MODELLING NEURAL MECHANISMS OF VISUOMOTOR COORDINATION IN FROG AND TOAD

Michael A. Arbib

COINS Technical Report 82-1

(February 1982)

To appear in:

S. Amari and M.A. Arbib (Eds.) Competition and Cooperation in Neural Nets, Lecture Notes in Biomathematics, Springer-Verlag (1982).

MODELLING NEURAL MECHANISMS OF VISUOMOTOR COORDINATION IN FROG AND TOAD*

Michael A. Arbib
Computer and Information Science Department,
Center for Systems Neuroscience
University of Massachusetts
Amherst, Massachusetts 01003, USA

ABSTRACT

Frogs and toads provide interesting parallels to the way in which humans can see the world about them, and use what they see in determining their actions. What they lack in subtlety of visually-guided behavior, they make up for in the amenability of their behavior and the underlying neural circuitry to experimental analysis. We provide an overview of problems involved in modelling neural mechanisms of frog and toad visuomotor coordination; and then present a number of background models "in search of the style of the brain." We then review three specific models of neural circuitry underlying visually-guided behavior in frog and toad. They form an 'evolutionary sequence' in that each model incorporates its predecessor as a subsystem in such a way as to explain a wider range of behavioral data in a manner consistent with current neurophysiology and anatomy. The models thus form stages in the evolution of Rana computatrix, an increasingly sophisticated model of neural circuitry underlying the behavior of the frog.+ Finally, we provide a quick tour of a number of studies which have developed from these basic models.

1. NEURAL SUBSTRATES FOR VISUALLY-GUIDED BEHAVIOR

Lettvin, Maturana, McCulloch and Pitts [1959] initiated the behaviorally-oriented study of the frog visual system with their classification of retinal ganglion cells into four classes each projecting to a retinotopic map at a different depth in the optic tectum, the four maps in register. In this spirit, we view the analysis of interactions between layers of neurons as a major approach to

---

* The research reported in this paper was supported in part by the National Institutes of Health under grant RO1 NS14971-03. My special thanks to Rolando Lara of Universidad Nacional Autonoma de Mexico with whom the recent modelling was conducted during his stay at the University of Massachusetts, 1978-1980. Portions of Sections 1 and 4 appeared in the earlier paper, Arbib (1982).

+ When both models and experiments are further advanced, the time will be ripe for the differential analysis of (different species of) frog and toad. In the present article, however, we conflate data gathered from both frog and toad studies to lay the experimental basis for the models that we discuss.

modelling "the style of the brain". In Section 3, we offer a general view of cooperative computation between neurons within a layer, and between layers within the brain. (The relation of "maps as control surfaces" to the general study of perceptual structures and distributed motor control is given in Arbib [1981].) In following sections, we shall then exemplify these general principles in three specific models of cooperative computation in neural circuitry underlying visuomotor coordination in frog and toad. The final section will then chart directions for further modelling.

Lettvin et al. found that group 2 retinal cells responded best to the movement of a small object within the receptive field; while group 3 cells responded best to the passage of a large object across the receptive field. It became common to speak of these cells as "bug detectors" (following Barlow [1953]) and "enemy detectors", respectively, though subsequent studies make it clear that the likelihood of a given frog behavior will depend on far more than activity of a single class of retinal ganglion cells (Ewert [1976], and Section 4 below). Given the mapping of retinal "feature detectors" to the tectum and the fact that tectal stimulation could elicit a snapping response, it became commonplace to view the tectum as, inter alia, directing the snapping of the animal at small moving objects -- it being known that the frog would ignore stationary objects, and would jump away from large moving objects. However, this notion of a simple stimulus-response chain via the tectum was vitiated by Ewert's observation that after a lesion to PT (pretectum-thalamus) a toad would snap at moving objects of all sizes, even those large enough to elicit escape responses in the normal animal. More detailed neurophysiological studies support the inference that the tectum alone will elicit a response to all (sufficiently) moving objects, and that it is PT-inhibition that blocks this response when the object is large, since tectal cells respond to visual presentation of large moving objects in the PT-lesioned animal [Ingle, 1973].

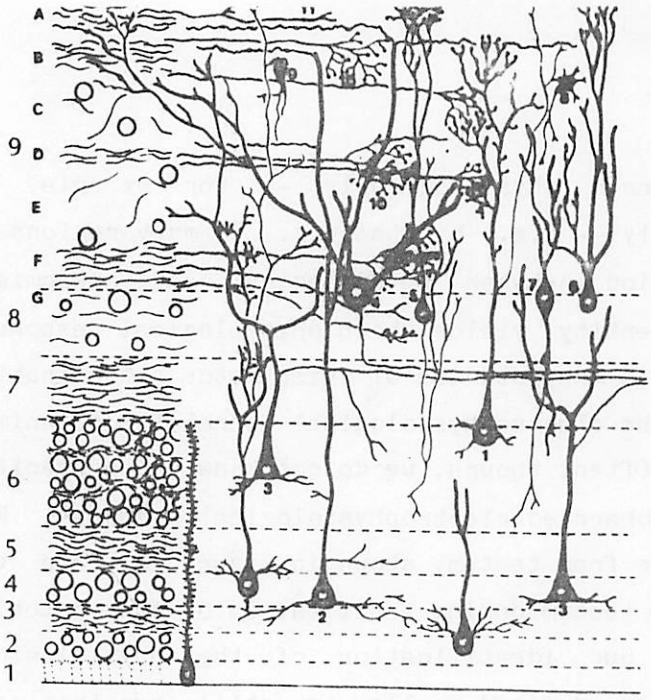
In Section 4a we present a model of local circuitry in the tectum (a 'tectal column') to explain certain facilitation effects in prey-catching behavior; then in Section 4b we study a linear array of such columns to model certain data on size-dependence of prey-catching activity in toads; and then, in Section 4c, we add PT-inhibition to such an array to model the behavior of an animal confronted with more than one prey-stimulus. These models form three stages in an evolutionary sequence for Rana Computatrix, our developing model of the neural circuitry underlying visuomotor coordination in frog and toad. Tectum and PT are but two of the many brain regions to be incorporated into the model during its further evolution. Section 5 provides a brief perspective of models discussed at greater length in later papers.

2. AN OVERVIEW OF MODELLING PROBLEMS

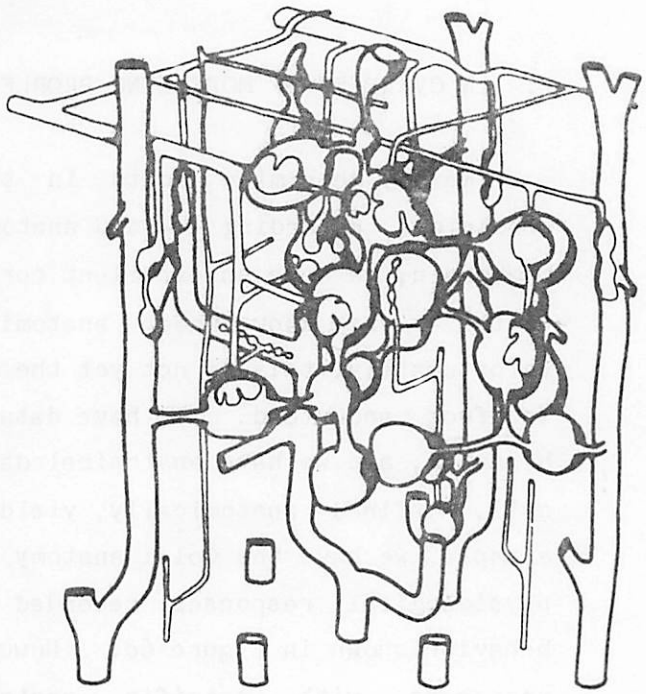
We may determine units in the brain physiologically -- for example, by electrical recording -- and anatomically -- e.g. by staining. In many regions of the brain, we have an excellent correlation between physiological and anatomical units -- we know which anatomical entity yields which physiological response. Unfortunately, this is not yet the case in many studies of visuomotor coordination in frog and toad. We have data on the electrophysiological correlates of animal behavior, and we have anatomical data. Often, though, we do not know which specific cell, defined anatomically, yields an observed electrophysiological response. For example, we have the Golgi anatomy of the frog tectum, shown in Figure 1a, and the physiological responses recorded from tectum during facilitation of prey-catching behavior shown in Figure 6d. However, our identification of the physiological responses with specific anatomically defined cells is still hypothetical. Nonetheless, such choices have to be made in formulating and testing our models.

Another problem that we confront in modelling is that we have both too much and too little anatomical detail: too much in that there are many connections that we cannot put into our model without overloading our capabilities for either mathematical analysis or computer simulation; and too little in that we often do not know which details of synaptology may determine the most important modes of behavior of a particular region of the brain. For example, in starting from the Golgi anatomy of frog tectum shown in Figure 1a, we can either follow Szekely and Lazar into the elaborate synaptology shown in Figure 1b, or we can rather accept their schematic view of a tectal column as the basic unit of structure, as shown in Figure 1c. In the modelling to be described in this paper, we have chosen the latter course, viewing the tectum as an array of interconnected columns each of which has the formal structure shown in Figure 1d, the behavior of the various neurons being described by coupled differential equations. In comparing the Golgi anatomy of Figure 1a with the model of Figure 1d, we see that a number of choices have been made. In Figure 1a we see that there are two types of output cells for the tectum, the pyramidal cell and the large tectal ganglionic neuron. Our model assumes that it is only the output of the former that is relevant to the phenomena that we are considering. Clearly, our models must be of such a kind that they are adaptable when we come to phenomena that in fact can be shown to depend upon the ganglionic output. Note, too, that we have ignored the bipolar neurons and amacrine cells, and that we have made certain assumptions about the connectivity between the neurons that are included in the model. However, an important point of our modelling methodology will be that we set up our simulation in such a way that we can use different connectivity on different simulations. In this fashion, we can generate hypotheses which can then be subjected to further experimental test.

