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Rana Computatrix: an evolving model of visuo – coordination in frog and toad

M. A. Arbib
Department of Computer and Information Science,
University of Massachusetts at Amherst

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 behaviour, they make up for in the amenability of their behaviour and the underlying neural circuitry to experimental analysis. This paper presents three specific models of neural circuitry underlying visually-guided behaviour 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 behaviour 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 behaviour of the frog.

1. NEURAL SUBSTRATES FOR VISUALLY-GUIDED BEHAVIOUR

Lettvin, Maturana, McCulloch & Pitts (1959) initiated the behaviourally-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 modelling 'the style of the brain'. A general view of cooperative computation between neurons within a layer, and between layers within the brain is developed in Arbib (1981b); while the relation of 'maps as control surfaces' to the general study of perceptual structures and distributed motor control is given in Arbib (1981a). Our aim in the present paper is to exemplify these general principles in three specific models of cooperative computation in neural circuitry underlying viscomotor coordination in frog and toad.

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 behaviour will depend on far more than activity of a single class of retinal ganglion cells (Ewert 1976, and section 3 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 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 this paper, then, we first model local circuitry in the tectum (a 'tectal column') to explain certain facilitation effects in prey-catching behaviour; we then study a linear array of such columns to model certain data on size-dependence of prey-catching activity in toads; and, finally, we add PT-inhibition to such an array to model the behaviour 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.

2. FACILITATION OF PREY-CATCHING BEHAVIOUR

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 accords well with this facilitation effect (Fig. 1). This leads us to a model (Lara, Arbib, & 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.

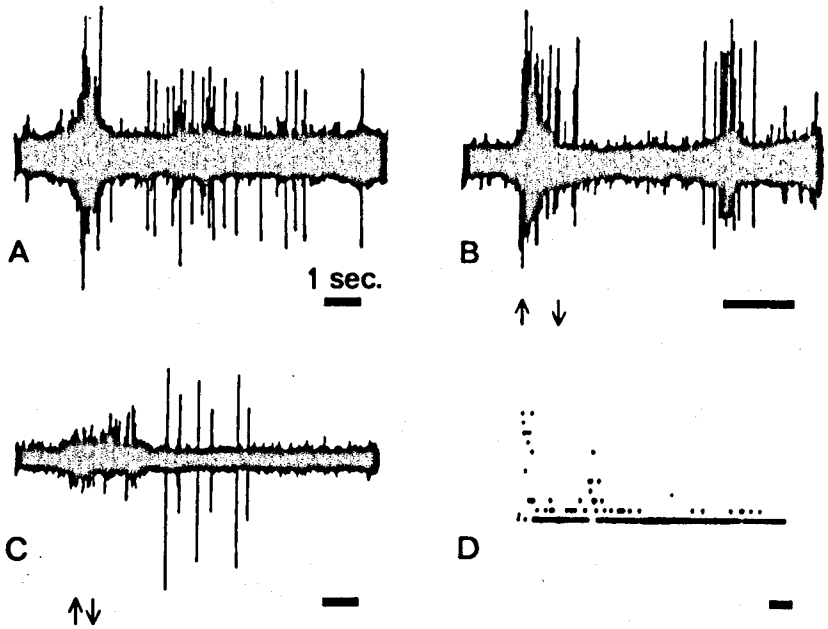


Fig. 1 – Physiological behaviour of cells related to prey catching facilitation. (A) Shows a brief class 2 burst followed by a delayed response of a tectal cell. (B) Shows the behaviour of a tectal cell responding to the presentation of the stimulus and design with a delay. (C) Shows a tectal neuron 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).

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 3 and 4.

The anatomical study of frog optic tectum by Székely & Lázár (1976) provides the basis for our model of the tectal column (Fig. 2). 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 the pyramidal cells as efferents; we shall ignore the tectal ganglion cells. 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, let alone the physiological parameters of their connections.

