THE ROLE OF THE TECTAL COLUMN IN FACILITATION OF AMPHIBIAN PREY-CATCHING BEHAVIOR: A NEURAL MODEL

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ABSTRACT

We use computer analysis of differential equations to study the properties of a family of models of a unit of neural circuitry in the amphibian tectum, the tectal column. Since model parameters are only loosely constrained by presently available neuroanatomical data, computer experiments are used to discriminate among various hypotheses and to suggest new experiments. Particular attention is paid to physiological data on facilitation of amphibian prey-catching behavior which lead us to model the facilitation in terms of dynamic activity in the tectal column rather than in terms of synaptic modification.

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1. INTRODUCTION

In the present paper we propose a mathematical model of the amphibian tectal column to account for the facilitatory effects of prey catching activity when prey stimuli are presented. The model is based on anatomical and physiological studies of this region, and behavioral results observed in facilitation of prey catching activity. The structure of the tectum shares many properties with other brain regions, which allow us to supplement the available tectal data. The model proposed is flexible, and offers several alternatives that could be tested experimentally to constrain the number of possible mechanisms proposed. The current model uses differential equations to represent the time course of the membrane voltage of the cell, and uses threshold functions to compute the corresponding action potentials. The model was simulated on a Digital Equipment Corporation VAX 11/780. Our study consists of the following sections: 1) the tectal column and its general behavior; 2) inhibition from the thalamus; 3) in Part II (Arbib and Lara, to appear) moving stimuli and multiple columns; and 4) in Part III (Lara and Arbib, to appear) interaction between tectum and pretectum in prey selection.

Our work is informed by the following features that brain regions share in their sensorimotor processing of information:

a) In many systems, afferents are initially processed by a synaptic complex, a glomerulus comprising specific connections between axonal and dendritic terminals which may be themselves

interconnected, enclosed in glial membranes, or otherwise set apart. This synaptic complex has been described in many brain regions, (Shepherd, 1970, 1974; Szentágothai, 1970; Székely and Lazar, 1976) such as the olfactory bulb, the thalamus, the cerebral cortex, the cerebellum, the spinal cord, and the amphibian optic tectum. There is a strong indication that a glomerulus functions to some extent as a functional unit.

b) Afferent information can be processed in both a lateral and a columnar organization, depending on the vertical or lateral dispersion of the information received. Vertical processing of information has been found (Shepherd, 1974; Szentágothai and Arbib, 1974; Mountcastle, 1957; Hubel and Wiesel, 1963) in the olfactory bulb, in many regions of the cortex (visual, auditory, and somatosensory), and in the optic tectum of amphibia. The elementary unit of vertical processing is held to be the column. From an anatomical-functional point of view it has been defined (Mountcastle, 1978; Szentágothai and Arbib, 1974) as an integrative unit of the neural tissue of minimal size that, on the basis of internal connectivity, could still be considered the unitary neuronal machine for elementary tasks of information processing, being composed of neurons activated by stimulation of the same single class of peripheral receptors and having almost identical peripheral fields. Lateral processing has been suggested as the dominant mode in some regions of the thalamus, hippocampus, olfactory cortex, and cerebellum. Some of these regions have been associated with processing of sequential information.

c) Sensorimotor processing involves a complex interaction of

excitation and inhibition. In many regions of the nervous system, circuits have been described which provide for an initial excitation folowed by a long lasting inhibition (Shepherd, 1974; Szentágothai and Arbib, 1974; Purpura, 1970). These include the spinal cord, cerebellum, olfactory bulb, olfactory cortex, hippocampus, the thalamus, and cerebral cortex. Some of these regions also exhibit rebound excitation due mainly to two mechanisms: one is through recurrent axons which may produce long lasting depolarization in their respective cells, as has been suggested to occur in the olfactory bulb (Shepherd, 1970), the thalamus (Scheibel and Scheibel, 1970; Purpura, 1970), and probably the amphibian optic tectum (Székely and Lazar, 1976); the other is the product of reverberatory circuits such as those involving the cerebellum and reticular formation (Eccles, 1973), the hippocampus and the septum (Raisman et al., 1965), and the thalamus and the cortex (Singer, 1978). All these processes are mediated by excitation, re-excitation, and inhibition.

We now incorporate these features in our analysis of tectal mechanisms in the facilitation of prey-catching behavior.

1.1 Behavioral and Physiological Studies Related to Facilitation of Prey Catching Behavior

David Ingle (1973, 1975) showed that a brief presentation of a wormlike stimulus of 5 degrees presented for an interval of 0.3 sec and moved 5 degrees of visual angle in a specific area of the retinal field of frogs does not induce prey catching behavior; but

if the same stimulus is presented again 2.3 sec later, the likelihood of snapping was increased to 75%. Moreover, this facilitation effect was restricted to the area previously stimulated, because re-presentation of the stimulus in a different region does not produce prey-catching behavior. These results indicate that the facilitatory effect involves little lateral spread of information, with the input processed in a predominantly vertical way. In the present paper, we shall analyse this vertical processing in terms of a posited anatomical column for optic tectum. A companion paper (Arbib and Lara, to appear) will consider the extension to an array of columns with local interaction.

Ingle (1975), trying to correlate the action of tectal cells with the observed behavioral results, found two cells whose temporal response could be related to the observed behavioral facts. He studied the activity of these cells presenting as stimulus a small square 2 degrees of size for an interval of 0.5 sec. One of these cells gives an initial response when the stimulus is shown, then a period of silence, and finally a slow steady discharge that lasts from 3 to 6 seconds. The second type of neuron does not respond when the stimulus is present, but gives a short delayed burst (see Fig 1). Moreover, Ingle has shown that these cells are located more easily after pretectum ablation, which suggests that these cells may play an important role in prey catching facilitation, and normally are controlled by the inhibitory effect of the thalamus pretectum. Anatomical studies of the tectum, described below, provides the basis for our model of

facilitation via reverberatory activity produced by recurrent axons. Other types of facilitation have been described in the literature, such as post-tetanic potentiation, and sensitization. Both types occur in a single synapse. Post-tetanic potentiation is produced as a result of repetitive activity that produces an increase in the amount of transmitter release in subsequent stimulations (Curtis and Eccles, 1960; Hubbard, 1963); while sensitization occurs as a consequence of the effect of another synapse on the mechanisms regulating the amount of transmitter release, increasing, in a long term way, subsequent transmitter liberation (Kandel, 1978). Both processes need the presence of the physiological stimulus to manifest its facilitation. On the other hand, a facilitatory effect has been described as a result of reduced inhibition over circuits with recurrent excitation (Tsukahara, 1979; Ayala et al., 1973), which is manifested as a paroxysmal neural activation, independently of any external stimulus. Given anatomical studies of the tectum which show that cells send recurrent axons to their dendrites, and given Ingle's data on the behavior of the tectal cells, which show a rebound excitation in the absence of stimulation, we have modelled the present facilitation phenomenon by recurrent excitation and inhibition, rather than in terms of synaptic facilitation.

1.2 Anatomy of the Tectum

Anatomical studies of the tectum (see Fig 2) indicate that this structure may be divided into nine alternating cellular and plexiform layers (Székely and Lazar, 1976). Layers 2, 4, 6 and 8 are cellular layers; layer 7 is a plexiform sheet constituting the main efferent pathway; and layer 9, which occupies one third of the tectum, contains a few cells and is where most of the retino-tectal synapses cluster into the tectal glomeruli in combination with recurrent tectal axons and diencephalic terminals.

Székely and Lazar (1976) have classified the tectal cells on the basis of their shape, dendritic and axonal arborization, and on the types of interactions with other cells. We will only describe those tectal cells that we have considered in our model; the other tectal cells described by these authors were not considered in this model either because they are hard to find in the tectum (as is the case for bipolar, amacrine and ganglionic cells), or because there is not enough anatomical information for their interactions with other tectal cells (as is the case for the second type of large pear cell). It must be stressed, however, that the formulation of the present model has involved a number of arbitrary choices that must be studied comparatively in the light of present and future experimental results. Moreover, the study of models based on alternative choices is an urgent topic for future theoretical research.

The tectal cells considered in our model are:

1) Large pear shaped cell (LP): these cells are located in layer 6; they have a long apical dendrite that projects toward the

surface and arborizes in a most variable manner, these arborizations being the main recipients of the optic terminals. The axon originates from the dendrites and takes an upward course close to the dendritic arbor of the parent cells.

2) Large pyramidal cell (PY): cells located in layer 6 with a wide dendritic tree; these cells are regarded as the efferent elements of the tectum in our model. (The present model does not take into consideration the less common tectal-ganglion efferents.)

3) Small pear shaped cells (SP): cells located in layer 8; these cells, like the LP cells, have a large apical dendrite that projects toward the surface but with a different arborization. Some of these neurons have very short axons terminating in a moderately arborizing formation next to the parent perykarion. In other cases, the axon descends into layer 6 and terminates among the cells of this layer. In the third type the axon descends for a while then with a sharp loop turns back and terminates in the lower part of layer 9. In the present model we have considered only the last type.

4) Stellate cells (SN): these cells are located in layer 9 and have many axons originating from the dendrites. Axo-somatic-somato-dendritic synapses have been found on these cells, with axons coming both from the LP cells and diencephalic terminals, and the dendrites of possibly LP and SP neurons.

The anatomical studies of the tectum indicate that this structure has mostly a vertical orientation: the cells having recurrent axons and small lateral spread of information. This has led to the suggestion that the tectum processes the optic

information in a vertical way by means of functional units comprising several cells. In the next section, we postulate on the basis of anatomical, behavioral, and physiological studies the synaptology of the functional unit of the tectum, the tectal column. Our model shows how tectal activity can last for several seconds after the stimulus has disappeared through a combination of excitatory and inhibitory interactions.

2. MODEL OF THE TECTAL COLUMN

2.1 General Structure.

We have considered the tectum as comprising functional units, the tectal columns, as described by Székely and Lazar (1976). The lateral extension of each column is determined by the arborization of the dendrites of the pyramidal cell, the main efferent cell in the tectum. The number of cells in each column was chosen as a proportion of the number of each cell type relative to the number of pyramidal cells. For this reason, each column has one pyramidal cell, three large pear shaped cells, two small pear shaped cells, and two stellate interneurons (of which only one is shown in the figure). Each of these cells has a receptive field and an effector field, the size of which is dependent on the degrees of dendritic and axonal arborization, respectively. The general configuration of the column is shown in Fig 3. The mathematical description of this system as a set of differential equations is provided in Appendix 1.