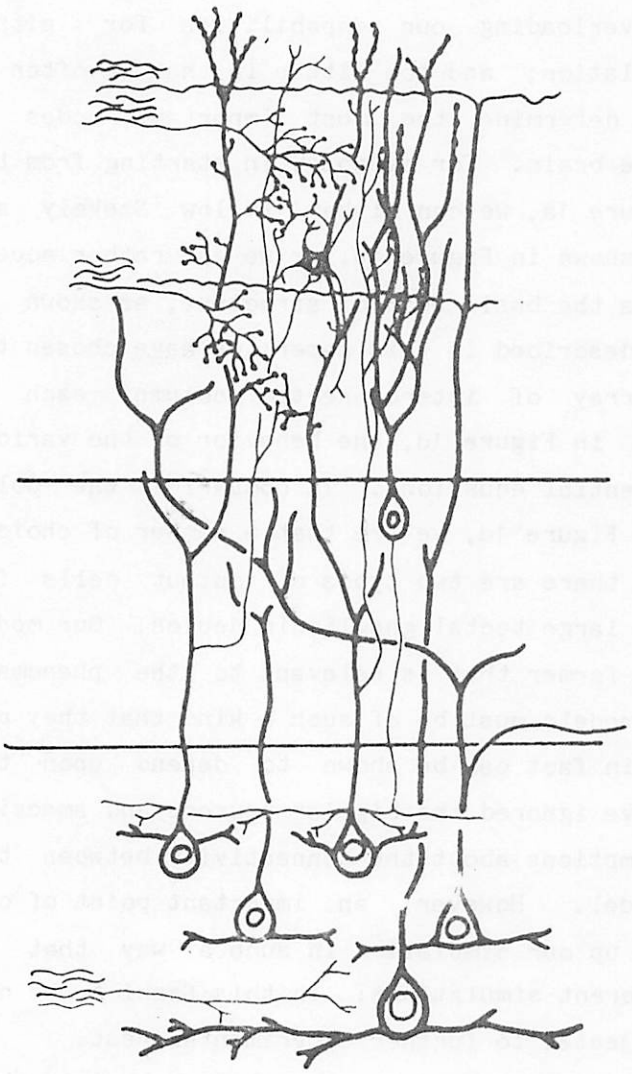
Even if we have made a satisfactory choice of how to correlate physiological units with anatomical units, and of the appropriate connectivity, we still have the



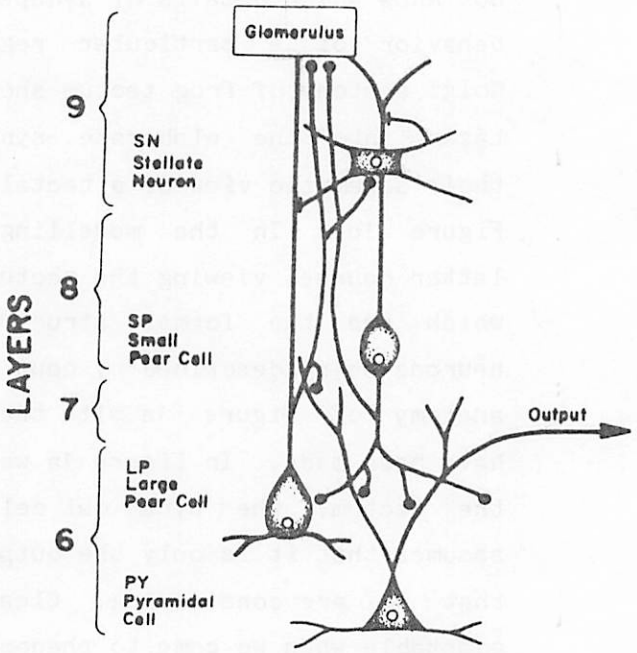
(a)



(b)



(c)



(d)

Figure 1.

Figure 1. (a) 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 & Lazar (1976)].

(b) Details of synaptic interaction of dendritic appendages, which exceed current models in intricacy [from Szekely & Lazar (1976)].

(c) Szekely and Lazar's schematic for a tectal column [from Szekely & Lazar (1976)].

(d) 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 PY, the SN, and the GL, and is inhibited by the SN. The SP excites the LP and PY 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.

problem of correlating cellular structure and function with the animal's overall behavior. In the modelling to be described in this paper, for example, we have assumed that the activity of the pyramidal cells correlates with the orienting response of the animal. We have also assumed that when a population of pyramidal cells is active, the resultant orientation is to the mean position corresponding to that population, though there is not yet evidence to discountenance further hypotheses, such as that the orientation will be to the spatial locus corresponding to the peak of the pyramidal cell activity.

We have already spoken of the need to have a family of models which allows one to experiment with a number of different connectivities and parameter settings for the cells of the model. There still remains the question of what is the appropriately detailed model. Is it the fact that the overall behavior of a large collection of cells depends critically on the fine details of the response performance of each individual neuron, or can we hope to use relatively simple, computationally efficient, neuron models and still derive significant information about the behavior of the population? In the models to be described below, we have described the behavior of the neuron by a simple differential equation linear in terms of the synaptically weighted input values, and have assumed that the input from one cell to another is given by a simple non-linear transformation of the membrane potential of the source cell. We believe that with such models we can probe whether the neural networks of different kinds can yield overall classes of behavior. Future research will be both less detailed -- trying to provide quantitative analyses correlating classes of neural networks with classes of behaviors--and more detailed, as we try to establish detailed parametric specifications which can be subjected to experimental test in the laboratory. Section 3 will provide a survey of some of the conceptual models that enter into our

search for "the style of the brain," while Section 4 will present the first three stages of our attempts to model in some detail the experimental studies of visuomotor coordination in frog and toad.

Our modelling methodology must be based not on a single "take it or leave it" model, but rather on the exploration of a variety of different connectivities within some overall paradigm of brain function. Thus, the models to be described below are dominated by two main considerations: the visual system of the animal must be considered in the context of the ongoing behavior of the animal -- thus the stress on visuomotor coordination, rather than on vision per se; and the analysis will be in terms of the interaction between concurrently active regions of the brain, rather than in terms of any simple one-way flow of information in a hierarchically organized system. We use the term cooperative computation to refer to this style of concurrent neural processing.

In addition to our concern for embedding the brain within the ongoing cycle of the animal's action and perception, and studying the brain itself in terms of the cooperative computation of interacting subsystems, the three models to be exhibited in Section 4 exhibit a style of "evolutionary" modelling. As a first approximation, we continually try to localize the neural processes underlying some overt behavior of the animal within some relatively small portion of the brain. As we come to analyze more functions, though, we find that each function may require activity in many portions of the brain, and that each portion of the brain will be involved in many different activities. Thus, having successfully modelled several phenomena, one should try as far as possible, when modelling a new phenomenon, to do it by minor adaptations of the previous model, preserving the earlier successes, rather than introducing an ad hoc model of a new brain region specifically designed to achieve the new specified task. Thus, in Section 4 we shall start with the model of the single tectal column shown in Figure 1d, and show that it is able to account for certain behavioral and neurophysiological data on facilitation; we shall then show how a linear array of such columns (Fig. 10a) can account for data on worm pattern recognition, without losing the ability of the individual column to exhibit facilitation to a localized stimulus. Finally, as shown in Figure 11a, we shall introduce pretectal cells and newness interneurons in interaction with the linear array of columns of Figure 10a, and see how, again without losing earlier properties of the model, we can account for the certain aspect of prey facilitation. In Section 5, we shall briefly outline further developments which continue to increase the behavioral repertoire of our evolving model, Rana computatrix.

3. BACKGROUND MODELS: IN SEARCH OF THE STYLE OF THE BRAIN

Before turning, in Section 4, to the first three stages in the evolution of Rana computatrix, we devote the present section to a number of background models

which establish the "style of the brain" with which we approach our modelling of visuomotor coordination in frog and toad.

Since we are concerned with motor control, we of course make use of such concepts as feedback and feedforward. In many treatments of these concepts in the literature on biological control systems, we see the use of lumped models. For example, the direction in which the animal should turn is encoded by a single angle variable. However, since we shall be concerned with the way in which patterns on the retina inpinge upon ongoing activity within the brain, we shall not consider it permissible to regard this angle as explicitly available in the brain as the value of, for example, firing of some neuron. Rather, we must consider it as encoded by the locus of the peak of activity within a neural array. Perhaps the first model of distributed motor control of this kind is that of Pitts and McCulloch (1947).

3a. Distributed Motor Control. Apter (1945, 1946) had shown that each half of the visual field of the cat maps topographically upon the contralateral superior colliculus. In addition to investigating this sensory map, she studied the motor map by strychninizing a single point on the collicular surface, flashing a diffuse light on the retina, and then observing which point in the visual field was affixed by the resultant change in gaze. She found that these sensory and motor maps were almost identical, and this basic finding has been replicated and extended in many recent studies. Starting from these data, Pitts and McCulloch developed the model shown in Figure 2. This outlined the reflex arc that extended from the eyes through the superior colliculus to the ocular-motor nuclei, thereby controlling the muscles that direct the gaze so as to bring the fixation point to the center of gravity of distribution of the visual input's brightness. (Our current knowledge of retinal preprocessing enables us to substitute for the term brightness such a term as contour information or an expression that describes some other feature of the input.) Pitts and McCulloch noted that excitation at a point on the left colliculus corresponds to excitation from the right half of the visual field and so should induce movement of the eye to the right; gaze is centered when excitation from the left is exactly balanced by excitation from the right. Their model is so arranged that each motor neuron controlling muscle fibers in the muscles that contract to move the eyeballs to the right, for example, should receive excitation summing the level of activity in a thin transverse strip of the left colliculus. This process provides all the excitation for muscles turning the eye to the right. Reciprocal inhibition by axons from nuclei of the antagonist eye muscles, which are excited similarly by the other colliculus, performs subtraction. The quasi-center of gravity's vertical coordinate is computed similarly. Eye movement ceases when and only when the fixation point is the center of gravity. Such a model leads to the idea that a plausible subsystem for vertebrate nervous systems may be one in which position of the input on the control surface encodes the target to which the muscular control will be sent. Of course, much remains to be done in turning such a

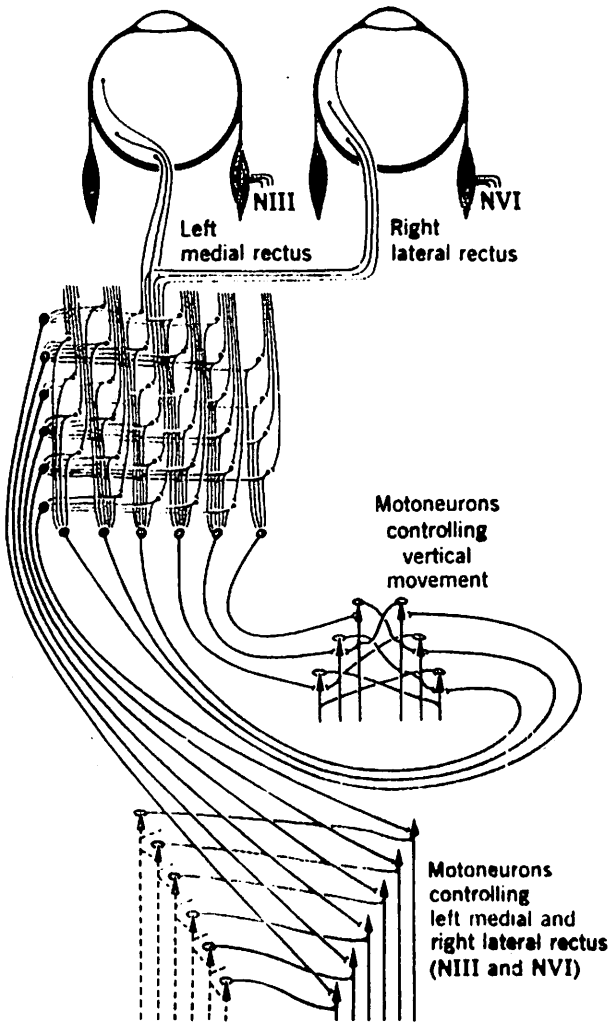


Figure 2. Pitts-McCulloch scheme for reflex control of eye position via superior colliculus. Eye can only be stationary when activity in two halves of colliculus is balanced. [Adapted from Pitts & McCulloch (1947).]

general scheme for distributed motor control into a specific model of a specific system. For example, the Pitts-McCulloch model does not give an account of ballistic movements. Again, it does not show us how, for increasing angles of deviation of the target, visual tracking might first evoke movement of eyes alone, then of eyes and head, and then of eyes, head, and trunk. It remains an important task in brain theory to explain how the output of a motor computer would control not a single pair of antagonist muscles, but rather a whole hierarchy of subcontrollers, in a distributed way.

3b. A Model of Frog's Snapping. Another problem is that in much visually guided behavior, the animal does not simply respond to "the center of gravity" of visual stimulation, but rather is responding to some property of the overall configuration. Consider, for example, the snapping behavior of frogs confronted with one or more fly-like stimuli.