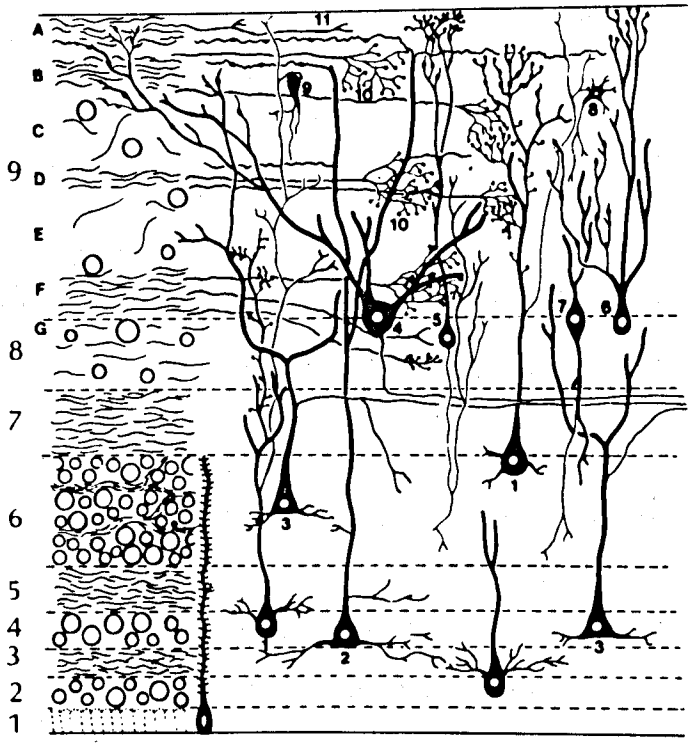


Fig. 2 – Diagramatic representation of the lamination and the representative types of neurons of the optic tectum. Numbers on the left indicate the different tectal layers. Numbered cell-types are as follows: (1) large pear-shaped neuron with dendritic appendages and ascending axon; (2) large pear-shaped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

The tectal column model (Fig. 3) comprises one pyramidal cell (PY) as sole output cell, three large pear-shaped cells (LP), two small pear-shaped cells (SP), and two stellate interneurons (SN), only one of which is shown in the figure. These numbers are based on the ratios of occurrence of these cells observed in frog tectum. 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 cells. LP axons return to the glomerulus, providing a positive feedback loop. A branch of LP axons also goes to the SN cells. 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, & Cromarty, to appear.)

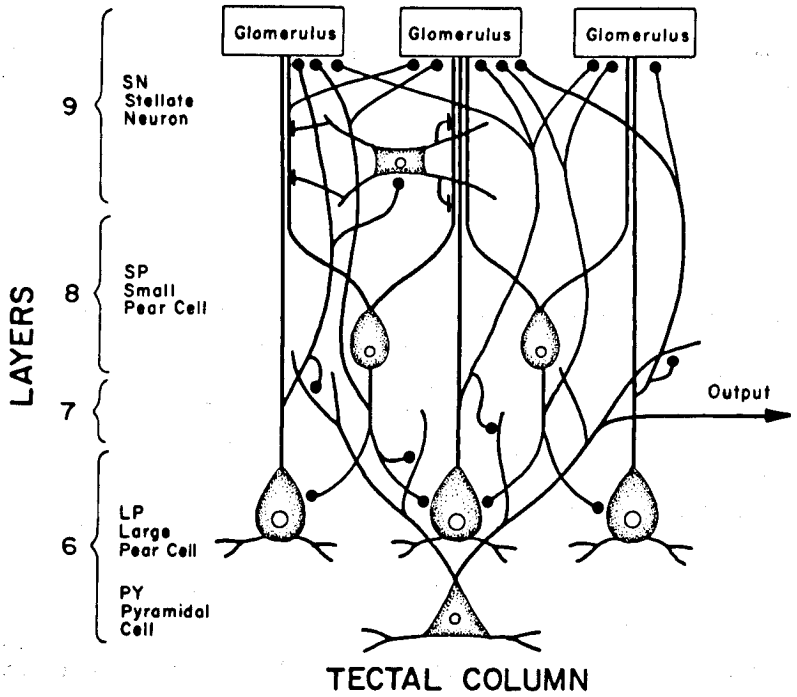


Fig. 3 – Neurons and synaptology of the model of the tectal column. The numbers at the left indicate the different tectal layers. The glomerulus is constituted by the LP and SP dendrites and recurrent axons as well as by optic and diencephalic terminals. The LP excites the PY, the SN, and the GL, and 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 neurones 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 neurone of the tectum.