2.2 Synaptology and Function of the Tectal Column.

The optic afferents from retinal ganglion cells innervate the tectum mainly at the tectal glomerulus, type 1 and 2 in the superficial zone of layer 9 and type 3 and 4 in the deeper section of this layer. (In this model we have only considered type 1 and 2; we do not model retinal processes, simply assuring that relevant stimulation is provided to each tectal neuron.) The glomerulus, besides the optic fibers, is composed of the dendrites and recurrent axons of LP and SP cells, and fibers of diencephalic origin; optic afferents, internal axons of the tectum and diencephalic fibers are all presynaptic with respect to the dendrites. The LP and SP dendrites can have dendrodendritic synapses in a non-reciprocal fashion. For comparison, note that the activity of the olfactory bulb shows a long lasting oscillatory behavior after the stimulus has disappeared. This behavior has led to the postulate that the stimulus in combination with the recurrent activity of some cells in this structure produces a long lasting depolarization in the glomerulus, which in combination with the inhibitory activity of the mitral cells produce the oscillatory response (Shepherd, 1970, 1974). We have proposed, similarly, that the state of excitation of the glomerulus in the optic tectum can be maintained for a long period after the stimulus has disappeared. This state of activity is maintained by the excitatory activity of both the dendrodendritic interactions and recurrent axons of LP and SP cells. We suggest this hypothesis, as did Szekely and Lazar, based on the fact that the optic input goes directly to the glomerulus which also receives the dendrites of the intratectal

cells. An inhibitory effect at this level would seem to be an unlikely means of sending sensory information to the tectal column. In this way, the glomerulus may act as a functional unit in the sensorimotor processing of information.

In some regions of the nervous system, physiological study has shown (Shepherd, 1974; Purpura, 1970; Eccles, 1973; Kandel et al., 1961a and b) that after an EPSP produced by a given stimulus there usually is a subsequent long lasting hyperpolarization. This inhibition can be obtained through feedback inhibition in e.g., the spinal cord, thalamus, cerebellum, cortex and hippocampus, or can be due to dendrodendritic inhibition, as in the olfactory bulb, thalamus, retina and possibly in the cortex. In the present model we propose that the stellate cell inhibits the activity of the tectal column through feedback inhibition in the following way:

When the glomerulus has been stimulated it produces a long-lasting depolarization that travels by the apical dendrite to the soma of LP and SP cells; if the excitation is strong enough, it may produce a neural response. When the LP cell is activated, it excites a stellate neuron, which recurrently inhibits the LP excitation. Moreover, the inhibitory effect of the SN neuron can lead to lateral inhibition or local control of the state of excitation of the column. We have postulated that the inhibitory effect of these cells is dependent on their state of excitation. This postulate is based on the fact that the peculiar synaptic organization of these cells allows it to exert both local and global control of the column activity. This control is obtained locally through complex units of axodendritic or axosomatic

afferents and somato-dendritic or dendrodendritic synapses that can function below action potential threshold; general control will be exercised if the level of afferent activity is sufficient to drive the stellate cell to action potential, silencing a broad area of the tectal column (Székely and Lazar, 1976).

At the same time, the recurrent axons of the LP cells maintain both the level of activity of their column's glomeruli in a vertical manner and the state of excitation of neighboring glomeruli through axon collaterals, thus laterally spreading the excitation across the tectum. This architecture permits us to simulate some important temporal patterns, such as excitation followed by inhibition and rebound excitation. Initial physiological results in the amphibian optic tectum (Ingle, 1973, 1975, 1976a and b) support these assumptions.

The SP cell also receives its input from the glomerulus, and we propose that its function is the integration of the general state of activity of the column for the purpose of determining the proper time(s) for vertical recruitment of excitation in order to produce a response in the efferent cell of the column. In this way, the SP samples the excitatory interaction betweeen the glomerulus and LP cell under the inhibitory effect of the stellate neuron.

We propose that the principal efferent of the column is the pyramidal cell (PY), which receives afferents from the LP and SP cells and acts as an output integrator for the activity of the column as a whole. The output of this cell could go to the spinal cord or reticular formation of the animal to yield motor output, or

could send axons to the thalamus which would establish tectal-thalamic loops.

It has been suggested (Ewert, 1970,1976) that the thalamus and pretectum exerts an inhibitory effect over the tectal activity. Fibers possibly diencephalic in origin have been found to form synapses in three areas of the tectum (Székely and Lazar, 1976): 1) around and within the glomerulus, where they may act through presynaptic inhibition; 2) in the intervening zone between glomeruli, where they probably exert postsynaptic inhibitory effects over the SP, LP, and PY dendrites; and 3) in some of the stellate cells, where they are likely to produce postsynaptic excitation. We model the effect of different paths for such inhibition.

3. COMPUTER SIMULATION.

To understand the general behavior of the column and the different hypotheses that can be postulated to underly the observed results, we used simulation to study the following aspects of the model:

1. Activity of the PY cell when the stimulus is presented for different intervals of time and the optic fibers only excite the glomerulus.
2. Facilitation of PY response when two stimuli are serially presented and the optic fibers only project to the glomerulus. In this section we also study the behavior of the tectal cells in order to reproduce the observed

physiological results.

3. Facilitation of PY response when two stimuli are serially presented and the optic fibers project to the glomerulus, LP, SP, and PY cells. In this section we also study the PY response for different durations of stimulus presentation.
4. Control of PY facilitation by the diencephalic fibers: a) inhibitory effects on the glomerulus; b) inhibitory effects on LP, SP, and PY cells; and c) excitatory and inhibitory effects on the SN.
5. Change of architecture to propose an alternative hypothesis of neural interaction within the column.
6. Minimum model of the tectal column to study the sensitivity of the different parameters.

The results obtained with these models will provide us with specific hypotheses that could be tested experimentally and that could help to narrow the number of alternative models to explain the behavior.

The results of the simulation are shown in two ways: through the simulated behavior of each of the cells considered in the model and through graphs showing the sensitivity of response to variation of different parameters of stimulation. In the first case, we have simulated 5 seconds of real time to reproduce the behavioral and physiological results. We show diagramatically the response of cells after the membrane potential has reached the threshold through the generation of spikes. We do not model the time course

of spike generation. The spikes in the figures are a graphical convention designed to aid understanding of the behavior of the model; the way the model behaves when the membrane potential reaches threshold is explained in the Appendix.

In 3.1 and 3.2 the tectal column acts as a controller of the output through its general state of excitation, with the optic input only arriving at the glomeruli so that the activity of the PY is controlled by the LP and SP neurons.

3.1 PY Response to Duration of Presentation of the Stimulus if the Optic Fibres only Arrive at the Glomeruli.

Fig 4A shows the neural response of tectal cells when a brief (0.5 sec) stimulus is presented. Notice that the LP cells give a short response when the stimulus is presented and then a delayed burst of activity, reproducing the observed physiological results of tectal cells described by Ingle (see Fig 1 A). The SP neuron gives a short delayed response after the stimulus has disappeared, reproducing the physiological behavior of tectal cells (Fig 1C). If we increase the period of presentation of the stimulus then the PY cell, the efferent neuron of the model column, gives a response (see Fig 4B). We consider PY response to code the location and speed of prey orienting behavior (Ewert, 1976), as we discuss in more detail in the accompanying paper (Lara and Arbib, to appear). The behavior of PY in Figs. 4A, B reproduces the observed behavior of amphibia when the period of presentation of a prey-stimulus is increased.

3.2 PY Facilitation When Two Stimuli are Serially Presented and the Optic Fibers only Arrive at the Glomeruli.

Fig 4C shows the temporal pattern of response of the tectal cells during facilitation of prey catching activity. When the stimulus is presented, the glomerulus produces a long EPSP. This potential makes the LP respond which in turn excites both the SN, which produces a long lasting hyperpolarization on LP and SP cells, and the glomerulus, which maintains its state of excitation. When the LP is released from the inhibitory effect of the SN, it produces a new response because the glomerulus is still active, repeating this cycle several times until it finally decays. The SP cell, in the meantime, is integrating the state of activity of the glomerulus, the LP and SN cells, until its membrane voltage reaches its threshold value which generates a response. The activity of the SP excites the LP cell, the glomerulus, and the PY, increasing in this way the general state of excitability of the column. This figure shows how the model reproduces the behavior of the tectal cells described by Ingle (1975) (see Fig 1), which are related to prey catching facilitation. The LP cell reproduces the behavior of the neuron which responds to the onset of the stimulus, then exhibits a period of silence and finally a rebound activity that lasts for several seconds (Fig 1A and B); while the behavior of the SP cell simulates the activity of the neuron which does not respond when the stimulus is present but gives a short delayed response (Fig 1C). Finally, the behavior of the PY cell, considered as responsible for prey catching activity, reproduces the behavioral results, because a response is only given to the

second presentation of the short stimulus.

In 3.3, we examine the postulate that the tectal column acts as a modulator of the PY response. In this model, the PY cell receives optic afferents directly, so that its response is dependent on the conjoint activity of LP, SP cells and the stimulus.

3.3 Facilitation of PY Activity when Stimuli are Serially Presented and the Optic Fibers Arrive at the Glomerulus, the LP, SP, and PY Cells.

Székely and Lazar (1976) have observed that the optic afferents to the tectum arrive mainly at the glomeruli, but there are also many synapses in the intervening zone between and somewhat lower than glomeruli arriving at light dendrites, probably of LP, SP, and PY cells. For this reason, we again study the behavior of the tectal column, but now so modified that optic input arrives at the LP, SP, and PY cells as well as the glomerulus. In this case, we postulate that the tectal column acts rather as a modulator than as a controller of the PY behavior. According to this, the PY cell only responds if the stimulus is present and if the column is in a state of hyperexcitation through the behavior of the LP and SP cells. The response of the tectal column to stimuli presented for different periods and the facilitation to the presentation of the second of two stimluli are shown in Fig 5. Fig 5 A shows the behavior of the tectal column when a single stimulus is presented. The LP cell gives a short response when the stimulus is present, a

period of silence, and then rebounding excitation; the SP gives a short delayed response; and the PY does not respond. If we increase the period of presentation of the stimulus (Fig 5 B), then the PY fires. Fig 5C shows the facilitation in the PY response when a stimulus, that initially does not make it fire, is presented for the second time. The physiological behavior of the LP, SP, and PY cells is similar to the above experiment, reproducing the physiological and behavioral experiments.