Ingle (1968) found that in a certain region around the head of a frog, the presence of a fly-like stimulus elicits a snap; that is, the frog turns so that its midline is pointed at the stimulus and zaps it with its tongue. When confronted with two "flies," either of which is vigorous enough that alone it could elicit a

snapping response, the frog exhibits one of three reactions: it snaps at one of the flies, it does not snap at all, or it snaps in between at the "average fly." Didday (1970, 1976) offered the simple model of this choice behavior shown in Figure 3a. It is presented not as the state of the art -- in fact, we shall see a more recent model built upon it in Section 4c -- but rather as a clear example of the processing of structured stimuli to provide the input to a distributed motor controller akin to that shown in Figure 2. Didday used the term foodness to refer to the parameter representing the extent to which a stimulus could, when presented alone, elicit a snapping response. The task was to design a network that could take a position-tagged "foodness array" and ensure that usually only one region of activity would influence the motor control system. The model maintains the spatial distribution of information, with new circuitry introduced whereby different regions of the tectum compete in such a way that in normal circumstances only the most active region provides an above-threshold input to the motor circuitry. To achieve this effect we first introduce a new layer of cells that is in retinotopic correspondence to the "foodness layer," and that yields the input to the motor circuitry. In some sense, then, it is to be "relative foodness" rather than foodness that describes the receptive field activity appropriate to a cell of this layer.

Didday's transformation scheme from foodness to relative-foodness employs a population of "S-cells" that are in topographic correspondence with the other layers. Each S-cell inhibits the activity that cells in its region of the relative-foodness layer receive from the corresponding cells in the foodness layer by an amount that augments with increasing activity outside its particular region.

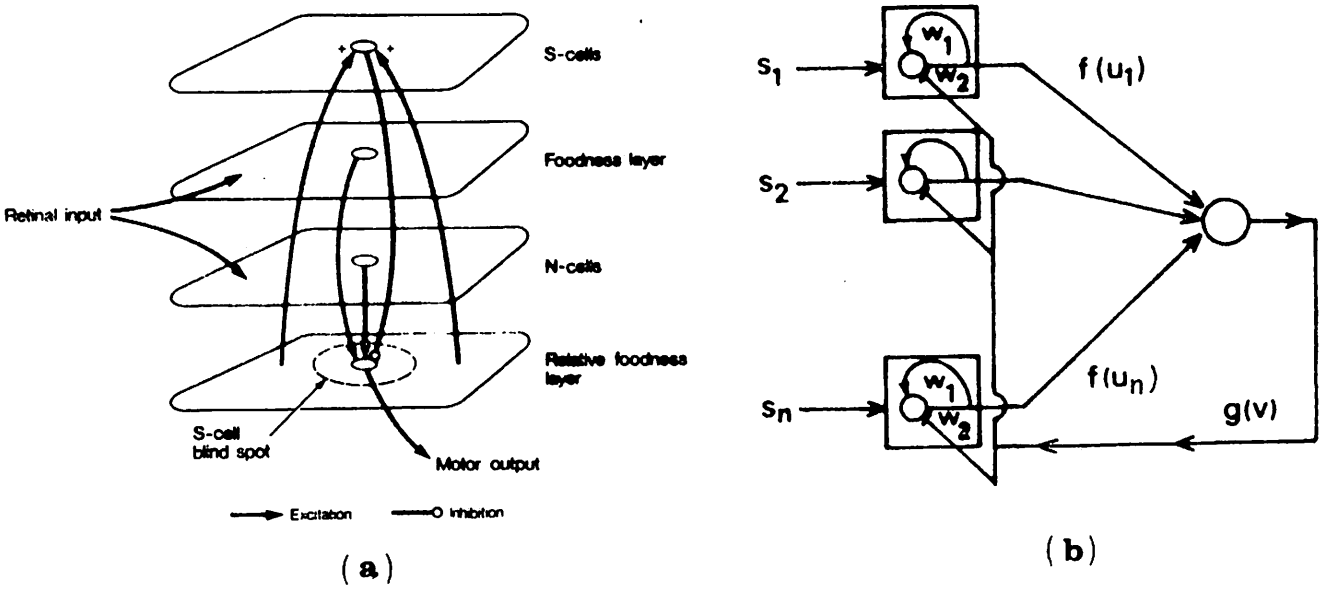


Figure 3. (a) Schematic view of Didday's model of interacting layers of neurons subserving prey-selection. (b) Primitive cooperation model in which the layer of S-cells of (a) is replaced by a single inhibitory neuron [from Amari & Arbib (1977)].

This ensures that high activity in a region of the foodness layer penetrates only if the surrounding areas do not contain sufficiently high activity to block it.

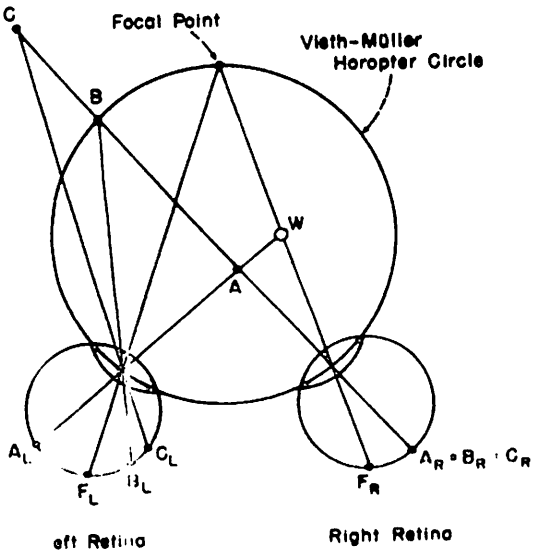
When we examine the behavior of such a network, we find that plausible interconnection schemes yield the following properties:

1. If the activity in one region far exceeds the activity in any other region, then this region eventually overwhelms all other regions, and the animal snaps at the corresponding space.

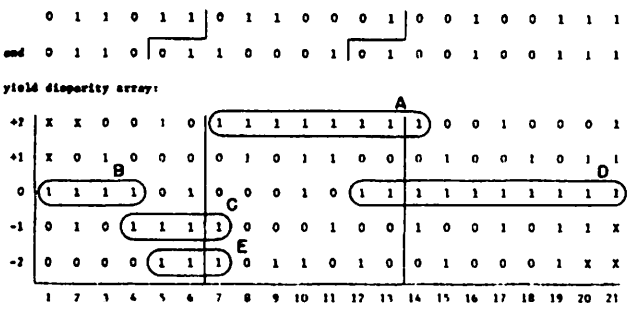
2. If two regions have sufficiently close activity then a) they may both (providing they are very active) overwhelm the other regions and simultaneously take command, with the result that the frog snaps between the regions; or b) the two active regions may simply turn down each other's activity, as well as activity in other regions, to the point that neither are sufficient to take command. In this case the frog remains immobile, ignoring the two "flies."

One trouble with the circuitry as so far described is that the buildup of inhibition on the S-cells precludes the system's quick response to new stimuli. If in case 2b above, for example, one of those two very active regions were to suddenly become more active, then the deadlock should be broken quickly. In the network so far described, however, the new activity cannot easily break through the inhibition built up on the S-cell in its region. In other words there is hysteresis. Didday thus introduced an "N-cell" for each S-cell. The job of an N-cell is to monitor temporal changes in the activity of its region. Should it detect sufficiently dramatic increase in the region's activity, it then overrides the inhibition on the S-cell and permits this new level of activity to enter the relative foodness layer. With this scheme the inertia of the old model is overcome, and the system can respond rapidly to significant new stimuli. Didday hypothesized that the S-cells and N-cells modelled the "sameness" and "newness" cells, respectively, that had been observed in the frog tectum. Regrettably, no experiments have been done to test this hypothesis.

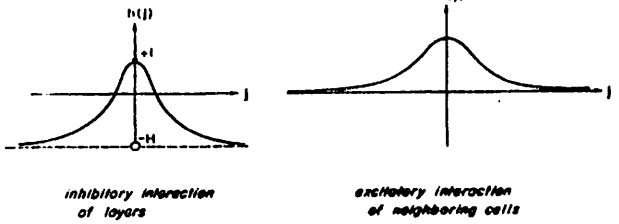
3c. Competition and Cooperation in Neural Nets. The above model of prey selection is an example of a broad class of models dealing with competition and cooperation in neural nets. As one example of a model of such a kind, let us consider the problem of stereopsis, or segmentation on depth cues. Julesz (1971) has designed "random-dot stereograms" in which each eye receives a totally random pattern, but in which there are correlations between the inputs to the two eyes. Specifically, the different regions in the two inputs are identical save for a shift in position, yielding a different disparity in the two retina (Fig. 4a). Although such a pattern for a naive subject can initially appear to be nothing but visual noise, eventually disparity matching takes place and the subject perceives surfaces at different depths. Barlow, Blakemore and Pettigrew (1967) and Pettigrew, Nikara, and Bishop (1968) have found that cells in cat visual cortex are tuned for retinal disparity, and similar cells are posited in the human. What presumably causes the initial



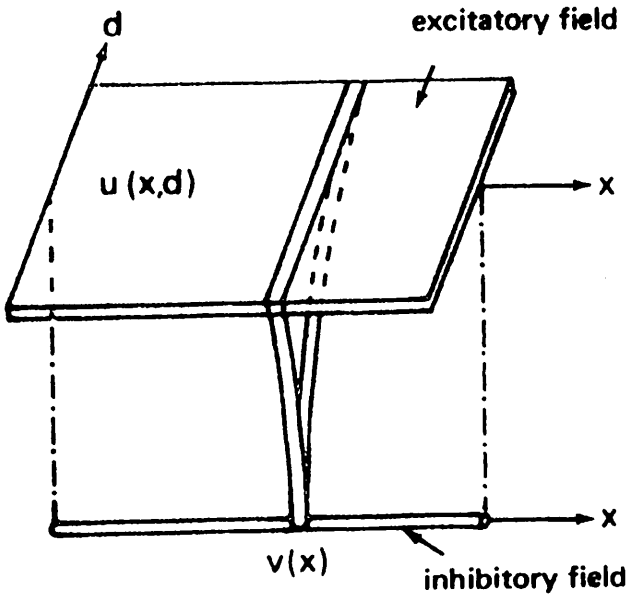
(a)



(b)



(c)



(d)

Figure 4. (a) Points projecting to the same point of one retina are projected to points with different disparities on the other retina. (b) The problem of resolving ambiguity: We conceptualize "layers" of cells (they are really in "columns"), one for each gross disparity. The aim is to segment the activity into connected regions. (c) Coupling coefficients for one approach to the problem: moderate local cross-excitation within layers; increasing inhibition between layers as difference in disparity increases. [From Arbib, Boylls & Dev (1974).] (d) The full model of competition and cooperation which allows the idea shown in (c) to be subject to mathematical analysis [from Amari & Arbib (1977)].

perception of visual noise is that in addition to the correct correlation of points in the two retinas, there are many spurious correlations, and computation is required to reduce them (Fig. 4b).

Dev (1975) [see also Sperling (1970), Arbib, Boylls and Dev (1974), Nelson (1975), and Marr and Poggio (1977)] has proposed that the cells of a given disparity be imagined as forming a population arrayed in a spatial map corresponding to the map of visual direction. Connections between cells could then be arranged so that nearby cells of a given disparity would be mutually excitatory, whereas cells nearby in visual direction but different in disparity would have inhibitory interaction (Fig. 4c). In this way, the activity of the array would organize into a pattern where in each region of visual direction, cells of only one disparity type would be highly active. As a result the visual input would eventually be segmented into a number of distinct surfaces.