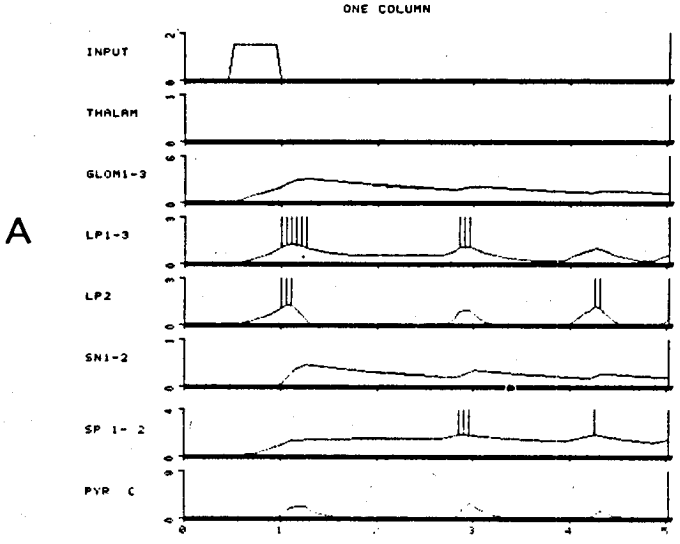
The role of SN in our tectum model is reminiscent of Purkinje inhibition of the positive feedback between cerebellar nuclei and reticular nuclei, a basic component of our group's model of cerebellar modulation of motor synergies (Boylls 1974, Szentagothai & Arbib 1974, Chapter V). Tsukahara (1972) found that reverberation was indeed established in this 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 cells which also send their axons back to the glomerulus. The SP cells also excite the LP cell to recruit the activity of the column. The PY cell is excited by both SP cells and LP cells. 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 behaviour. 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 cells have a longer time constant, and are 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. 4A, B). There is, as in the experimental data, a critical presentation length below which there is no pyramidal response. Input activity activities 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 reverberation will die out without activating the SP cells enough for their activity to combine with the LP activty 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. 4C) 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.



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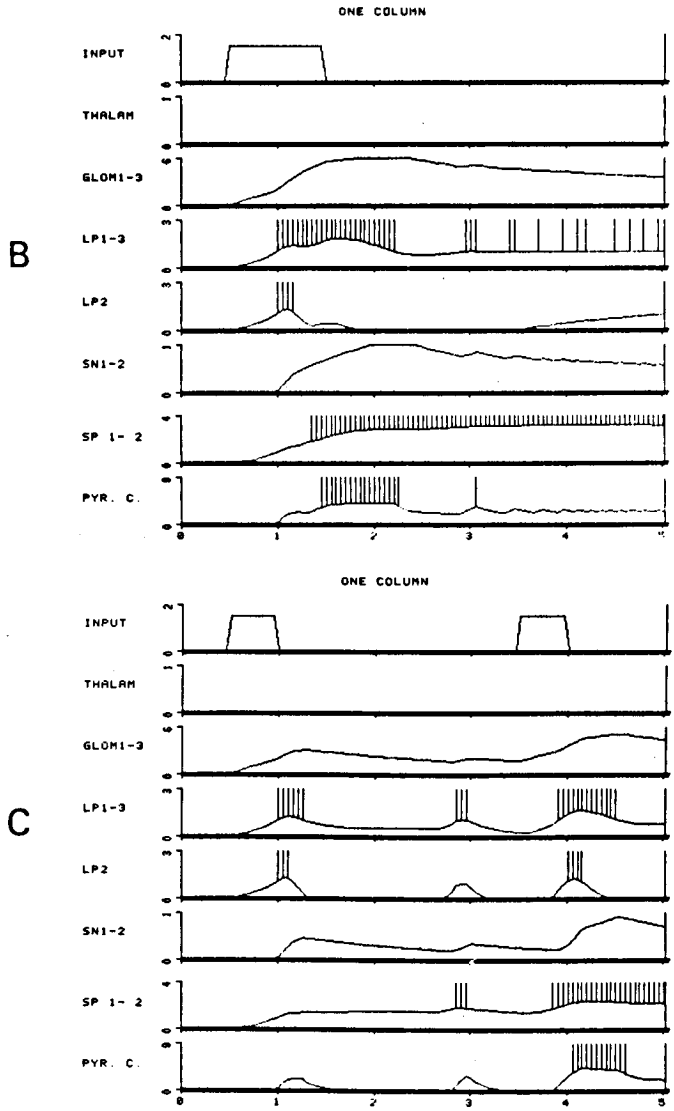

Fig. 4 – (A) Computer simulation of tectal cells response when a brief stimulus is presented. The onset of the stimulus produces a long-lasting depolarization in the glomerulus which then fires the large-pear shaped cell (LP). This neurone in turn sends recurrent axons to the glomerulus and the stellate 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 and of LP and SN neurones to give a delayed short response. (B) If in the above situation we present a stimulus of longer duration, the pyramidal neurone now 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.)

3. A SIMPLE MODEL OF PATTERN RECOGNITION IN THE TOAD

The facilitation model was 'local' in that it analysed activity in a small patch of tectum rather than activity distributed across entire brain regions. We now outline Ewert's study of pattern recognition in the toad, (see Ewert 1976 for a review) analysing 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. Future research will explore constraints on a two-dimensional array of such columns required 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 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. For $d = 2$, the three stimuli were, of course, the same. They elicited an orienting activity of 10 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 behavioural 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 tectical 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 – mono-

tonically 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 behaviour.)