Fig 6 shows the PY response when stimuli are presented for different intervals. This figure shows that the PY activity increases with the period of stimulation.

Fig 7 shows the temporal pattern followed by the facilitation effect when a stimulus is applied. It can be seen that the facilitation reaches its maximum after a short period (2.5 sec), then decays slowly with sporadic rebounding facilitation.

In the structure of 3.1 and 3.2, the PY response is present long after the stimulus has disappeared; while in the structure of 3.3, the response is only present when the stimulus is present or has just disappeared. In the accompanying papers (Arbib and Lara; Lara and Arbib, to appear) we postulate that the activity of PY cells in combination with pretectal neurons plays a role in the location and intensity of the orienting response to a given stimulus. For this reason we consider that the second model reproduces better the observed results, because it gives more precise information about the actual site of the stimulus.

It is also interesting to note that both models present phases of excitation and inhibition during the facilitatory period, which

indicates that the speed of the response will also be modified depending on which of these phases the column happens to be in during the presentation of the second stimulus.

In the next subsections, we will test the behavior of the second model, which we think is more closely related to behavioral results (although the determination must be given by experiments).

3.4 Facilitation of PY Activity and Diencephalic Inhibition.

Anatomical studies of the tectum (Székely and Lazar, 1976; Trachtenberg and Ingle, 1974; Scalia, 1976) have shown that afferent fibers distinct from the optic terminals arrive at the tectum. It is also well known that diencephalic terminals project to the tectum, suggesting that the fibers observed in Golgi studies come from these regions. These fibers have been found in the following sites: 1) the periphery of and within the glomerulus; 2) in the zone intervening between and somewhat below the glomeruli to the dendrites of LP, SP, and PY cells; and 3) the soma of the stellate neuron. We have simulated the possible inhibitory effects of diencephalic terminals on the facilitation of prey catching activity. In this way, we can propose three possible ways in which diencephalic terminals could affect the facilitatory behavior of the column: 1) controlling the expression of facilitation; 2) erasure of the facilitation; and 3) suppression of facilitation.

We activate the diencephalic fibers after the presentation of the first stimulus and then study the effects of the inhibitory action on the PY response when the second stimulus appears. Fig 8

shows the effect on PY response when the optic afferents are presynaptically inhibited by diencephalic terminals; the response of the PY cell decreases with increasing inhibition; notice, however, that in this situation the state of excitation of the column is not directly affected. Fig 9 shows the PY response when the inhibitory action is over the dendrites of the glomerulus, suppressing the excitatory dendrodendritic activity. The general results are similar to the above case but with the difference that in this condition the facilitatory state of the column is erased, because the activity of the glomerulus, which is responsible for oscillatory behavior, has been suppressed.

Fig 10 shows PY activity when the inhibitory action is exerted on the dendrites of LP, SP, and PY cells. As in the above cases, the facilitation decreases with increasing inhibition; the main difference in this experiment with respect to the other ones is that the inhibitory effects over the LP and SP cells prevent the continuation of the facilitatory effects, regulating in this way the development of the change. Notice that the rebounding excitation of LP cells and the SP activity are delayed and that the PY neuron does not respond as a consequence of the thalamic inhibition. Finally, Fig 11 A shows what would happen if the diencephalic terminals were excitatory, and served to control the inhibitory effect of the SN cell over LP and SP cells. In this case the inhibitory action of these fibers is stronger than in the above cases, preventing the facilitation effect with small values of excitation. In this condition, similar to the previous one, the diencephalon controls the development of the change. Fig 11 B

shows the effect on the tectal column when the diencephalic terminals inhibit the SN. Because the thalamic inhibition supresses the inhibitory effect of SN, all tectal cells respond in a paroxysmal manner. Figure 12 summarizes these different schemes for thalamic inhibition.

3.5 Change of Architecture.

One of the main goals in simulating the visuomotor system of amphibia is to present alternative models that reproduce the observed behavioral and physiological results and to propose specific experimental questions that will assist us in determining which of the models is closest to "reality". For this reason, the following change of architecture in the tectal column is tested to see if it can also reproduce the physiological and behavioral results observed by Ewert and Ingle. This change of architecture was motivated by anatomical evidence provided by Szekely and Lazar (1976) which indicates that the interaction between LP and SP cells could form a loop independent of that involving the glomerulus. Moreover, these authors have noted that SP cells do not have the same type of dendritic trees as LP cells, suggesting that they may have partially independent sources of excitation. For these reasons, we briefly studied a modified tectal column where the glomerulus receives the optic afferents and recurrent axons and contains the LP cell dendrites; the LP and SP cells form an independent loop, wherein the SP cell is excited by the LP cell and the SP cell provides feedback in an excitatory loop to the LP cell;

and where both cells are inhibited by the SN. The behavior of this model is shown in Fig 13, where it can be seen that SP, as well as LP, behavior is identical to the behavior of LP neurons in the above models; any possibility of reverberatory activity between these cells is precluded by the inhibitory action of SN. In this case the integrative activity proposed for the SP cell is lost and the physiological and behavioral results obtained by Ingle are not reproduced.

3.6 Minimal Model of the Tectal Column.

In order to understand the basic features that we have proposed for the behavior of the tectal column in prey catching facilitation, it is important to define the minimum structure that reproduces the desired behavior. This minimum model will allow us to study the sensitivity of its response to values of the different parameters involved in the model.

The structure of the minimum model is shown in Fig 14, and comprises the glomerulus, one LP, one SP, one SN, and one PY cell. The parameters used in the model are given in Appendix 2, where all the factors have been described. As the table shows, only a few of the parameters have been changed because of the reduced number of cells.

3.7 Sensitivity of the Behavior of the Tectal Column to the Parameters.

Fig 15 shows the sensitivity of the minimal column's behavior to changes in the parameters. The graphs are shown in relation to two extremes of the behavior of the tectal column: unstable behavior and non-oscillatory activity, therefore no facilitation. The graphs show that almost all the parameters can change the behavior of the tectal column between these extremes. The sensitivity analysis shows that the crucial factor in the stability of the system is the predominance of the inhibitory effect over the excitatory activity. An increase in each one of the terms related to the first ($k_1, w_{lp \cdot sn}, w_{sp \cdot sn}, w_{sn \cdot lp}$) makes the system more stable; while an increase in the factors regulating the state of excitability ($k_2, w_{gl \cdot lp}, w_{gl \cdot sp}$) tends to instability.

4. DISCUSSION.

The present model reproduces the facilitation of prey catching behavior when a brief stimulus, that initially does not produce a response, is presented for a second time. The model is based on anatomical studies of the optic tectum, physiological studies obtained in the cells of this region, and on the behavioral results observed in prey-catching facilitation. Other assumptions considered in the model were taken from the results obtained in other brain regions that have similar structures and that present similar physiological behavior. For this reason, this model can be

used to study the possible mechanisms responsible for oscillatory activity found in different regions, where periods of excitation, inhibition, and rebounding excitation have been indicated.

The correlation found by Ewert and Ingle between tectal cells and behavior is suggestive but by no means determinant. It is important that a formal causal relationship be found and that the behavior of these neurons be correlated with other cells in the tectum within and outside the postulated column. For example, Ingle's recordings were obtained in the superficial layers of the tectum, where SP cells are found, but the tectal efferents are located in deeper layers; thus indicating that a close relationship may exist between these two group of neurons. The present model proposes the following hypothesis that could be tested experimentally:

1. The input produces a long lasting depolarization in the glomerulus.
2. The initial response is produced by cells located in the sixth layer and are silenced by the inhibitory effect of neurons located in the ninth layer. These cells, which Ingle found rarely in the eighth layer, should appear more frequently if the electrode goes deeper.
3. The combined effect of the long-lasting depolarization and the inhibition produces the observed physiological behavior of excitation, silence, and rebounding excitation.
4. The delayed response is produced by the integration of the glomerulus, LP, and SN neurons in the SP cell located in

the 8th layer.

5. The efferent cell of the column is only activated if the state of excitation of the column has been increased, as measured by the activity of tectal neurons.
6. Diencephalic terminals may control the state of excitation of the tectum in several ways, depending on the site of stimulation. The model indicates that excitation of the SN has a stronger effect on the general state of excitation of the tectum than inhibition at LP, SP, and PY cells or the glomerulus. When thalamic fibres arrive at the tectal dendrites, the manifestation of the facilitation effects is suppressed but the long-lasting depolarization of the glomerulus is still present; while the effect of diencephalic fibres over the glomerulus disrupts completely the facilitatory effect. These results suggests that the interaction between thalamus and tectum can play different roles and may have different temporal consequences. Each of these postulates could be tested following the same paradigm we used in our simulation: presenting a brief stimulus, then exciting the thalamus, and observing the consequences when the second stimulus is presented. If the relationship of tectal cells has hitherto been studied, then the different effects of different thalamic regions could be studied.

The present model only considers the effect of ganglion cells type 1 and 2, but the tectum also receives excitation from ganglion cells type 3 and 4. The latter neurons have been associated with

avoidance behavior, indicating that the tectum may play a role in this activity through columnar structures similar to the ones proposed in this paper, and with close interactions with the thalamus. The study of the possible role of the tectum in avoidance behavior and its relationship with the orienting response still deserves more experimental and theoretical work.

APPENDIX 1: MATHEMATICAL DESCRIPTION OF THE COLUMN

We represent the behavior of tectal neurons by a system of simultaneous differential equations which permit us to model the local (somatic membrane) potential, the threshold function, and the action potential of the cell. Due to the lack of adequate physiological information regarding the values of membrane constants for the different cells, we will use as approximate values the membrane constants of cells for which this parameter has been determined (Eccles, 1973).