In the stereopsis model, then, we have competition in the disparity dimension and cooperation in the other dimensions. The Didday model (Fig. 3a) can be regarded as the limiting case where there is only a competition dimension, namely that of prey location. Such informal observations have laid the basis for rigorous mathematical analysis of competition and cooperation in neural nets. For example, Amari and Arbib (1977) both offer the "primitive cooperation model" of Figure 3b which allows us to gain a mathematical handle on Didday's results, as well as a more sophisticated model, shown in Figure 4d, which allows us to provide a stability analysis of a model of the kind studied by Dev for stereopsis. Amari (1982) gives an up-to-date perspective on such models.

3d. Motor Schemas. We owe to the Russian school founded by Bernstein the general strategy which views the control of movement in terms of selecting one of a relatively short list of modes of activity, and then within each mode specifying the few parameters required to tune the movement. Where the Russians used the term synergy, we will use the term motor schema. The problem of motor control is thus one of sequencing and coordinating such motor schemas, rather than directly controlling the vast number of degrees of freedom offered by the independent activity of all the motor units. We have, to use the language of Greene, to get the system "into the right ballpark," and then to tune activity within that ballpark -- the dual problems of activation and tuning.

In the familiar realm of feedback control theory, a controller (which we will now think of as a motor schema) compares feedback signals from the controlled system with a statement of the desired performance of the system to determine control signals which will move the controlled system into ever greater conformity with the given plan. However, the appropriate choice of control signal must depend upon having a reasonably accurate model of the controlled system -- for example, the appropriate thrust to apply must depend upon an estimate of the mass of the object that is to be moved. Moreover, there are many cases in which the controlled system

will change over time in such a way that no a priori estimate of the system's parameters can be reliably made. To that end, it is a useful practice to interpose an identification algorithm which can update the parametric description of the controlled system in such a way that the observed response of the system to its control signals comes into greater and greater conformity with that projected on the basis of the parametric description. We see that when a motor schema is equipped with an identification algorithm (Fig. 5a) and when the controlled system is of the class whose parameters the algorithm is designed to identify, and when, finally, the changes in parameters of the controlled system are not too rapid, then in fact the combination of controller and identification algorithm within the motor schema provides an adaptive control system, which is able to function effectively despite continual changes in the environment.

3e. A Model of the Cerebellum. We have suggested that the problem of motor control is one of sequencing and coordinating motor schemas, rather than directly controlling the vast number of degrees of freedom offered by the independent activity of all the muscles. We have suggested that an "identification algorithm" can adapt a motor schema to changing conditions within some overall motor task. To see how this analysis can make contact with an interacting layers approach to neural circuitry, we now examine a model of the cerebellum (Arbib, Boylls, and Dev, 1974; Boylls, 1975, 1976). The model brings together the notion of a motor schema with the notion of maps as control surfaces, and is important in that it exhibits neural layers acting as control surfaces representing levels of activation for the coordination of muscles, complementing our study of retinotopic representations of visual input.

To provide neurophysiological data for the model, we consider cerebellar function in locomotion of the high decerebrate cat (Shik et al., 1966). Where Sherrington had noticed that stimulation of Deiter's nucleus in the standing animal would lead to extension of all the limbs, Orlovskii found that in the high decerebrate cat, stimulation of Deiter's nucleus during locomotion would not affect extension during the swing phase, but would increase extension during the support phase. Since the locomotory "motor schema" has been shown to be available even in the spiral cat (both in classical work by Sherrington (1910) and in modern studies (compare Herman et.al. 1976)), it seems reasonable to view the system in which the cerebellum and Deiter's nucleus are involved as providing an identification algorithm for the parametric adjustment of the spinal schema (Fig. 5b). We now turn to Boylls' model which shows how the adjustment of these parameters might be computed within the cerebellar environs.

As is well known (Eccles et al., 1967), the only output of the cerebellar cortex is provided by the Purkinje cells, which provide inhibitory input to the cerebellar nuclei. Each Purkinje cell has two input systems. One input is via a single climbing fiber which ramifies and synapses all over the Purkinje cell's

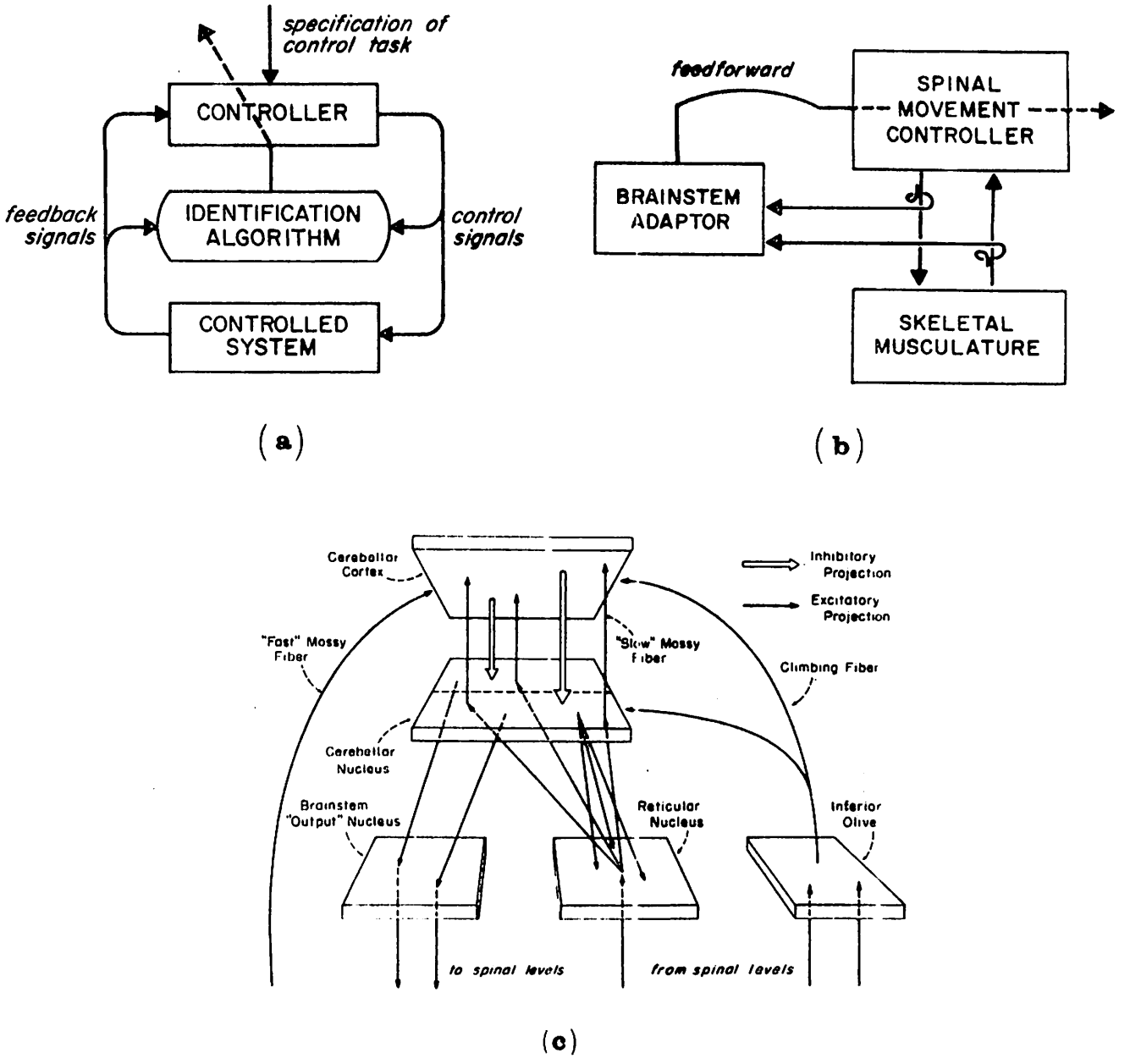


Figure 5. (a) An identification algorithm updates the parameters used to match the controller to the current properties of the system being controlled. (b) It is posited that the brainstem (cerebellum in interaction with various nuclei) serves as an identification algorithm for spinal movement controllers. (c) Schematic of the interacting control surfaces in the Boylls model of the tuning of motor schemas by cerebellum and related nuclei [from Arbib, Boylls & Dev (1974)].

dendritic tree. The other input system is via the mossy fibers, which activate granule cells whose axons rise up into the layer of Purkinje cell dendrites (which are flat, with the planes of all their dendritic trees parallel to one another) to form T's, whose crossbars run parallel to one another at right angles to the planes of the Purkinje dendritic trees. (There are a number of interneurons in the cerebellar cortex, but we shall not model these here, but shall instead concentrate on the basic cerebellar circuit of mossy and climbing fibers, and of granule and Purkinje cells.)

The climbing fiber input to a Purkinje cell is so strong that, when its climbing fiber is fired, a Purkinje cell responds with a sharp burst of four or five spikes, known as the climbing fiber response (CFR). Many authors have thought that the 'secret' of the climbing fiber is this sharp series of bursts, but Boylls suggested below that the true role of the climbing fiber input is to provide the suppression of Purkinje cell activity for as much as 100 milliseconds which has been found to follow the CFR (Murphy and Sabah, 1970).

The overall architecture of Boylls' model as played over an array of interacting control surfaces is shown in Figure 5c, which is an anatomical template of circuitry ubiquitous in cerebellar transactions. That is, specific labels could be given to, say, the 'brainstem output nucleus' as red or Deiters nucleus, the 'reticular nucleus' could be reticularis tegmenti pontis or paramedian, etc. From this architecture we gather that the output from the cerebellar nuclei via the brainstem 'output' nucleus results from the interaction between cerebellar cortical inhibition as supplied by the Purkinje cells and between drives from the reticular nucleus. Tsukahara (1972) has demonstrated the possibility of intense reverberation between the reticular and cerebellar nuclei following removal of Purkinje inhibition, and Brodal and Szikla (1972) and others have demonstrated the anatomical substrate for such loops, with a somatotopic mapping in both directions. We thus postulate that there will be explosively excitatory driving of the cerebellar nucleus by reticulo-cerebellar reverberation unless blocked by Purkinje inhibition.

The output of cerebellar tuning is expressed as a spatio-temporal neuronal activity pattern in a cerebellar nucleus, which can then be played out via the brainstem nuclei to spinal levels. A careful analysis of the anatomy enabled Boylls to predict that the agonists of a motor schema would be 'represented' along a saggital strip of the cerebellar cortex, while its antagonists will lie orthogonal to that strip (in the medio-lateral plane). Applications of this formula to cortical topography of the anterior lobe, as developed by Voogdt (1969) and Oscarsson (1973), allowed Boylls to identify particular cortical regions as associated with equally particular types of hindlimb-forelimb, flexor-extensor synergic groupings. This led to conclusions which are experimentally testable.

The Boylls model suggests that activity within the cerebellar nucleus is initiated through topically precise climbing fiber activity; the mechanism involves their direct cerebellar nuclear activation coupled with the suppression of the target Purkinje cell activity in the cortex via the above-mentioned 'inactivation response'. Once activity is installed in cortico-nuclear interactions via climbing fiber intervention, the underlying reverberatory excitation helps to retain or 'store' it. At the same time, this activity is transmitted to the cerebellar cortex on mossy fibers, eventually altering the inhibitory pattern in the nuclear region surrounding the active locus. The spread of parallel fibers yields a form of lateral inhibition which provides spatial 'sculpting' in a way depending on the elaborate geometry of cerebellar cortex and cortico-nuclear projections. Mossy

inputs of various types tune the resultant patterns to the demand of the periphery; and the program is spinally 'read out' as appropriate. Testing of the various hypotheses has required computer simulation of this neuronal apparatus. Simulation results corroborated the conjecture that cerebellar related circuitry could support the short-term storage of motor schema parameters initiated (and periodically refreshed) by climbing fiber activity.

