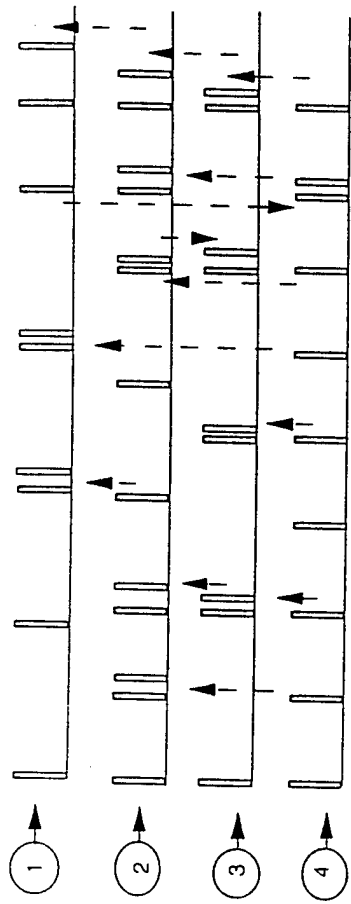


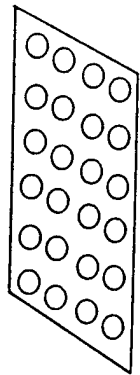
Figure 8. Johnson's Model of the Neuron and the Neural Net (a) and Neuron Function (b) (continued).

is examined. With beta equal to zero, the membrane potential is equal to the feeding input only. When the membrane potential exceeds the threshold value, a pulse is generated. If the threshold value is increased to a value greater than the membrane potential, then the threshold value decays with time. When the membrane potential exceeds the threshold value again, then another pulse is generated, and the cycle repeats. This time, where the threshold is decaying prevents another pulse from generating immediately after a pulse, thus setting the refractory period or the pulse period. This is the same way Eckhorn's model operates. Now let us consider when beta is greater than zero. A pulse has just occurred, the threshold value is increased and starts to decay. If a linking input is added to the membrane potential, it causes the value of the membrane potential to increase. By the same token, if the membrane potential is greater than the threshold value, a pulse is generated. But this pulse has occurred sooner than expected. This implies that the linking input (or the feedback from the neighboring neurons) has changed when the pulse occurs. The linking input synchronizes the pulses to some desired pattern. This effect of the linking input is also found in Eckhorn's model; however, the linking input is multiplied by a constant of one, not beta. Thus, in Johnson's model the linking inputs for the current neuron can be weighted differently from linking inputs of neurons in other receptive fields. These weights are set arbitrarily by Johnson. Since these weights are not from biological responses, they do not represent synaptic strengths generated by a specific stimulus. Therefore, this model does not use these weights to represent data.

Taking this concept further, Johnson looks at four individual modeled neurons, each neuron outputs a pulse at its own frequency (see Figure 9). When a neuron receives a linking input from another neuron within the tau-c pulse-capture zone, the neuron generates an output pulse sooner than it normally would (i.e., the output pulse frequency is increased). Successive interaction through the linking inputs readjust the pulses from each neuron, thus synchronizing them. If the pulses from the four neurons are added together, a unique pulse train is formed. The pulse train is then considered the response for a particular stimulus or image in the field of view. Each image generates its own pulse train. This idea could be justified because a stimulus does produce oscillations in the neurons of the primary visual cortex. Johnson's theory also assumes that this pulse train repeats over some interval; each image produces a periodic pulse train.