The mathematical model of the tectal column is defined in terms of the different cells described anatomically for this structure. As we have mentioned, each column comprises 3 glomeruli, 3 large pear shaped cells, 2 small pear shaped cells, 2 stellate neurons (only 1 of which is shown in Figure 3), and 1 pyramidal cell. Each glomerulus contain optic fibers, dendrites and recurrent axons of LP and SP cells, and diencephalic terminals. We have treated the firing rate of SN as proportional to its membrane potential (above a threshold) because it seems (Székely and Lazar, 1976) that SN can exert increasingly widespread effects as its state of excitation increases. The inputs to the tectal column are the optic and diencephalic afferents, while the response of the column is given by the large pyramidal cell. In the present paper we do not model retinal response to the optic input, but simply provide each neuron with an input which encodes the

physiologically appropriate stimulus. The tectal column is shown in Fig 3.

We associate with each element a membrane potential (denoted in lower case) and, with the exception of the glomerulus, a firing rate (denoted in upper case).

The fundamental equation describing the dynamics of each membrane potential m(t) will be of the form

$$\tau \dot{m}(t) = -m(t)+I(t)$$

where τ is the membrane constant and I(t) represents the weighted sum of excitatory and inhibitory inputs.

The firing rate will be related to the membrane potential by a transfer function F(m−θ) where θ is a suitable threshold value, and F may be of either the form

$$f(x)= \begin{cases} 1 & \text{if } x>0 \\ 0 & \text{if not} \end{cases}$$

or

$$h(x)= \begin{cases} x & \text{if } x>0 \\ 0 & \text{if not} \end{cases}$$

Our specific choices are shown in Table 1.

Element	Membrane Potential	Firing Rate
Glomerulus	gl	--
Large Pear-Shaped Cell	lp	LP = f(lp - 1.0)
Small Pear-Shaped Cell	sp	SP = f(sp - 2.0)
Stellate Neuron	sn	SN = h(sn - 0.2)
Thalamic Input	th	TH = h(th)
Pyramidal Cell	py	PY = h(py - 0.8)

Table 1. Threshold functions for cell firing.

A1.1 Glomerulus:

The glomerulus is considered as a functional unit which receives as inputs the optic fibers and the recurrent axons of LP and SP cells. We have simulated the dendrodendritic activity of this structure simply by the decay constant of the state of activity of the glomerulus.

We use $gl_i(t)$ (i=1,2,3) to represent the membrane potential of the ith glomerulus of the tectal column at time t. The basic equation for the dynamics of each $gl_i(t)$ is

$$\tau_{gl}\ gl_i(t) = -k_1\ gl_i(t) + s \cdot u + I_i(t)$$

where: $\tau_{gl} = k_1 = 0.5$, so that (τ_{gl}/k_1) equals 1.0 and is the time constant of the glomerulus potential, and is chosen to simulate the excitatory dendrodenritic synapses that maintain a long EPSP (Shepherd, 1970) which is responsible for the rebound excitation after inhibition has been inactivated.

$w_{gl \cdot lp} = 1.0$	LP to GL.
$w_{gl \cdot sp} = 0.1$	SP to GL.
$w_{lp \cdot sp} = 0.8$	SP to LP
$w_{lp \cdot sn} = 8.0$	SN to LP
$w_{lp \cdot th} = *$	TH to LP
$w_{sp \cdot sn} = 15.0$	SN to SP
$w_{sp \cdot th} = *$	TH to SP
$w_{sn \cdot lp} = 1.0$	LP to SN
$w_{sn \cdot th} = *$	TH to SN
$w_{py \cdot lp} = 1.0$	LP to PY
$w_{py \cdot sp} = 1.0$	SP to PY

Table 2. Weights.

The weights marked with an * are given zero or non-zero values in different experiments.

u is the optic input, while s is a habituation factor. We do not consider habituation in the present study, and thus take s to be 1.

$I_i(t)$ is the recurrent input from the LP and SP cells. As shown in Figure 3, it takes the form

$$I_1(t) = w_{gl \cdot sp} SP_1(t) + w_{gl \cdot lp}(LP_1(t) + LP_2(t))$$
$$I_2(t) = w_{gl \cdot sp}(SP_1(t) + SP_2(t)) + w_{gl \cdot lp}(LP_1(t) + LP_2(t) + LP_3(t))$$
$$I_3(t) = w_{gl \cdot sp} SP_2(t) + w_{gl \cdot lp}(LP_2(t) + LP_3(t))$$

where the different values of w, the weighting factors, are given in Table 2. These values indicate that the recurrent axons of the LP cells, $w_{gl \cdot lp}$, have a stronger effect over the glomerulus than those of the SP cells, $w_{gl \cdot sp}$. This choice is made because when the

latter are active, the general level of activity of the column is very strong and the recurrent effect could result in unstable behavior (see the sensitivity analysis of these factors above).

A1.2 Stellate Interneurons:

We model each stellate cell as inhibiting the tectal column through feedback inhibition when it is stimulated by the LP cell. The SN can also be activated by the diencephalic terminals; for this reason, the dynamics of this cell are expressed as follows (See Fig. 3):

$$\tau_{sn} \frac{dsn_1(t)}{dt} = -k_2 sn_1(t) + w_{sn\cdot lp}(LP_1(t) + LP_2(t)) + w_{sn\cdot th}TH(t)$$

$$\tau_{sn} \frac{dsn_2(t)}{dt} = -k_2 sn_2(t) + w_{sn\cdot lp}(LP_2(t) + LP_3(t)) + w_{sn\cdot th}TH(t)$$

where the membrane constant $\tau_{sn} = 0.65$, $k_2 = 0.5$ and $(\tau_{sn}/k_2) = 1.3$, which is chosen to simulate the long lasting hyperpolarization described for this type of cell in other brain regions. The weighting factors are defined in Table 2. The effect $w_{sn\cdot lp}$ of the LP over the SN cell was chosen to simulate the rapid inhibition that follows the excitation of the LP cells; $w_{sn\cdot th}$ defines the effect of the thalamic input over the SN (to be explained in detail later when we describe the general effect of diencephalic fibers over the tectum).

A1.3 Large Pear-shaped Cells:

LP cells receive excitatory input from the glomerulus and the SP cells, and are inhibited by the SN and TH input. The equations defining the dynamics of this cell are expressed as follows:

$$\tau_{lp} \frac{dlp_i(t)}{dt} = -lp_i(t) - w_{lp\cdot th} TH(t) + J_i(t) + gl_j(t)$$

where the intracolumnar inputs $J_i(t)$, $(i=1,2,3)$, are given by:

$$J_1(t) = w_{lp\cdot sp} SP_1(t) - w_{lp\cdot sn} SN_1(t)$$
$$J_2(t) = w_{lp\cdot sp}(SP_1(t) + SP_2(t)) - w_{lp\cdot sn}(SN_1(t) + SN_2(t))$$
$$J_3(t) = w_{lp\cdot sn} SP_2(t) - w_{lp\cdot sn} SN_2(t).$$

where $\tau_{lp} = 0.3$ is the membrane constant defined so as to achieve the desired time-course of response in the LP cells to the afferent stimulus from the glomerulus and the optic input; The $gl_i(t)$ represent the transmission of excitation from the glomeruli to the LP cells; w are the weighting factors defined in Table 2, where $w_{lp\cdot sp}$ has been chosen to simulate the recruitment of cells produced by the SP neuron; $w_{lp\cdot sp}$ was chosen to simulate the strong long-lasting hyperpolarization produced by the SN over the LP cell to control the general state of excitation of the column; and $w_{lp\cdot th}$ is the weight factor of the thalamic input over the LP neuron.

A1.4 Small Pear-shaped Cells:

As indicated above, the SP cells integrate the state of activity of the glomeruli and of the SN cells. The SP cells also receive an inhibitory input from the thalamus and from the SN neurons. The equations that define their behavior are expressed as follows:

$$\tau_{sp} \frac{dsp_1(t)}{dt} = -sp_1(t)+(gl_1(t)+gl_1(t))-w_{sp\cdot sn}SN_1(t)-w_{sp\cdot th}TH(t)$$

$$\tau_{sp} \frac{dsp_2(t)}{dt} = -sp_2(t)+(gl_2(t)+gl_3(t))-w_{sp\cdot sn}SN_2(t)-w_{sp\cdot th}TH(t)$$

where $\tau_{sp} = 0.9$ is the membrane constant of SP. This membrane constant is slow in comparison to the other cells to simulate the long lasting integration proposed for this neuron. The weighting factors, w, are shown in Table 2, where it can be seen that the inhibitory effect of the SN is very strong, controlling in this way the general state of excitation of the column. The threshold function SN = h(sn-0.2) was chosen in order to ensure the stability of the behavior of the column through an inhibition proportional to the state of activity of the stellate cell, which is also in accordance with the role proposed for the SN by Székely and Lazar (1976). The SP cells receive optic afferents through the glomeruli; we have proposed that they receive information from two different glomeruli, to ensure the general recruitment of all tectal cells.

A1.5 Pyramidal Cell:

Finally, the cell that integrates the state of activity of the column is the pyramidal cell, which receives inputs from the LP and SP cells. The dynamic behavior of this cell can be expressed by the following equation:

$$\tau_{py} \frac{dpy(t)}{dt} = -py(t) + w_{py \cdot sp}(SP_1(t)+SP_2(t)) + w_{py \cdot lp}(LP_1(t)+LP_2(t)+LP_3(t))$$

where $\tau_{py} = 0.4$ is the membrane constant of the PY, which value was chosen to simulate a fast response when the tectal column is highly excited; the threshold functions of LP, SP, and PY cells are shown in Table 1 above; and the w are the weighting factors, shown in Table 2. In this way the PY responds when both the LP and SP cells are simultaneously active.

The Nth stellate neuron behavior can be defined as follows:

$$\tau_{sn}\, \dot{sn}_N(t) = -k_2\, sn_N(t) + w_{sn\cdot lp}(LP_N(t) + LP_{N+1}(t))$$

receiving inputs from two LP cells, one coming from its own unit column and the other from the right neighbor.

The Nth LP neuron is defined as:

$$\tau_{lp}\, \dot{lp}_N(t) = -lp_N(t) + w_{lp\cdot sp}(SP_{N-1}(t) + SP_N(t)) + gl_N(t) - w_{lp\cdot sn}(SN_N(t) + SN_{N-1}(t)) + u_N(t)$$

where the LP cell is excited by two SP neurons, one coming from its own unit column and the other from the left neighbor. This cell is also activated by the optic input, and is inhibited by two SN, one is part of its column unit and the other is the left neighbor.