4. THE FIRST THREE STAGES OF RANA COMPUTATRIX

4a. Facilitation of Prey-Catching Behavior. Frogs and toads take a surprisingly long time to respond to a worm. Presenting a worm to a frog for 0.3 sec may yield no response, whereas orientation is highly likely to result from a 0.6 sec presentation. Ingle [1975] observed a facilitation effect: if a worm were presented initially for 0.3 sec, then removed, and then restored for only 0.3 sec, the second presentation would suffice to elicit a response, so long as the intervening delay was at most a few seconds. Ingle observed tectal cells whose time course of firing accorded well with this facilitation effect (Fig. 6d). This leads us to a model [Lara, Arbib and Cromarty, in press] in which the "short-term memory" is in terms of reverberatory neural activity rather than in terms of the short-term plastic changes in synaptic efficacy demonstrated, for example, by Kandel [1978] in Aplysia. Our model is by no means the simplest model of facilitation -- rather, it provides a reverberatory mechanism for facilitation consistent with Ingle's neurophysiology and the known local neuroanatomy of the tectum. Unfortunately, the current knowledge of tectal circuitry is scanty, and much of the structure of the tectal column to be postulated below is hypothetical, and is in great need of confrontation with new and detailed anatomy and neurophysiology.

The model described in this section addresses facilitation at a single locus of tectum. Further developments address the interaction of a number of columns, and we shall discuss these in Sections 4b and 4c.

The anatomical study of frog optic tectum by Szekely and Lazar [1976] provides the basis for our model of the tectal column (Fig. 1a). In the superficial sublayers of tectum we see the thalamic input (which may also ramify in deeper layers), below which are the retinal type 1 and 2 inputs, with the retinal type 3 and 4 inputs deeper in turn. Deeper still, in layer 7, are the tectal efferents, which come from two cell types, the pyramidal cells and the so-called tectal ganglion cells. Our model of prey-catching will use only the pyramidal cells as efferents; we shall ignore the tectal ganglion cells which may (this is speculative) provide the output path for avoidance behavior. We incorporate the stellate cells as inhibitory interneurons, and ignore the amacrine interneurons. The other major components to be incorporated in our model are the large and small pear-shaped cells. Little of the anatomical connectivity of these cells is known,

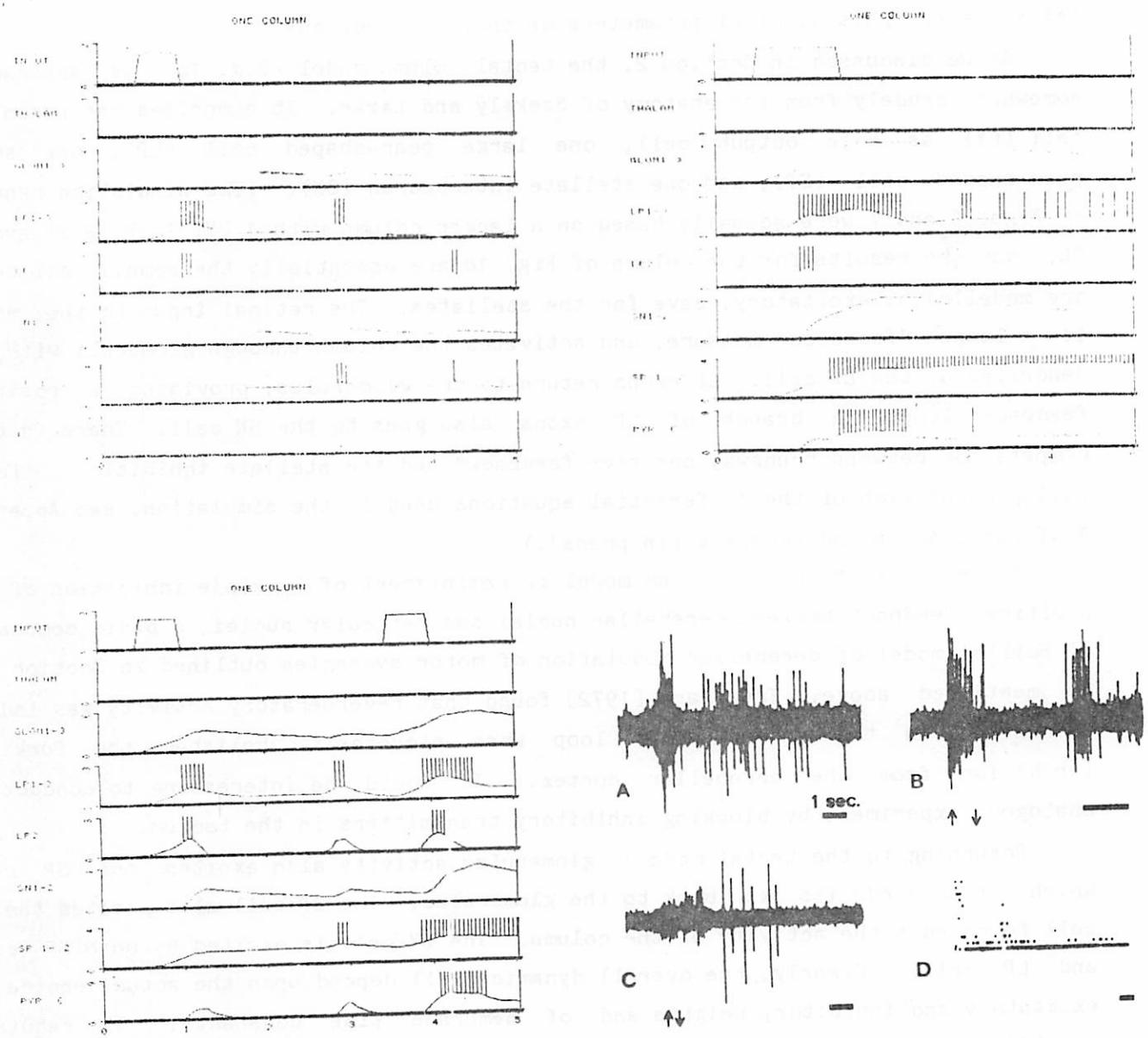


Figure 6. (a) Computer simulation of tectal cells response when a brief stimulus is presented. The onset of the stimulus produces a long lasting depolarizatin in the glomerulus which then fires the large pear-shaped cell (SP). This neuron in turn sends recurrent axons to the glomerulus and the stellate cell (SN) which acts as the inhibitory neuron in the column. When the inhibitory effect of SN releases the LP cell, a rebounding excitation occurs. 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) responds. (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.) [From Lara, Arbib & Cromary (in press).]

(d) 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 the behavior of a tectal cell is shown, 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).]

let alone the physiological parameters of their connections.

As we discussed in Section 2, the tectal column model (Fig. 1d) is abstracted somewhat crudely from the anatomy of Szekely and Lazar. It comprises one pyramidal cell (PY) as sole output cell, one large pear-shaped cell (LP), one small pear-shaped cell (SP), and one stellate interneuron (SN). (The simulation results of Figs. 6 and 7 were actually based on a larger column with 1 PY, 3 LP, 2 SP and 2 SN, but the results for the column of Fig. 1d are essentially the same.) All cells are modelled as excitatory, save for the stellates. The retinal input to the model is a lumped "foodness" measure, and activates the column through glomeruli with the dendrites of the LP cell. LP axons return to the glomerulus, providing a positive feedback loop. A branch of LP axons also goes to the SN cell. There is thus competition between "runaway positive feedback" and the stellate inhibition. (For a full presentation of the differential equations used in the simulation, see Appendix 1 of Lara, Arbib and Cromarty [in press].)

The role of SN in our tectum model is reminiscent of Purkinje inhibition of the positive feedback between cerebellar nuclei and reticular nuclei, a basic component of Boylls' model of cerebellar modulation of motor synergies outlined in Section 3. As mentioned above, Tsukahara [1972] found that reverberatory activity was indeed established in the subcerebellar loop when picrotoxin abolished the Purkinje inhibition from the cerebellar cortex. It would be interesting to conduct an analogous experiment by blocking inhibitory transmitters in the tectum.

Returning to the tectal model: glomerular activity also excites the SP cell which also sends its axon back to the glomerulus. The SP cell also excites the LP cell to recruit the activity of the column. The PY cell is excited by both SP cell and LP cell. Clearly, the overall dynamics will depend upon the actual choice of excitatory and inhibitory weights and of membrane time constants. It required considerable computer experimentation to find the weights that yielded the neural patterns discussed below. Further study was devoted to a sensitivity analysis of how weighting patterns affect overall behavior. It is our hope that our hypotheses on the ranges of the parameters involved in the model will stimulate more detailed anatomical and physiological studies of tectal activity.

Excitation of the input does not lead to runaway reverberation between the LP and its glomerulus; rather, this activity is "chopped" by stellate inhibition and we see a period of alternating LP and SN activity. The SP cell has a longer time constant, and is recruited only if this alternating activity continues long enough.

In one simulation experiment, we graphed the activity of the pyramidal cell as a function of the time for which a single stimulus is applied (Fig. 7a). There is, as in the experimental data, a critical presentation length below which there is no pyramidal response. Input activity activates the LP, which re-excites the glomerulus but also excites the SN, which reduces LP activity. But if input continues, it builds on a larger base of glomerular activity, and so over time there is a build-up of LP-SN alternating firing. If the input is removed too soon, the

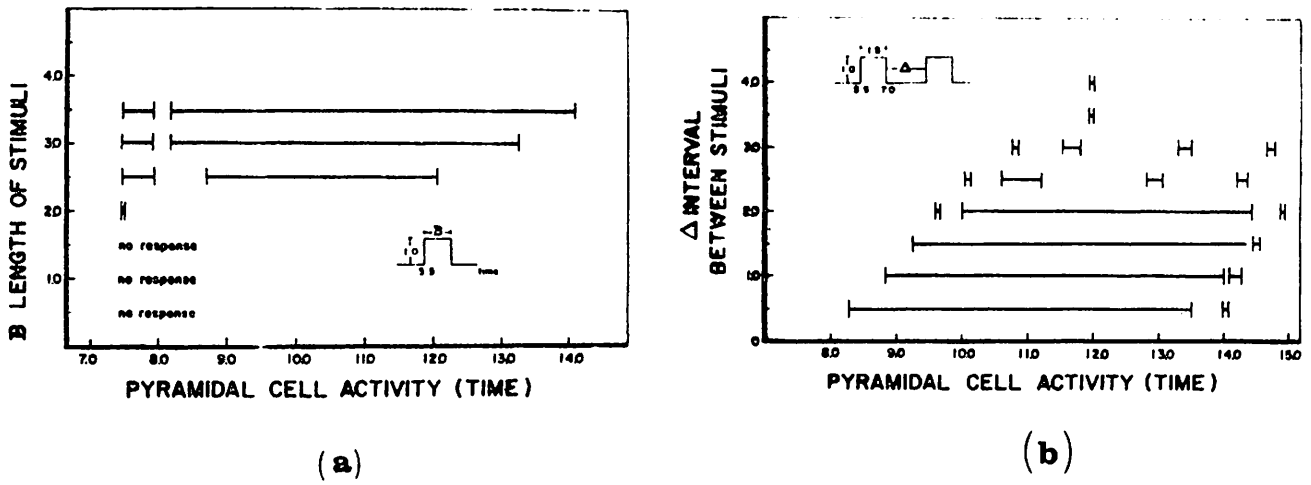


Figure 7. (a) Computer simulation of the PY behavior when stimuli are presented for different intervals. (b) Computer simulation of the temporal pattern of the facilitation process after the presentation of a brief stimulus.

reverberation will die out without activating the SP cells enough for their activity to combine with the LP activity and trigger the pyramidal output. However, if input is maintained long enough, the reverberation may continue, though not at a level sufficiently high to trigger output. However, a second simulation experiment (Fig. 7b) shows that re-introduction of input within a short time after cessation of this "subthreshold" length of input presentation can indeed "ride upon" the residual reverberatory activity to build up to pyramidal input after a presentation time too short to yield output activity on an initial presentation.