Input Intensity Scene



PCNN Slab

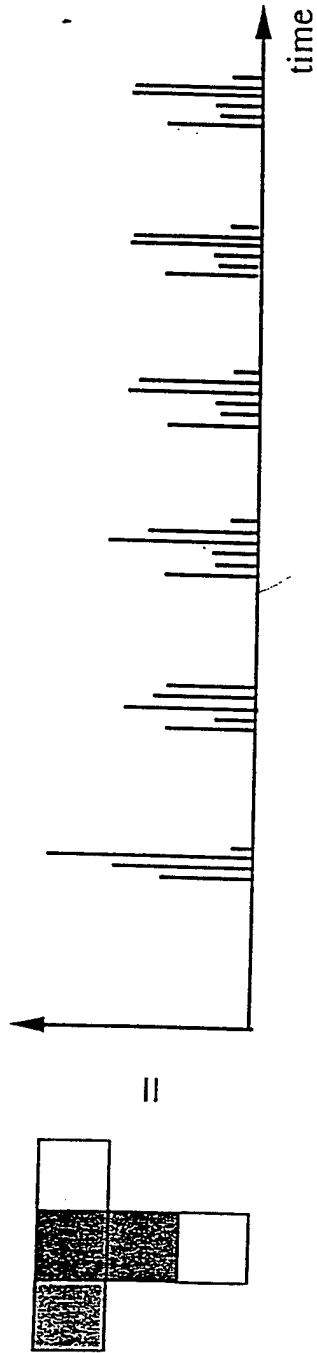
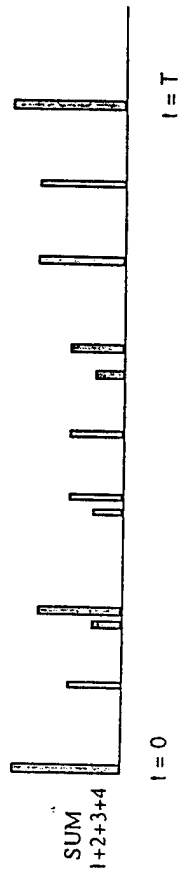


Figure 9. Time Series.

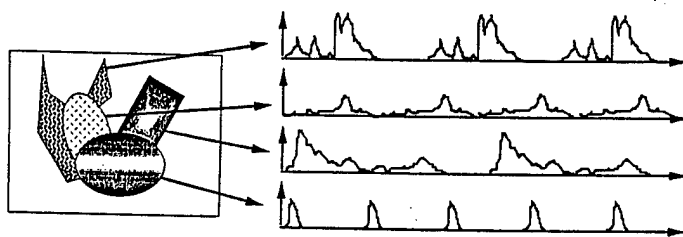
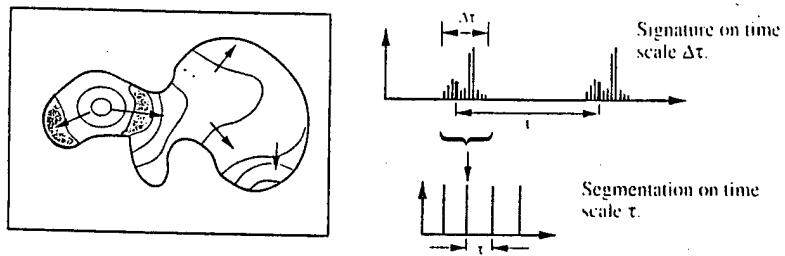
4.3.2 Simulations. Johnson simulated his model in order to observe if any meaningful data could be extracted. For example, Johnson simulates a T shape with varying intensity blocks from white to black (see Figure 9). The image produces its own periodic pulse train. The fourth and fifth interval and presumably the following intervals would be identical. The simulation shows that for the first four intervals, the pulse train varies. The theory is that these pulse trains represent changes as the linking inputs between neurons modulate the output of the neurons; this is then the time where the synchronization is occurring.

The pulse train is generated in the simulation for the images starting at the highest intensity section and propagating to the section with the lowest intensity (see Figure 10a). Thus, if you look at an image with four different intensity patterns, each pattern produces its own periodic pulse train or, as the simulation shows, its own propagating wave. These values are arbitrarily set; hence, there are no data to show what the interval of time to synchronize might be.