The Nth SP neuron is defined as follows:

$$\tau_{sp}\, sp_N(t) = -sp_N(t) + gl_N(t) + gl_{N+1}(t) - w_{sp\cdot sn}\, SN_N(t) + u_N(t)$$

this neuron receives inputs from the glomerulus of the unit column and the glomerulus from its right neighbor; it is also excited by the optic input; and it receives inhibition from the SN of the unit column.

The Nth PY cell is defined in the following way:

$$\tau_{py}\, py_N(t) = -py_N(t) + w_{py\cdot sp}\, SP_N(t) + w_{py\cdot lp}(LP_N(t) + LP_{N+1}(t)) + u_N(t)$$

receiving input from the retina, from the SP of the unit column, and from two LP cells, one from the unit column and one from the right neighbor.

The parameters for this column are the same as those for the column of Appendix 1, save that (cf. Table 1) SP = f(sp−1.0) and PY = h(PY−0.4).

In the simulations of the unit column presented in this paper, we hold all membrane potentials for neighboring columns equal to zero.

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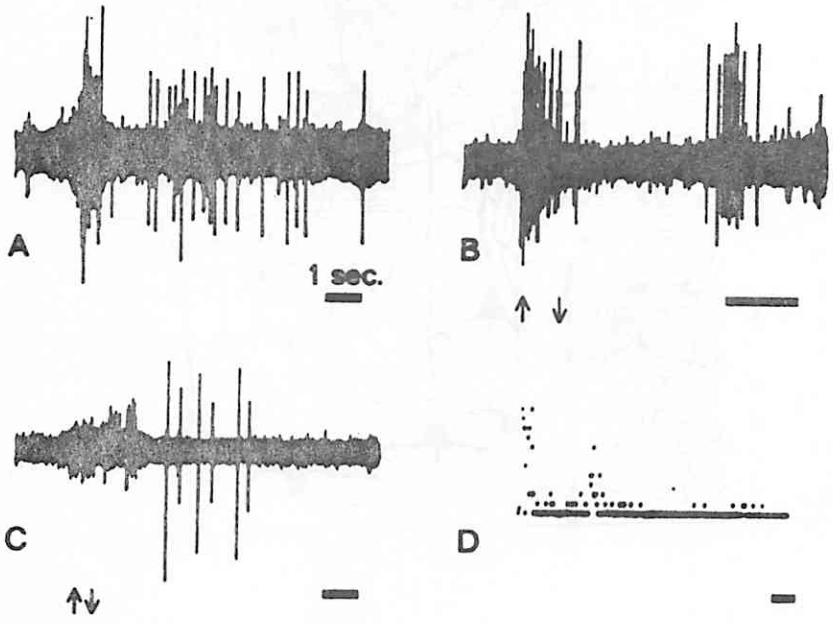
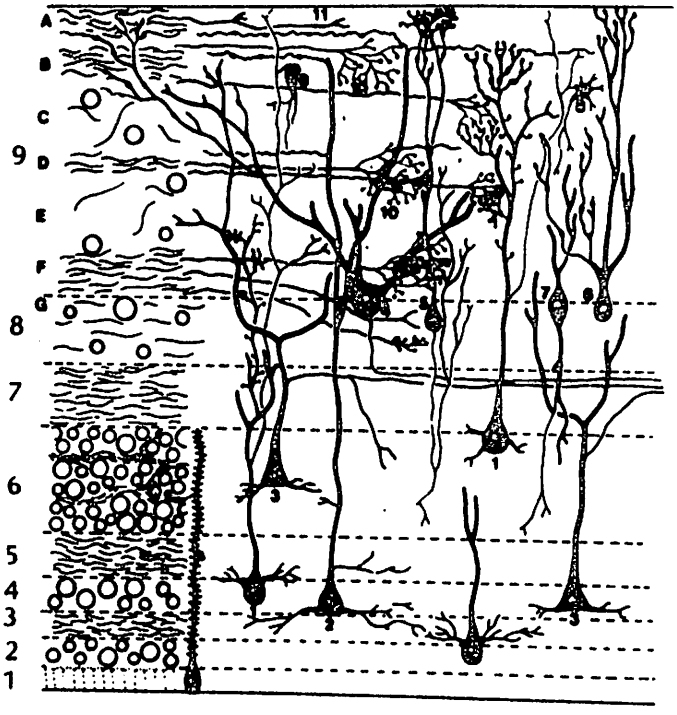
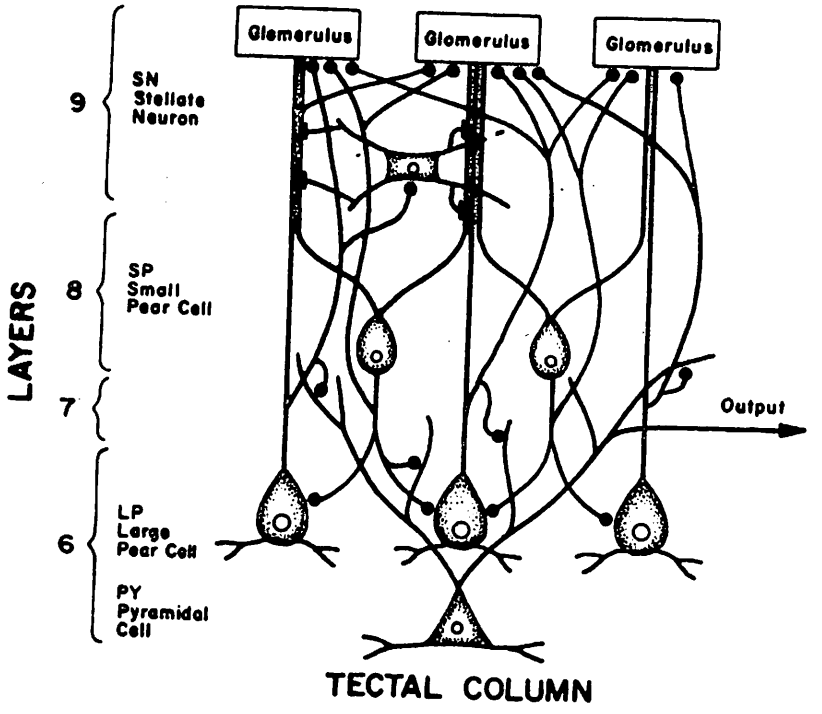
FIGURES



1. Physiological behavior of cells related to prey catching facilitation. A shows a brief class 2 burst followed by a delayed response of a tectal cell. In B it is shown the behavior of a tectal cell responding to the presentation of the stimulus and again with a delay. C shows a tectal neuron that produces a delayed response to the presentation of the stimulus. Finally D shows the postimulus histogram of a tectal cell showing a delayed peak at 3 to 4 seconds (from Ingle, 1975).



2. Diagramatic representation of the lamination and the representative types of neurons of the optic tectum. Numbers on the left indicate the different tectal layers. Numbered cell-types are as follows: (1) large pear-shaped neuron with dendritic appendages and ascending axon; (2) large pear-shaped neuron with dendritic collaterals; (3) large pyramidal neuron with efferent axon; (4) large tectal ganglionic neuron with efferent axon; (5-6) small pear-shaped neurons with descending and ascending axons respectively; (7) bipolar neuron; (8) stellate neuron; (9) amacrine cell; (10) optic terminals; (11) assumed evidence of diencephalic fibres (from Szekely and Lazar, 1976).



3. Neurons and synaptology of the model of the tectal column. The numbers at the left indicate the different tectal layers. The glomerulus is constituted by the LP and SP dendrites and recurrent axons as well as by optic and diencephalic terminals. The LP excites the PC, the SN, and the GL, and is inhibited by the SN (of which only one of the two in our model of the column is shown). The SP excites the LP, and PC cells and it sends recurrent axons to the glomerulus; it is inhibited by the SN. The SN is excited by LP neurons and diencephalic fibres and it inhibits the LP and SP cells. The PY is activated by the LP, SP, and optic fibres, and is the efferent neuron of the tectum.