4b. A Simple Model of Pattern Recognition in the Toad. The facilitation model was 'local' in that it analyzed activity in a small patch of tectum rather than activity distributed across entire brain regions. We now outline Ewert's [1976, for a review] study of pattern recognition in the toad, analyzing what features of a single moving pattern will increase the animal's snapping responses. We then show how a one-dimensional array of tectal columns, of the type studied in the previous section, can model certain of these data. In Section 5, we briefly discuss our use of a two-dimensional array of such columns to model the whole range of Ewert's data on pattern recognition.

The toad is placed in a transparent cylinder. An object moves around a circular track concentric with, and on the floor outside, the cylinder. Some objects elicit no response. Other objects do elicit an orienting response (though the cylinder wall prevents the toad from actually snapping). Since the object keeps moving along its track, it can elicit a second response, and a third, and so on. Ewert's suggestion, then, is that the more 'attractive' is the object, the more frequently will the toad orient to it, so that the response rate is a measure of foodness. (Note a paradox here. The less attractive the object, the greater the

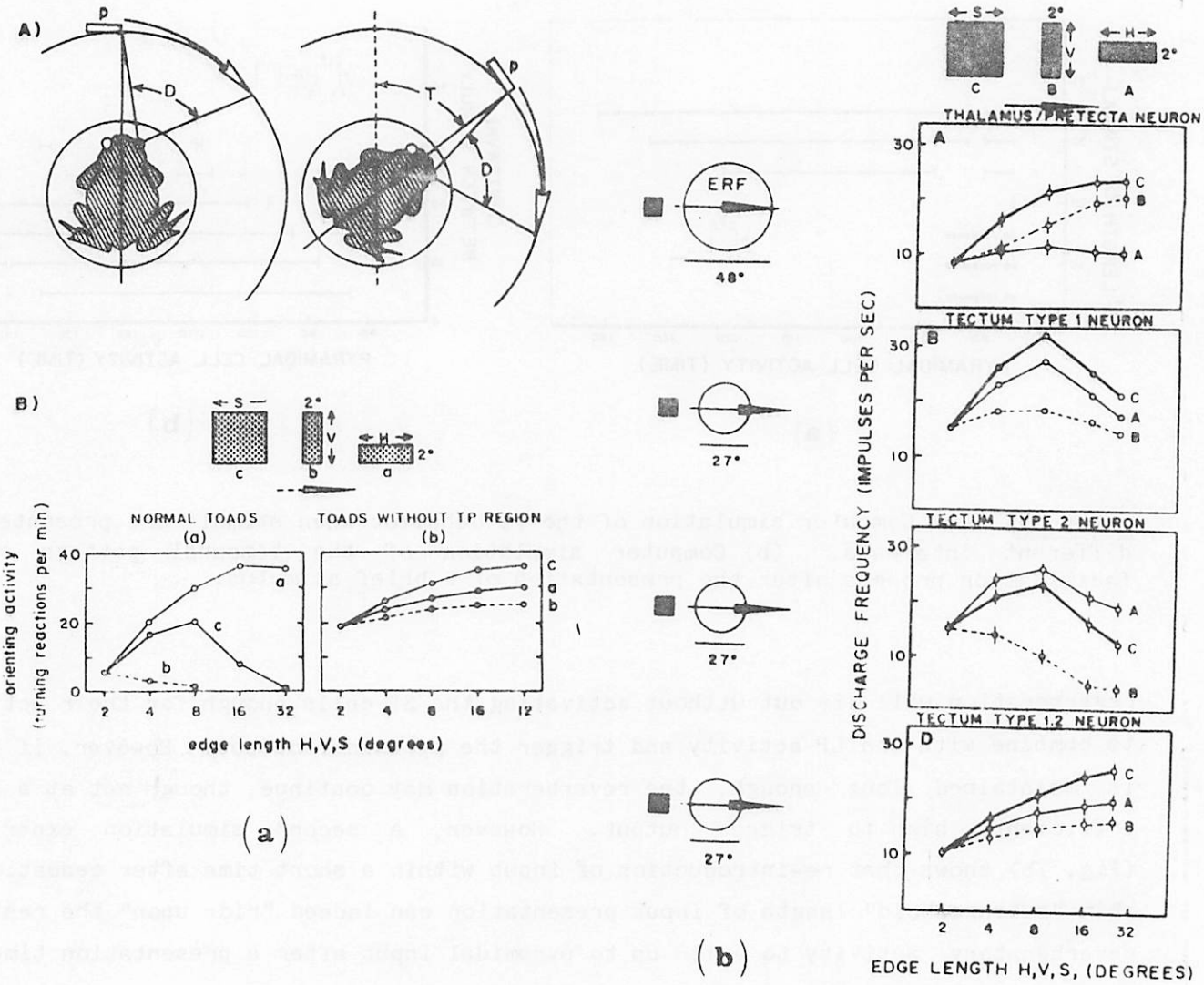


Figure 8. (a) Prey orienting behavior to different configuration of the stimulus. A) Turning reaction to the stimulus presentation. B) Orienting activity to three configurations (a,b,c): facilitation to stimulus a, inhibition to stimulus b, and an initial facilitation and then an inhibition to stimulus c. When pretectum ablation occurs this discrimination disappears.

(b) Tectal and pretectal cell activity to different configurations of the stimulus (a,b,c). A) Response of a pretectal neuron which is mostly sensitive to stimulus b and c. B and C show the response of two tectal cells to the three types of stimuli. Neuron C response is mostly sensitive to stimuli type a and c, and its response is greatly reduced for stimulus type b. This response is similar to the observed behavioral response. D shows the response of both tectal cells (B and C) without pretectum and how the discriminative abilities of these cells are lost. [From Ewert (1976).]

integration time to a response, and thus the greater the distance the animal has to move to orient towards the object if it orients at all.)

Ewert presented three types of rectangular stimuli: a "worm" subtending 2 degrees in the direction normal to the motion, and some d degrees in the direction of motion; an "antiworm" subtending some d degrees in the direction orthogonal to motion, and 2 degrees in the direction of motion; and a "square" subtending d degrees in both directions. The prey dummy was moved at 20 degrees per second at a distance of about 7 cm from the toad. Ewert studied the toad's response rate for

each stimulus for a range of different choices of d degrees (fixed for each trial) from 2 degrees to 32 degrees (Fig. 8). For d = 2, the three stimuli were, of course, the same. They elicited an orienting activity of 2 to 3 turning reactions per minute. For the "worm", the orienting activity increased to an asymptote of 35 turns per minute at d = 16; for the "antiworm", the orienting activity decreased rapidly to extinction at d = 8; while for the square the orienting activity reached a peak of about 20 turns per minute at d = 8, and then decreased to zero by d = 32. (The square gives the impression of a competition between "worm" excitation and "antiworm" inhibition.)

Ewert repeated this series of behavioral experiments in toads with PT-lesions, and found that for none of the stimuli was there decreased response with increased values of d. This more detailed evidence for PT inhibition of tectally-mediated orienting was further elaborated by neurophysiological recording of PT and tectal neurons in the behaving toads. In the intact toad, PT-neurons had a response rate insensitive to increasing d for "worms", but the response increased with d for "antiworms", and even more rapidly for squares. Tectum type 1 neurons were insensitive to changing d for "antiworms", but had a peak of response at d = 8 for both "worms" and squares; while the firing rate of tectum type 2 neurons was similar to the orienting activity of the intact toad -- monotonically declining with d for "antiworms", peaking at d = 8 for squares, and declining slightly after d = 8 for "worms". (Note the slight discrepancy here -- one would expect the response to "worms" to be non-decreasing if, as Ewert does, one takes tectal type 2 activity as the neural correlate of orienting behavior.)

On this basis, Ewert postulated a simple model: A filter in PT responds best to an antiworm stimulus; a tectum type 1 cell responds as a filter tuned to a worm

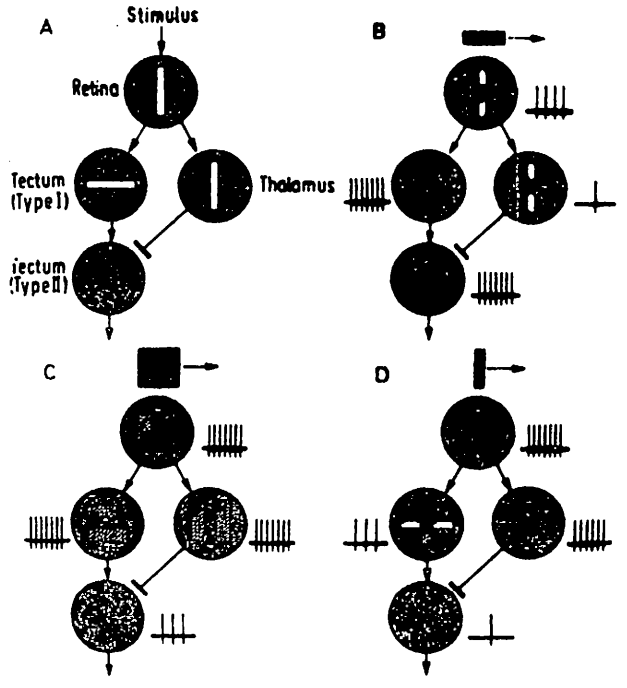


Figure 9. Schematic representation of the Ewert-von Seelen model of worm-'antiworm' discrimination. The tectum type II cell is excited by a tectal 'worm filter' and a thalamic-pretectal 'antiworm filter'.

stimulus; and a tectum type 2 cell is excited by the tectal type 1 cell and inhibited by a PT-cell (Fig. 9). Thus the type 2 cell responds with increased activity to increasing d for a worm stimulus; with decreased activity to increasing d for an antiworm stimulus; and with some tradeoff (dependent upon the actual parameters of the filters and the connectivity) for a square. Ewert and von Seelen [1974] fitted parameters to a linear formulation of this model to fit (part of) the response curves observed by Ewert. Note, however, that the domain of linearity is strictly limited; and that the model yields the average firing rate of the neuron: the model is thus lumped over time, and says nothing about the temporal pattern of neuronal interactions. Arbib and Lara [in press] have studied a one-dimensional array of tectal columns as in Figure 10a (without PT interaction) to provide a model of spatiotemporal neural interactions possibly underlying Ewert's "worm" phenomena. For example, in the Ewert study of the toad's response to an object moving along a track, we may regard the object's movement at one position as facilitating the animal's orientation to the object in a later position. The key question here is "How does the facilitation build up in the right place?" Part of the answer lies in noting the large receptive fields of the tectal columns; and analyzing how activity in a population of tectal columns can yield orientation in a particular direction. Thus, rather than analyzing activity in a single column, Arbib and Lara [in press] study the evolution of a waveform of activity in a one-dimensional array of columns (Fig. 10a). We show in Figs. 10b, c, d the response to a moving stimulus of various lengths. These reproduce Ewert's observations on the increasing attraction of a 'worm' with increasing length; Arbib and Lara also report a number of other computational experiments. The elaboration of this model to a two-dimensional array of columns (Lara, Cervantes and Arbib, 1982) is integrated with our model (Section 3c) of tectal-pretectal interactions in prey-selection to yield a model rich enough to extend an explanation of Ewert's data on pattern recognition into the temporal domain in a way which addresses the antiworm and square data, as well as the worm data.