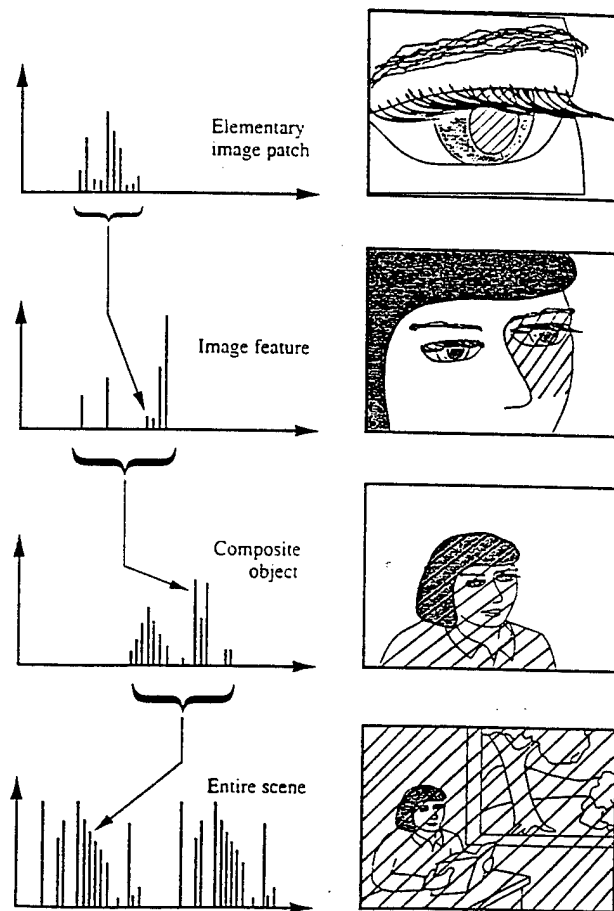
4.3.3 Behavioral Aspects. Johnson proposes that the periodic pulses that are generated by a particular stimulus can be used to identify segments of an image (see Figure 10b). Each small section of an image would produce its individual pulse train. As the field of view is expanded, it would produce its individual pulse train until the entire scene is covered. The smaller pulse trains would actually become subgroups within the pulse trains produced by the larger section of the scene. Therefore, you would have a supergroup pulse train that would represent the entire scene and would consist of subgroups of pulse trains at each level of the expanded view.

5. Conclusion and Issues

This report has presented three models that attempt to describe the oscillations produced in the primary visual cortex. Even though these models do not model the biological system, they do allow exploration into the response of the biological system. Modeling the oscillations found in the visual cortex has provided a possible tool for segmenting images.



(a)



(b)

Figure 10. Segmentation of Patterns (a) and Subgroups and Supergroups of Pulses (b).

Synchronous oscillations are found in the visual cortex. However, more investigation would help determine how these oscillations correspond to various stimulus. Stimulus-induced oscillations seem to indicate that feedback increases the pulse frequency and causes pulses to occur periodically. However, interaction between the feeding and linking inputs must be examined more closely to understand this function. Moreover, the weak linking variable and the pulse capture zone concept used to create periodic pulse trains needs to be investigated to determine its link with the biological system. Eckhorn's and Johnson's models can be viewed as first-stage systems. These models use the oscillatory property that can be achieved in modeling of neural networks to reproduce the response of neurons found in the visual cortex.

The work done by Eckhorn and Johnson raises several interesting questions. Some of these questions include:

- (1) Are responses to stimuli the same for different levels of alertness?
- (2) How useful are the models as they move away from biology (the beta and tau-c experimental values)?
- (3) How could the individual periodic pulse trains that correlate to segmented objects be filtered?

Obviously these questions cover a wide range—from the way the biological system truly functions to the way the simulations could be validated. Addressing these issues should provide a better understanding into cortical oscillations and the way they can be used in other levels of recognition.

Further modeling can be done to incorporate more of the functionality of the real biological system. Thus, if Johnson's model can be validated biologically, then inhibitory neurons should be modeled and added to the overall network model. These inhibitory neurons can possibly define where the boundaries of the propagating waves are and, thus, define the boundaries for segmentation of objects.

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