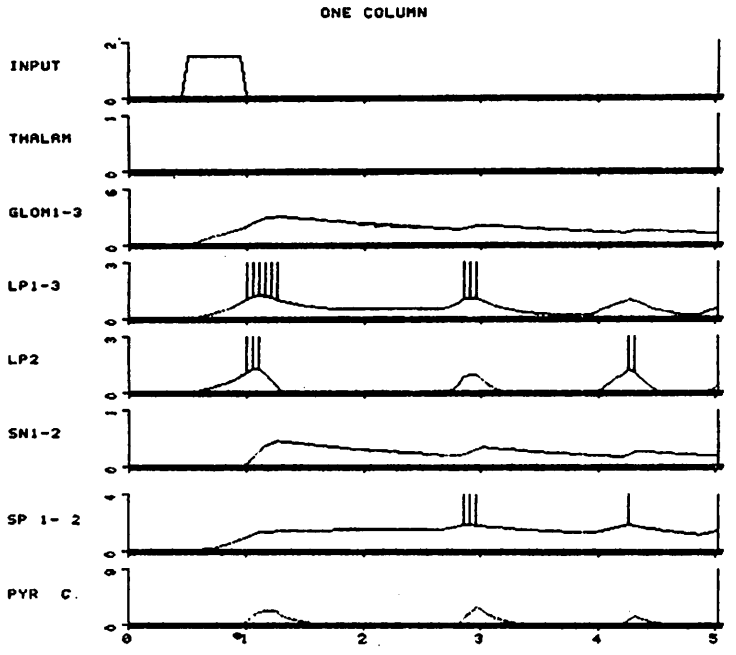


Fig. 4A

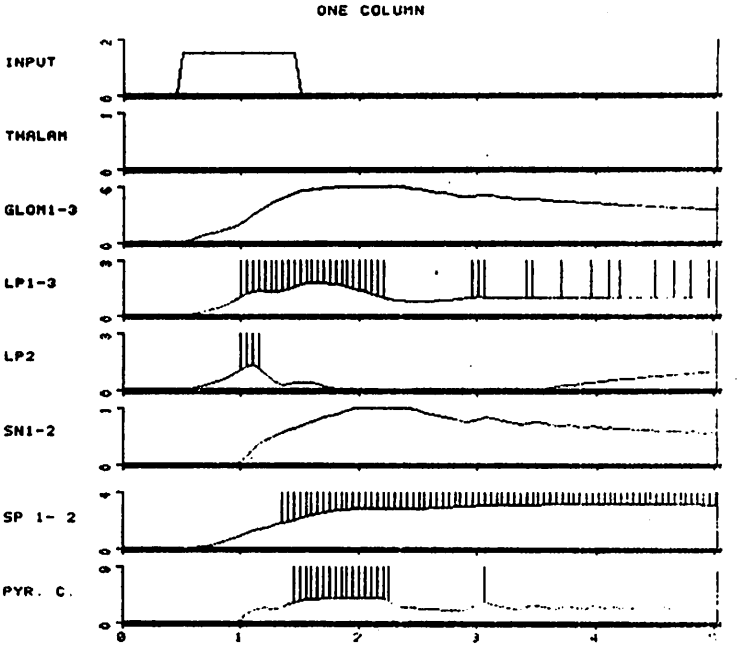


Fig. 4B

4. (A) Computer simulation of tectal cells response when a brief stimulus is presented. The onset of the stimulus produces a long lasting depolarization in the glomerulus which then fires the large-pear shaped cell (LP). This neuron in turn sends recurrent axons to the glomerulus and the stellate cell (SN) which acts as the inhibitory neuron in the column. When the inhibitory effect of SN releases the LP cell, a rebounding excitation occur. The small pear-shaped cell is integrating the activity of GL, LP, and SN neurons to give a delayed short response. (B) If in the above situation we present a stimulus of longer duration then we show that now the pyramidal neuron fires. In (C) we show that when a second stimulus of the 'subthreshold duration' used in (A) is presented, the pyramidal cell (PY) reponds. (The frequency of the spikes are a graphical convention. The spikes are drawn simply to highlight when the membrane potential of a cell is above threshold.)

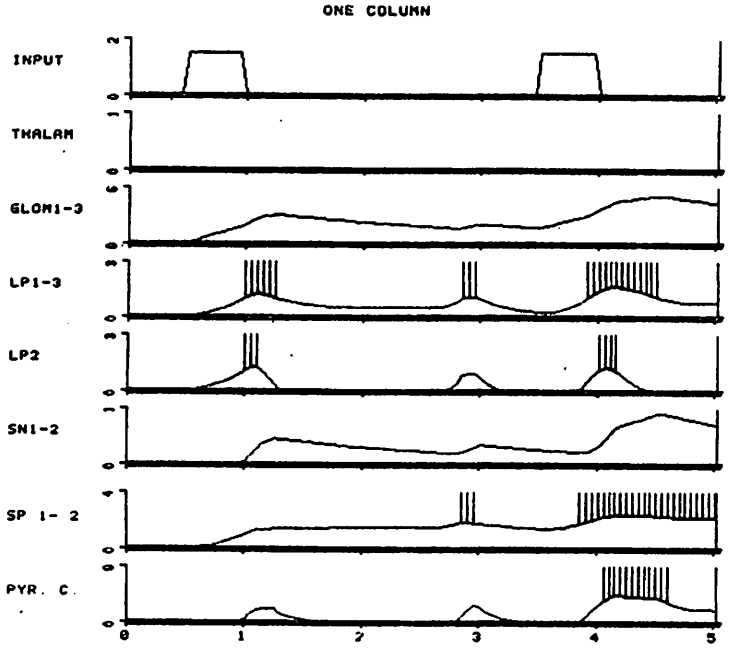


Fig. 4C

(See caption, previous page)

Fig. 5A

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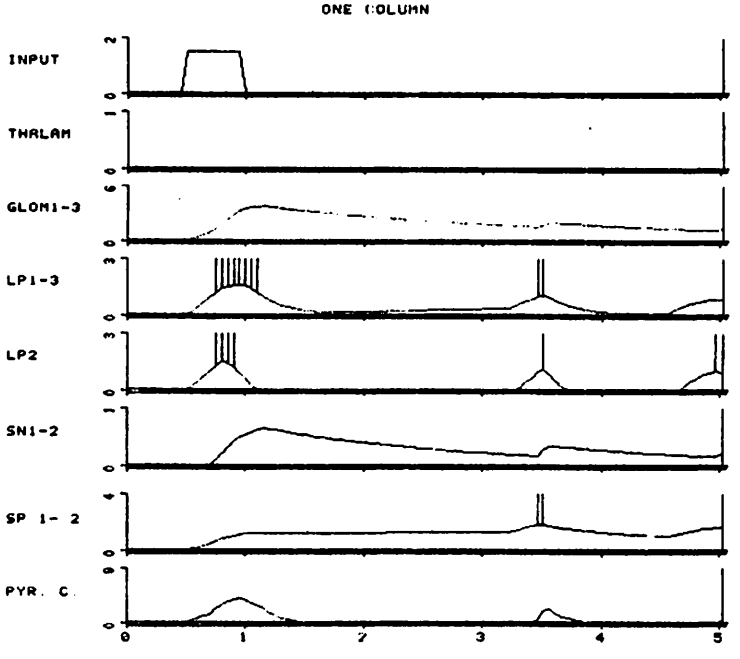


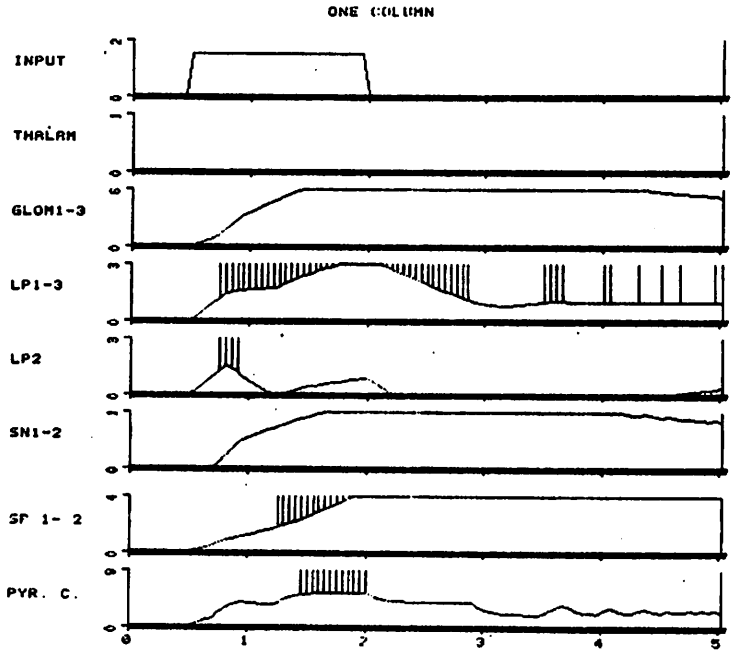
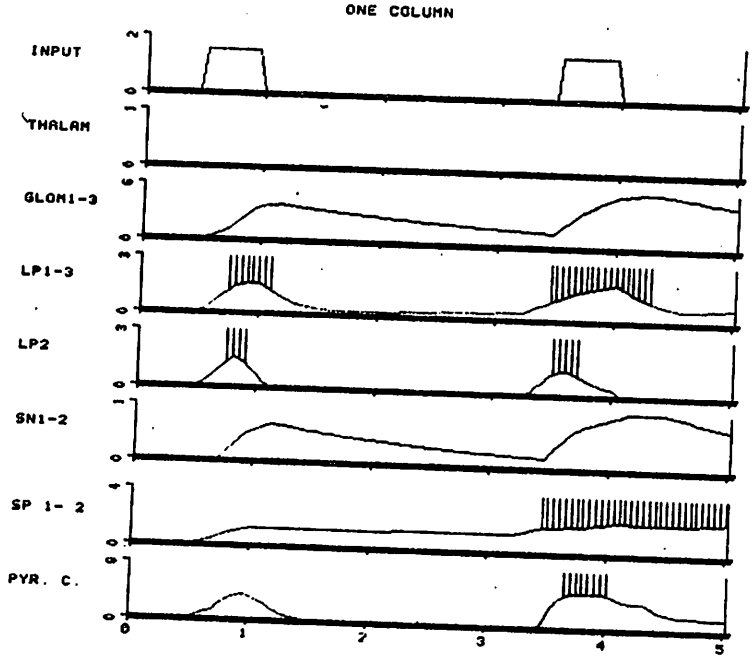
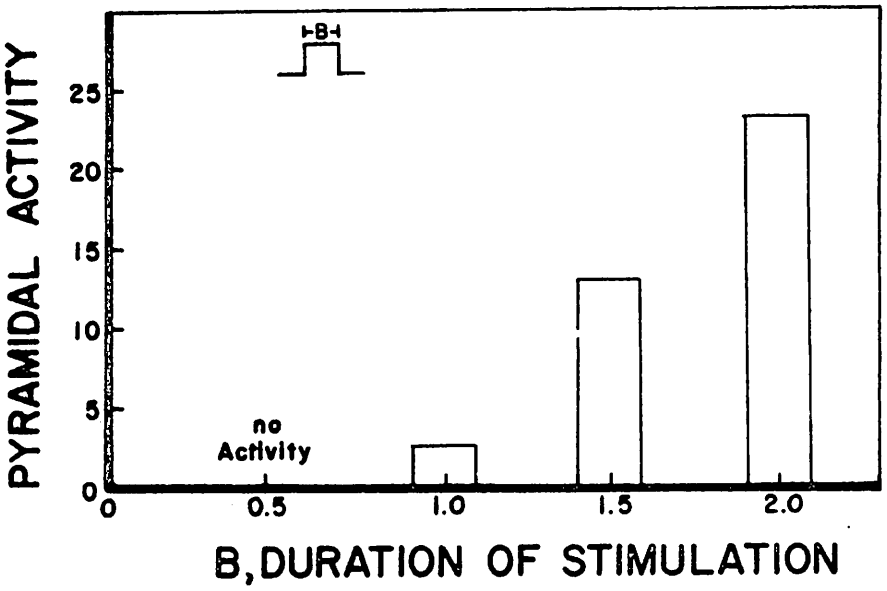