4c. A Model of Prey-Selection. We saw in Section 3b that Ingle [1968] had studied the response of frogs to pairs of fly-like stimuli, each of which was such that when presented alone it would elicit a snapping response, and found that, under differing conditions, the animal would snap at one of the stimuli, snap between them, or not snap at all. We now turn to a model of such prey-selection which refines the Didday model discussed above (Fig. 3a) but differs in that -- in view of Ewert's study of PT-lesions -- it uses PT-tectal interactions, rather than positing that all the necessary circuitry is embedded in the tectum. Moreover, the new model extends the 'array of tectal columns' model of Section 4b to provide yet a third stage in the evolution of Rana Computatrix.

To make that transition from the Didday model, we now identify the "foodness layer" of Figure 3 with the retinal outflow to tectum and pretectum, and identify

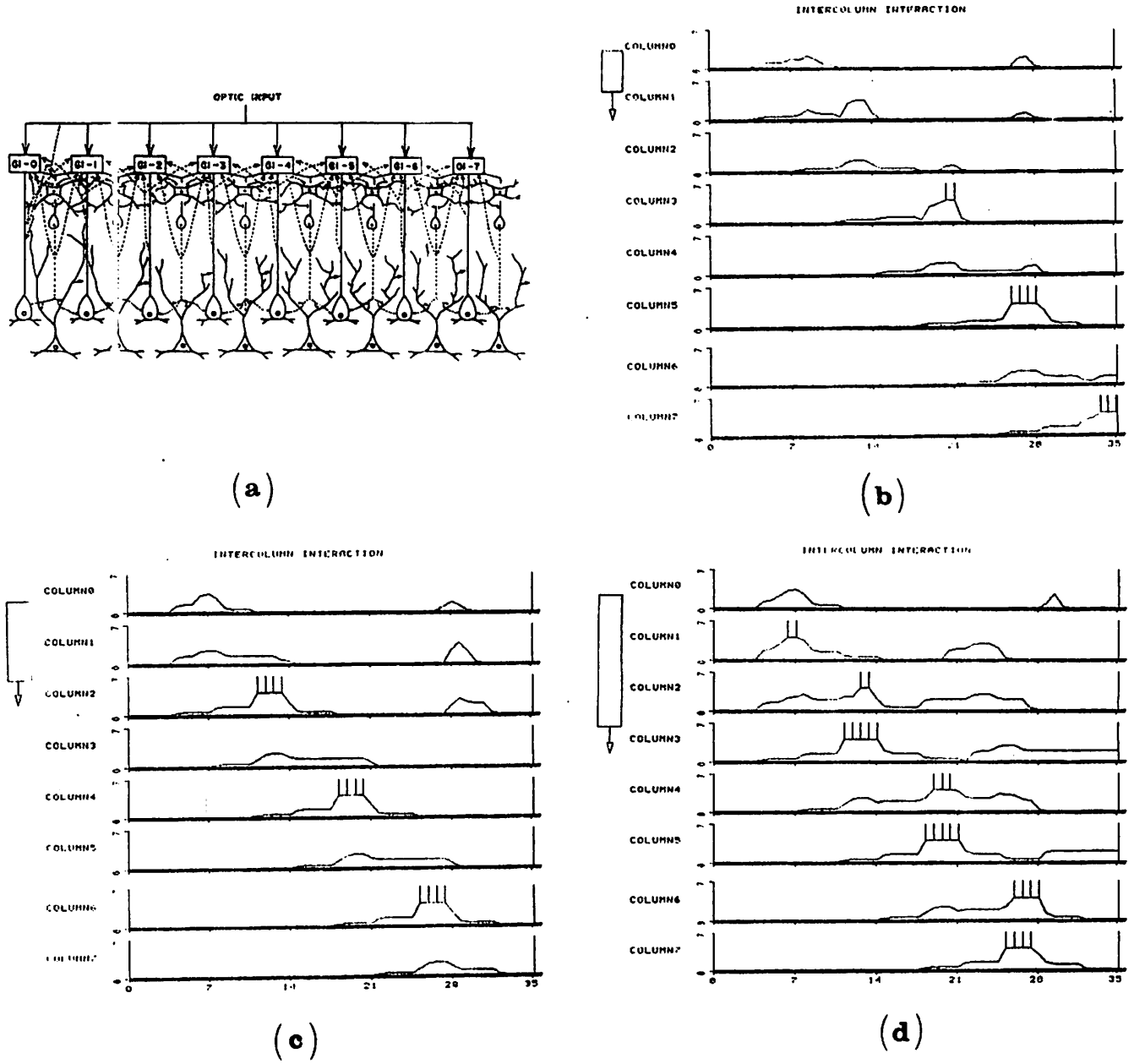


Figure 10. (a) Architecture of the model of the tectum. Each column is constituted by one GL (glomerulus), one LP (large pear-shaped) cell, one SP (small pear-shaped) neuron, one SN (stellate neuron), and one PY (pyramidal cell). The afferents are the optic fibres that arrive at the GL, LP, SP, and PY cells, and the efferents are the PY axons. LP cells are activated by the GL and the optic input, and they send recurrent axons to their own as well as neighboring glomeruli. The SN are activated by the LP cells, and they inhibit LP and SP neurons of their own as well as neighboring columns. The SP receive excitation from GL and are inhibited by SN; finally, PY receives afferents from the retina, the LP and SP neurons.

(b), (c) and (d) present a computer simulation of tectal response to a moving stimulus of different sizes. The graphs show the behavior of the 8 PY neurons of the tectal model of (a) to a moving stimulus. (b) Notice that in this case an alternate response is given in columns 3, 5, and 7 when the stimulus size only covers one glomerulus. (c) Here the stimulus covers 2 glomeruli simultaneously. The results show that the strength of activation increases when the size of the object is elongated. The latency of response is also shorter (column 2). (d) In this figure the stimulus covers 3 GL simultaneously. It can be seen that the latency of response is shorter and the total activity is greater than in (b) and (c). Notice that all columns fire with this stimulus. [From Arbib & Lara (in press).]

the "relative foodness layer" with the pyramidal cells of tectum. We now see that Figure 3a is too simple because it does not include other cells of the tectal column. The new model [Lara and Arbib, in press] interconnects a one-dimensional array of tectal columns with a layer of cells called S-cells, in retinotopic correspondence with the columns, which represent cells of the pretectum-thalamus (Fig. 11a). (In the 1970 model, the S-cells were identified with the sameness cells reported in the tectum by Maturana, Lettvin et al.) Each S-cell is excited by activity in the relative foodness layer, save for a blind spot centered at the locus corresponding to that of the S-cell. In the Didday model, the S-cell then provides an inhibitory input to cells within its blind spot on the relative foodness layer. Lara and Arbib [to appear], however, do not make the corresponding assumption that an S-cell must inhibit the PY cell in the corresponding tectal column. Rather they conduct a number of experiments on the dynamic consequences of choosing different sites for pretectal inhibition of columnar activity. The reader is referred to their paper for details.

The system described so far exhibits hysteresis. Should a new peak be introduced in the input array, it may not affect the output activity even if it is rather large, for it may not be able to overcome the considerable inhibition that has built up on the S-cells. The model thus follows Didday in postulating a further array of NE-cells (representing the newness cells of Lettvin et al.) which register sudden changes in input, and uses these to interrupt the ongoing computation to enable new input to affect the outcome.

Clearly, the detailed dynamics of the model will depend on the size of the blind spot, and the relative parameters of excitation and inhibition. We were able to adjust the coefficients in such a way that with several peaks in the foodness input array, the activity passed through to the tectal column would excite the S-cells in such a way that they would lower the corresponding peaks in tectal activity. However, if one peak were stronger than the others, it would be less inhibited, and would begin to recover; in doing so, it would suppress the other peak more, and thus be inhibited less; the process continuing until the stronger peak recovered sufficiently to control a "snap" in the corresponding direction (Fig. 11c). However, there were cases in which the mutual suppression between two peaks sufficed to hold each below a level sufficient to release behavior (Fig. 11b). We also showed that if the tectum became habituated to one of the stimuli, a standoff would be resolved in favor of the novel stimulus (Fig. 11d).

5. A PERSPECTIVE FOR FURTHER MODELLING

In Section 4, we exhibited an evolutionary sequence of models -- tectal column, one-dimensional array of columns; array with pretectal inhibition -- which explains an increasingly broad range of behavioral data on visuomotor coordination in frog