Fig. 5B

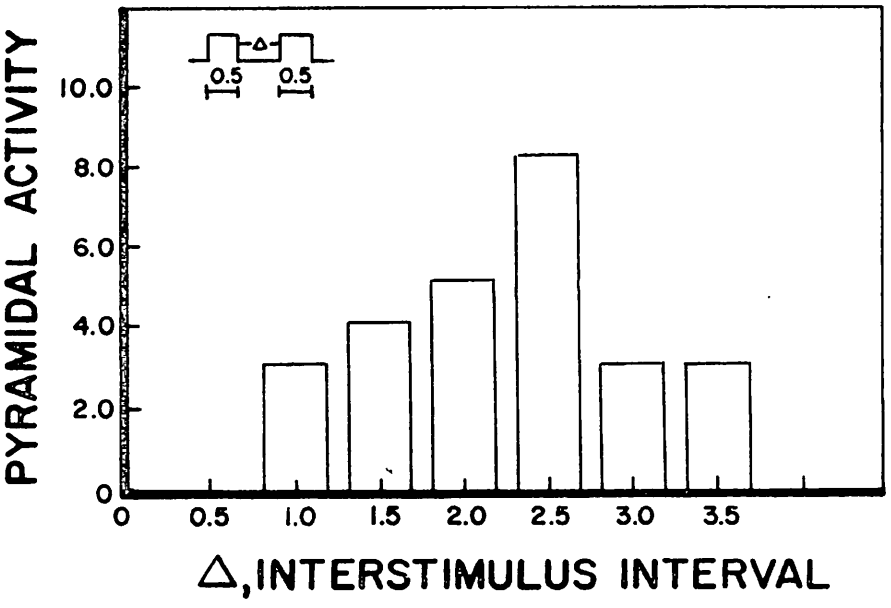
Fig. 5C



5. Computer simulation of the behavior of tectal neurons during facilitation to the repeated presentation of a stimulus when the optic input arrives at the GL, LP, SP, and PY neurons. A shows the behavior of tectal cells to the presentation of a single stimulus; notice the response of the LP cell when the stimulus is presented and then a delayed reactivation in combination with the delayed response of the SP neuron; the PY cell is only depolarized but does not produce a response. (B) When the stimulus is presented for a longer period the PY neuron is now activated. (C) Facilitation of the PY activity when a second stimulus is presented; notice that the PY responds only when the stimulus is present.

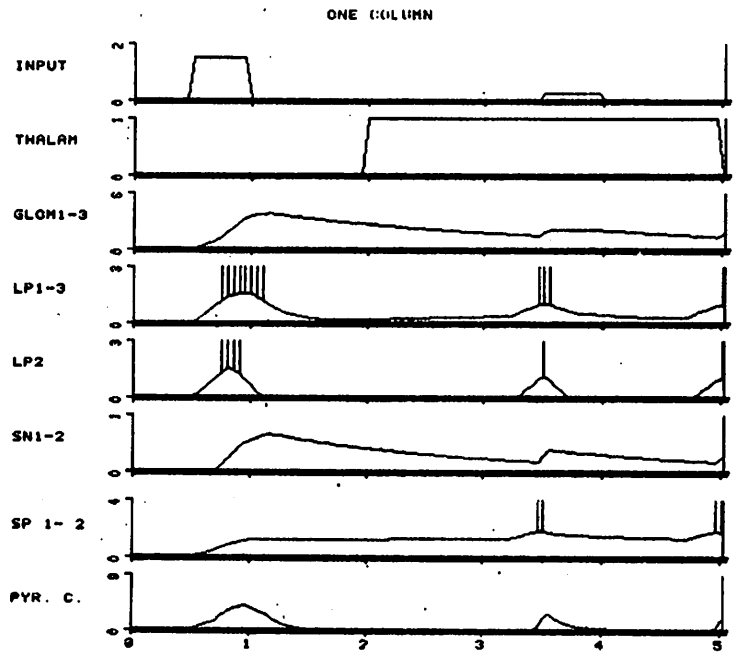


6. Computer simulation of the PY behavior when stimuli are presented for different intervals. The graph shows that the longer the presentation of the stimulus the larger the PY response.

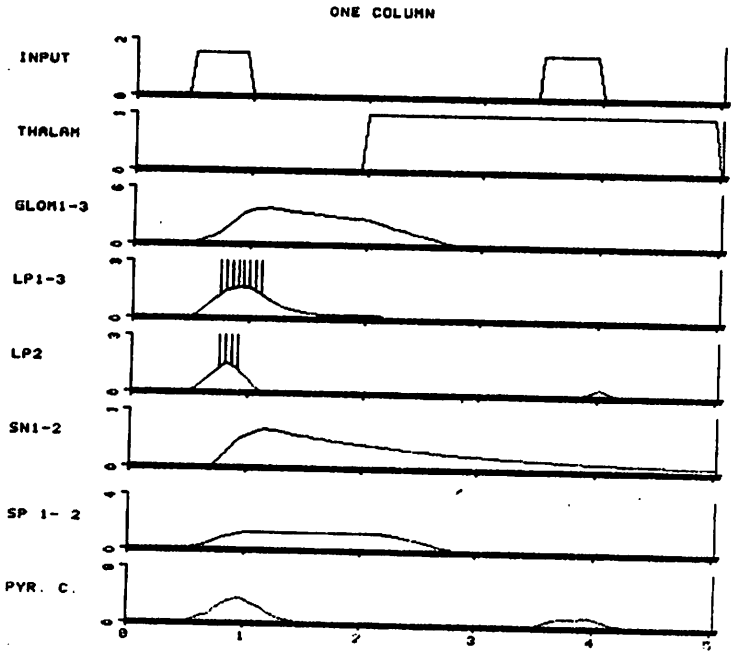


7. Computer simulation of the temporal pattern of the facilitation process after the presentation of a brief stimulus. Each bar shows the duration of PY activity if the two stimuli of 0.5 sec duration are presented with the inter-stimulus interval given below each bar.

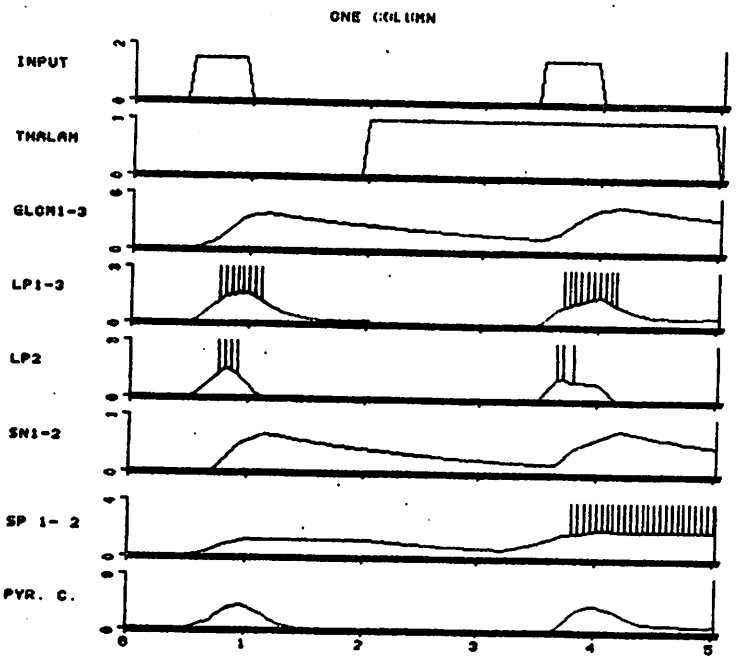
The graph shows that the maximum facilitation is presented 2.5 sec after the presentation of the first stimulus.



8. Computer simulation of the effects of diencephalic inhibition over the optic fibres, possibly through presynaptic inhibition, on the behavior of tectal cells during facilitation. The graphs show that facilitation is prevented, but the general state of excitation of the column is not modified.



9. Computer simulation of the effects of diencephalic inhibition over the glomerulus. The graph shows that thalamic inhibition completely supresses the glomerulus depolarization, therefore, erasing the state of facilitation of the column.



10. Computer simulation of the effects of diencephalic inhibition over the LP, SP, and PY cells. Notice that the rebounding excitation of the LP neurons and the initial activity of the SP cells is delayed (see Fig 7C for comparison), preventing the PY response, as a consequence of the thalamic inhibition. The state of excitation of the column, however, is still present, showing that in this case the thalamus acts as a controller of the development of the facilitatory process.