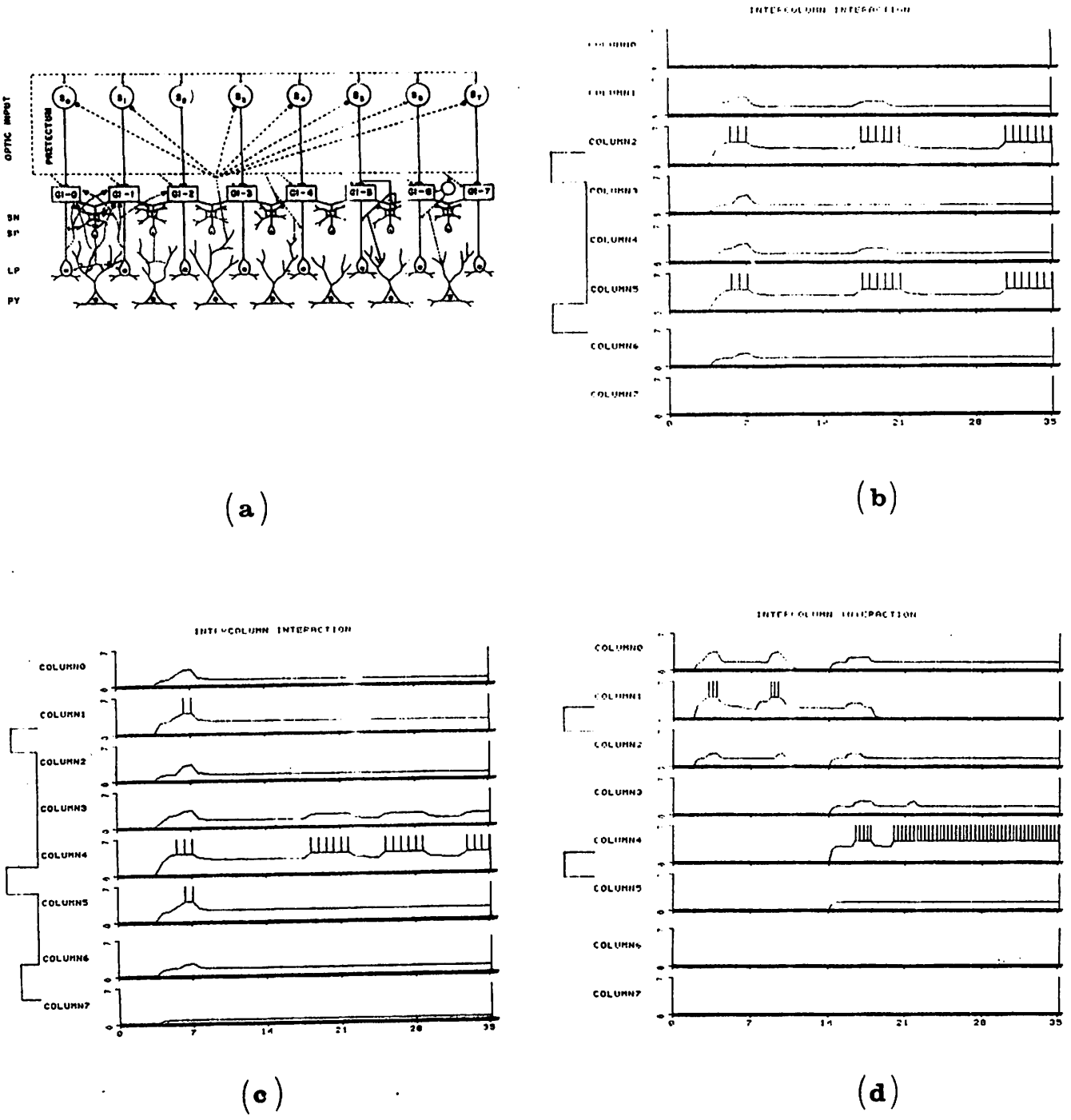


Figure 11. (a) Architecture of the model for the interactions between tectum and pretectum in prey selection. Each column receives the afferents from one sameness neuron; each PY (pyramidal) neuron excites all pretectal cells except the one whose blind spot is in its receptive field. The NE (newness) neurons arrive at the same site as the corresponding optic fibres. (b) Computer simulation of the behavior of PY neurons to two equally intense stimuli. The stimuli are presented in columns 2 and 5. Notice that an alternation of excitation and inhibition is present without convergence to any of the stimuli. (c) Computer simulation of the behavior of PY neurons to two equally intense stimuli to columns 2 and 5 biased by a third one. When the third stimulus is applied in column 7, then the response converges to the stimulus presented in column 5. (d) Computer simulation of habituation effects on PY activity. We first present a stimulus in column 1. After a period of rest, we present two equally intense stimuli in columns 1 and 4, the response converging to column 4, because the pathway of column 1 is habituated.

and toad. We note three important features of the style of modelling developed here.

1. New phenomena are addressed not by the creation of ad hoc models but by the orderly refinement and expansion of models already created. Of course, we expect that future development along this line will lead to redefinition and refinement of earlier models, rather than simple addition of new circuitry in each case. On the other hand, we would expect that the model, once sufficiently developed, will explain many data beyond those which specifically entered into its design.

2. Each 'model' in the sequence is in fact a 'model-family'. We design a family of overall models, and then conduct simulation experiments to see which choices -- of connectivity, synaptic weights, time constants -- yield neural dynamics, and input-output relations, compatible with available data.

3. The choices mentioned above are only loosely constrained by the experimental data presently available. To carry out simulations, we make choices which often must, perforce, go beyond these data. In making such choices, we form explicit hypotheses (whose details are spelt out in our papers cited in Section 4) which may serve to stimulate new experiments. These experiments in turn will stimulate more refined modelling. The continuing cycle will lead to an increasingly sophisticated understanding of the neural mechanisms of visuomotor coordination.

We close with a brief discussion of future directions for this modelling effort. We have already mentioned the transition from a one-dimensional to a two-dimensional array of tectal columns (and corresponding pretectal elements) as a further development of Rana Computatrix in Lara, Cervantes and Arbib (1982).

The models of Section 4 have nothing to say about the control of avoidance behavior, nor does the basic version described here address more than a few of the prey-predator discrimination phenomena discussed in Section 4b. A two-dimensional array of columns will allow us to study the full range of these phenomena.

Other developments in the modelling of frog brain visuomotor coordination will come as we try to take more and more regions of the brain into account -- for example, the cerebellum, the retina, and the forebrain. We will also want to look at more complex behaviors of the animal, not only prey-selection and predator avoidance, but also behaviors which require the integration of a number of motor schemas. For example, we are currently considering the way in which an animal will approach a worm when a vertical paling barrier is interposed. In this case, the animal's behavior can be analyzed in terms of the coordinated activation of three motor schemas: one for side-stepping, one for orienting, and one for snapping. The understanding of this behavior, then, reinforces our need to model the animal's behavior in terms of the cooperative computation of a number of brain regions. We have now adapted the Dev conceptual model of stereopsis, described above in Section 3c, to a model of depth perception in the frog, in which we take account not only of the disparity cues available in the binocular field of the animal, but also of

accommodation cues available in the monocular field (the animal can still strike with an accurate depth estimation if it has only one eye, and the worm is presented in the monocular field corresponding to that eye).

Clearly, then, developments in modelling will require both the generation of general concepts for vision and motor control, as well as specific studies which try to provide a variety of detailed models adapted to experimentation on different kinds of animals and different kinds of situations. We shall also need to get a better understanding of how regions of the brain are coordinated in complex behaviors. Finally, it will not be enough to understand how the adult brain behaves in any given situation; we must also understand the development of the brain (for example, by modelling the development of retinal-tectal connections), and by studying learning mechanisms.

There are further refinements not incorporated into the basic model. Increased motivation (due, e.g., to food odor or to hunger) will cause the animal to snap at larger moving objects than it would otherwise approach. Such an effect might be modelled by direct excitation of tectal columns, or by diffuse inhibition of the S-cells, probably under the control of telencephalic regions. Forebrain mechanisms allow the animal to learn simple discriminations. And there are habituation phenomena which we have begun to model (Fig. 11d). Habituation disappears when there is PT ablation. Moreover, the habituation is stimulus specific, and it appears that pattern recognition is necessary both for habituation and dishabituation to occur. For example, Ewert has studied habituation of a toad's snapping response to simple moving patterns and has discovered a hierarchy -- an ordering $A \leq B$ of patterns,such that if the toad habituates to A it will automatically be habituated to B, but not vice versa. Such data provide a continuing challenge to the theory-experiment interaction that will drive the future evolution of Rana Computatrix.

REFERENCES

Amari, S., 1982, Competitive and Cooperative Aspects in Dynamics of Neural Excitation and Self-Organization. In: Competition and Cooperation in Neural Nets (S. Amari and M.A. Arbib, Eds.), Lecture Notes in Biomathematics, Springer-Verlag (this volume).

Amari, S., and Arbib, M.A., 1977, Competition and cooperation in neural nets. In: Systems Neuroscience, (J. Metzler, Ed.), New York: Academic, p. 119-165.

Apter, J.T., 1946, Eye movements following strychninization of the superior colliculus of cats. J. Neurophysiol. 9: 73-85.

Apter, J.T., 1945, Projection of the retina on the superior colliculus of cats. J. Neurophysiol. 8: 123-134.

Arbib, M.A., 1981, Perceptual structures and distributed motor control. In: Handbook of Physiology: The Nervous System II. (V.B. Brooks, Ed.), Bethesda, Md.: Amer. Physiological Society, 1449-1480.

Arbib, M.A., 1982, Rana Computatrix: An Evolving Model of Visuomotor Coordination in Frog and Toad. In: Machine Intelligence 10 (J. Hayes and D. Michie, Eds.), Ellis Horwood.

Arbib, M.A., Boylls, C.C., and Dev., P., 1974, Neural models of spatial perception and the control of movement. In: Cybernetics and Bionics, (W.D. Keidel, W. Handler, and M. Spreng, Eds.), Munich: Oldenbourg, 216-231.

Arbib, M.A., and Lara, R. (in press), A neural model of the interaction of tectal columns in prey-catching behavior. Cognition and Brain Theory, 5.

Barlow, H., 1953, Summation and inhibition in the frog's retina. J. Physiol. (Lond.) 119: 69-88.

Barlow, H.B., Blakemore, C., and Pettigrew, J.D., 1967, The neural mechanism of binocular depth discrimination. J. Physiol. 193: 327-342.

Boylls, C.C., 1974, A Theory of Cerebellar Function with Applications to Locomotion. Ph.D. Thesis, Stanford University.

Dev, P., 1975, Computer simulation of a dynamic visual perception model. Int. J. Man-Mach. Stud., 7: 511-528.

Didday, R. L., 1970, The Simulation and Modelling of Distributed Information Processing in the Frog Visual System. Ph.D. Thesis, Stanford University.

Didday, R. L., 1976, A model of visuomotor mechanisms in the frog optic tectum. Math. Biosci. 30: 169-180.

Ewert J. P., 1976, The visual system of the toad: behavioral and physiological studies in a pattern recognition system. In: The Amphibian Visual System: A Multidisciplinary Approach. (K. Fite, Ed.), Academic Press, pp. 142-202.

Ewert, J. P. and von Seelen, W., 1974, Neurobiologie and System-Theorie eines visuellen Muster-Erkennungsmechanismus bei Kroten. Kybernetic 14: 167-183.

Ingle, D., 1968, Visual releasers of prey-catching behavior in frogs and toads. Brain Behav. Evol. 1: 500-518.

Ingle, D., 1973, Disinhibition of tectal neurons by pretectal lesions in the frog. Science 180: 422-424.

Ingle, D., 1975, Focal attention in the frog: behavioral and physiological correlates. Science 188: 1033-1035.

Ingle, D., 1976, Spatial visions in anurans. In: The Amphibian Visual System. (K. Fite, Ed.), Academic Press, New York, pp. 119-140.

Julesz, B., 1971, Foundations of Cyclopean Perception. Chicago: Univ. of Chicago Press.

Kandel, E.R., 1978, A Cell Biological Approach to Learning. Grass Lecture No. 1. Society for Neuroscience: Bethesda, MD.

Lara, R., and Arbib, M.A. (to appear), A neural model of interaction between tectum and pretectum in preytectum in prey selection.

Lara, R., Arbib, M.A., and Cromarty, A.S. (in press), The role of the tectal column in facilitation of amphibian prey-catching behavior: a neural model. J. Neuroscience.

Lara, R., Cervantes, F., and Arbib, M.A., 1982, Two-dimensional Model of Retinal-Tectal-Pretectal Interactions for the Control of Prey-Predator Recognition and Size Preference in Amphibia. In: Competition and Cooperation in Neural Nets (S. Amari and M.A. Arbib, Eds.), Lecture Notes in Biomathematics, Springer-Verlag (this volume).

Lettvin, J. Y., Maturana, H., McCulloch, W. S. and Pitts, W. H., 1959, What the frog's eye tells the frog brain. Proc. IRE. 47: 1940-1951.

Marr, D., and Poggio, T., 1977, Cooperative computation of stereo disparity. Science. 194: 283-287.

Nelson, J. I., 1975, Globality and stereoscopic fusion in binocular vision. J. Theor. Biol. 49: 1-88.

Pettigrew, J.D., Nikara, T., and Bishop, P.O., Binocular interaction on single units in cat striate cortex. Exp. Brain Res. 6: 391-410.

Pitts, W.H., and McCulloch, W.S., 1947, How we know universals, the perception of auditory and visual forms. Bull. Math. Biophys. 9: 127-147.

Rosenfeld, A., Hummel, R.A. and Zucker, S.W., 1976, Scene labelling by relaxation operations. IEEE Trans. Syst. Man Cybern. 6: 420-433.

Sperling, G., 1970, Binocular vision: a physical and a neural theory. Am. J. Psych. 83: 461-534.

Szentagothai, J. and Arbib, M. A., 1974, Conceptual Models of Neural Organization. NRP Bulletin vol. 12, no. 3: 310-479. (Also: The MIT Press, 1975.)

Tsukahara, N., 1972, The properties of the cerebello-pontine reverberating circuit, Brain Res. 40: 67-71.

Waltz, D.L., 1978, A parallel model for low-level vision. In: Computer Vision Systems (A.R. Hanson and E.M. Riseman, Eds.), New York: Academic, p.175-186.