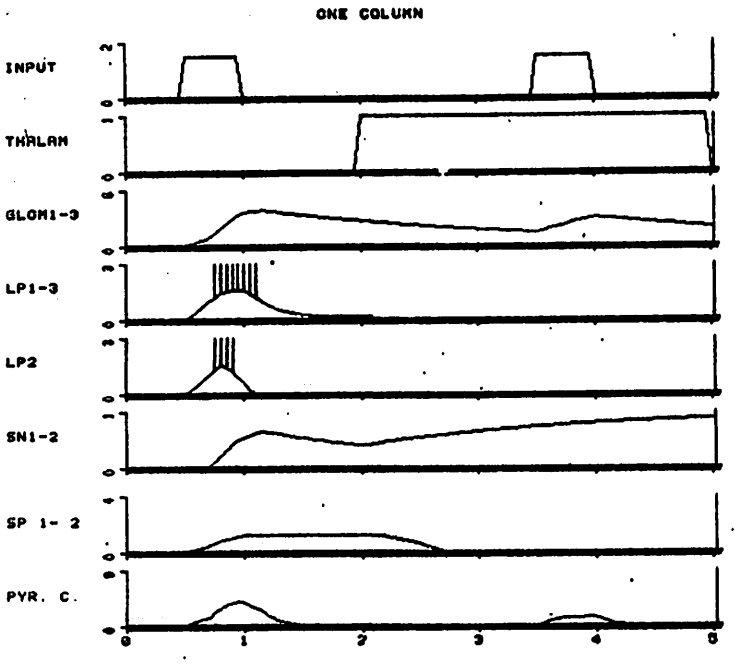
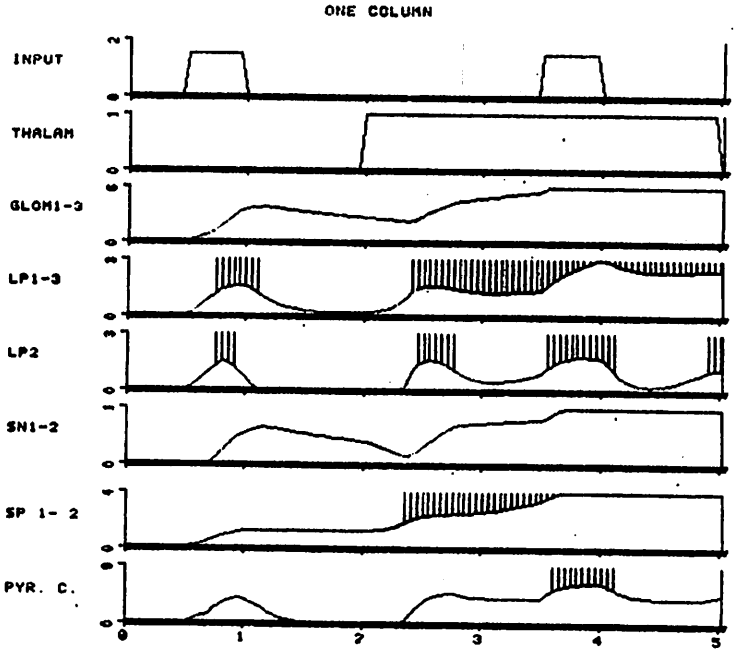
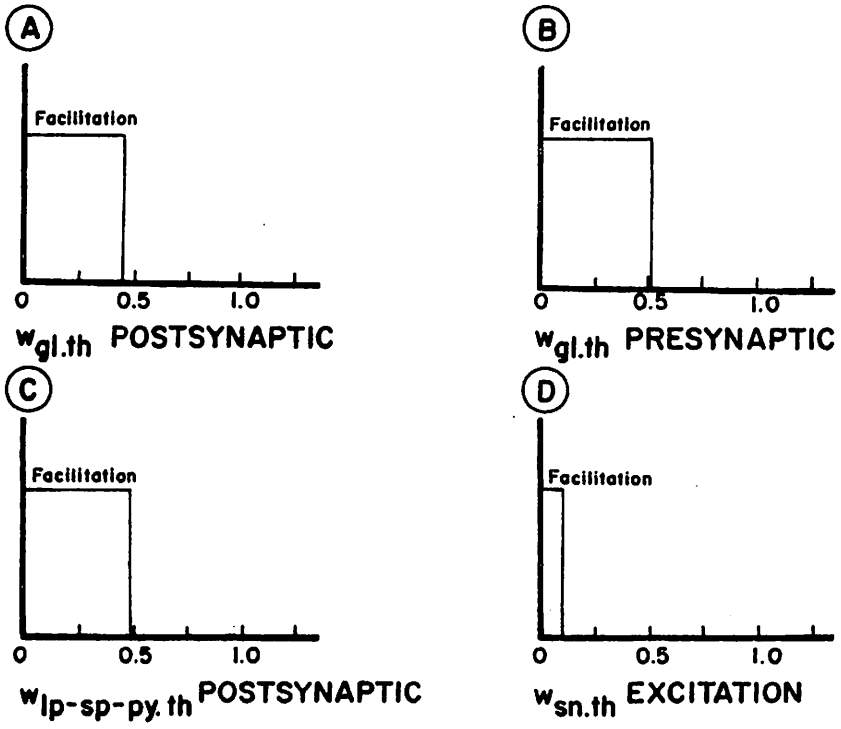


Fig. 11A

Fig. 11B



11. Computer simulation of diencephalic action over SN during facilitation to the presentation of a second stimulus. (A) When the thalamus excites the SN, the activity of the LP and SP neurons is totally suppressed, therefore no facilitation occurs. The state of excitation of the glomerulus is still high, showing that in this case the diencephalon prevents the manifestation of the change. (B) When the diencephalon inhibits the SN, there results an inhibition of an inhibitor so that a paroxysmal activity of all tectal cells is present.



12. Sensitivity analysis of the effect of diencephalic inhibition over tectal facilitation in different zones. In A), B), and C), diencephalic fibers are posited to inhibit excitatory elements of the column. In D), the fibers are posited to excite stellate neurons, the inhibitory elements of the column. (A) Postsynaptic inhibition to the glomerulus. (B) Presynaptic inhibition to the optic input. (C) Postsynaptic inhibition to the LP, SP, and PY cells. (D) Postsynaptic excitation to the SN. It can be seen that the strongest effect is produced through the SN neuron.

13. Computer simulation of the new architecture of the tectum in which the SP neuron is now only activated by LP neurons, forming a loop with positive feedback, but without receiving any optic input from the glomerulus. Notice that during double presentation of a stimulus, the LP and SP cells respond simultaneously, so that the dissociated behavior of these neurons is not present.

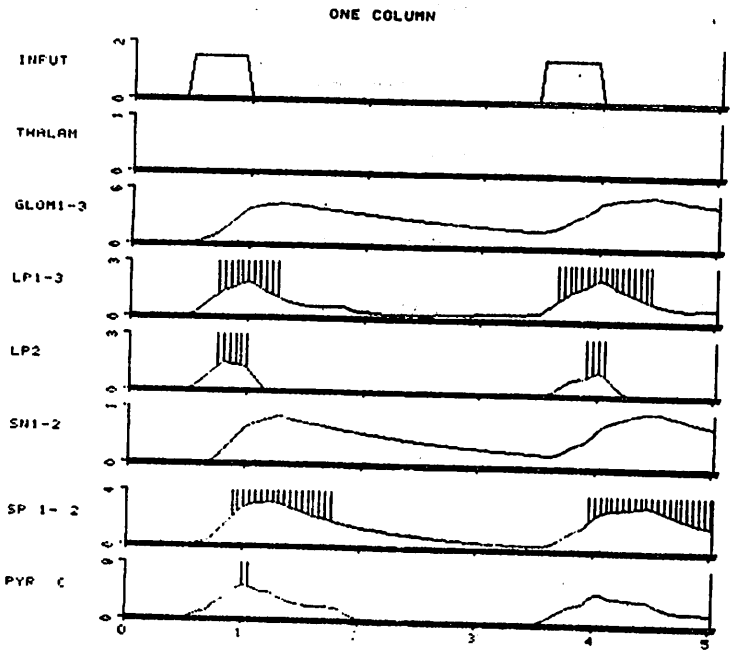


Fig. 13

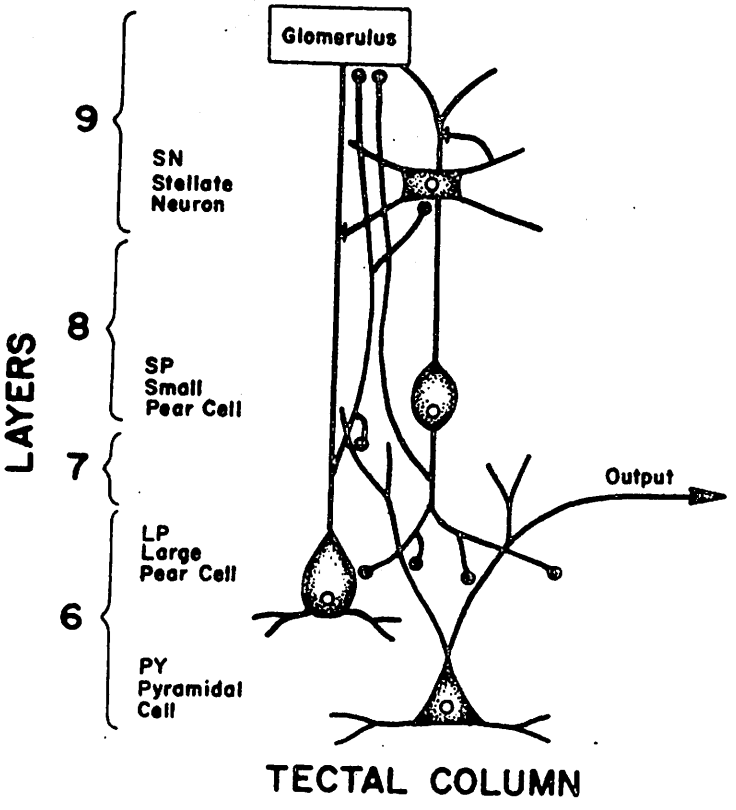
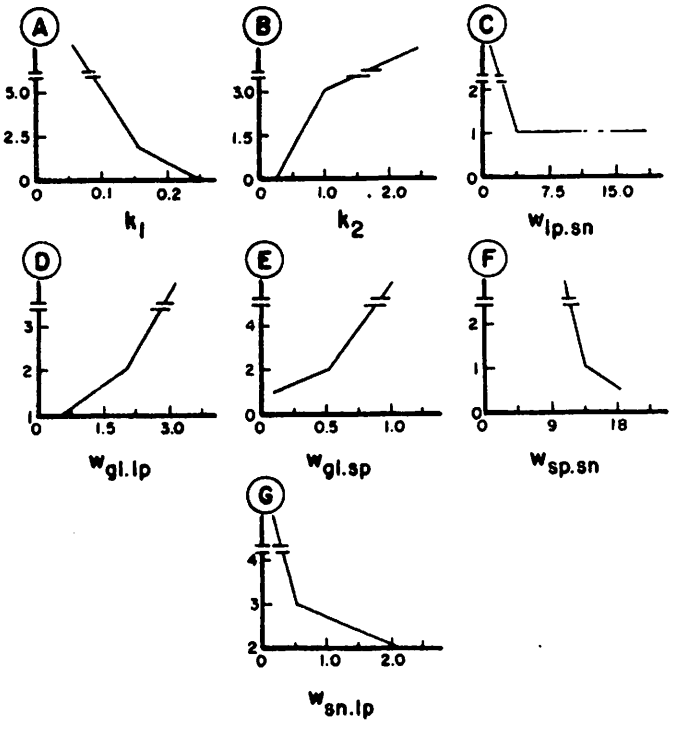


Fig. 14

14. Diagram of the cells and interactions of the simplified tectal column comprising a glomerulus, one LP, one SP, one SN, and one PY cell. The interactions are otherwise the same as those described in Fig 3.



15. Sensitivity analysis of tectal behavior to the different parameters of the model. (A) k_1 regulates the decay constant of the glomerulus' potential; for low values (0.1), the GL depolarization lasts longer and the system is unstable. (B) k_2 regulates the decay constant of the inhibitory effect of SN. For big values of k_2, the inhibitory effect lasts a short period, making the system unstable. An increase in the inhibitory effect, through $w_{lp\cdot sn}$ (C), $w_{sp\cdot sn}$ (F), $w_{sn\cdot sp}$ (G), makes the system more stable; while an increase in the excitation, by means of $w_{gl\cdot lp}$ (D), $w_{gl\cdot sp}$ (E), leads the system to instability.