On this basis, Ewert postulated a simple model: A filter in PT responds best to an antiworm stimulus; a tectal type 1 cell responds as a filter tuned to a worm stimulus; and a tectum type 2 cell is excited by the tectal type 1 cell and inhibited by a PT-cell. Thus the type 2 cell responds with increased activity to increasing d from a worm stimulus; with decreased activity to increasing d for an antiworm stimulus; and with some trade-off (dependent upon the actual parameters of the filters and the connectivity) for a square. Ewert & 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 & Lara (to appear) have studied a one-dimensional array of tectal columns (without PT interaction) to provide a model of spatio-temporal 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

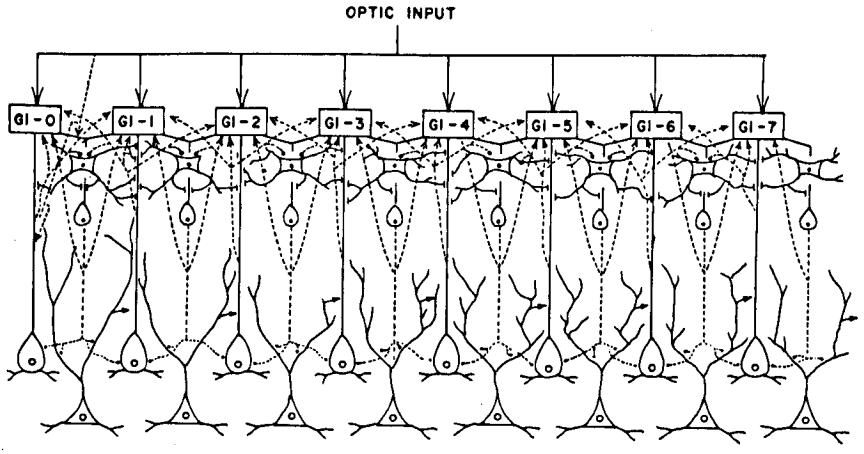

Fig. 5 – A one-dimensional array of tectal columns. Each column is constituted by one GL (glomerulus), one LP (large pear-shaped) cell, one SP (small pear-shaped) neuron, one SN (stellate neuron), and one PY (pyramidal cell). The afferents are the optic fibres that arrive at the GL, LP, SP, and PY cells, 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 neighbouring glomeruli. The SN neurons are activated by the LP cells and they inhibit LP and SP neurons of their own as well as neighbouring columns. The SP receive excitation from GL and are inhibited by SN; finally PY receives afferents from the retina, the LP and SP neurons.

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 analysing how activity in a population of tectal columns can yield orientation in a particular direction. Thus, rather than analysing activity in a single column, Arbib & Lara (to appear) study the evolution of a waveform of activity in a one-dimensional array of columns (Fig. 5). The columns of this array are somewhat simpler than that of Fig. 3, having only one neurone of each cell type. We show in Figs. 6, 7, and 8 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 & Lara also report a number of other computational experiments. The elaboration of this model to a two-dimensional array of colums will, in our future research, be integrated with our model (section 4) of tectal-pretectal interactions in prey-selection to yield a model that should be 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.

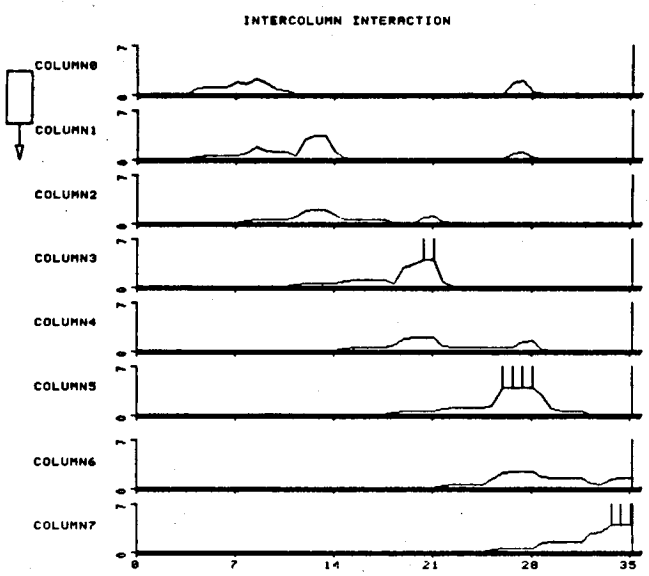

Fig. 6 – Figs. 6, 7, and 8 show the results of a computer simulation of tectal response to a moving stimulus of different sizes. The graphs show the behaviour of the 8 PY neurons of the tectum to a moving stimulus. Notice that in this case an alternate response is given in columns 3, 5 and 7 when the stimulus size only covers one glomerulus.

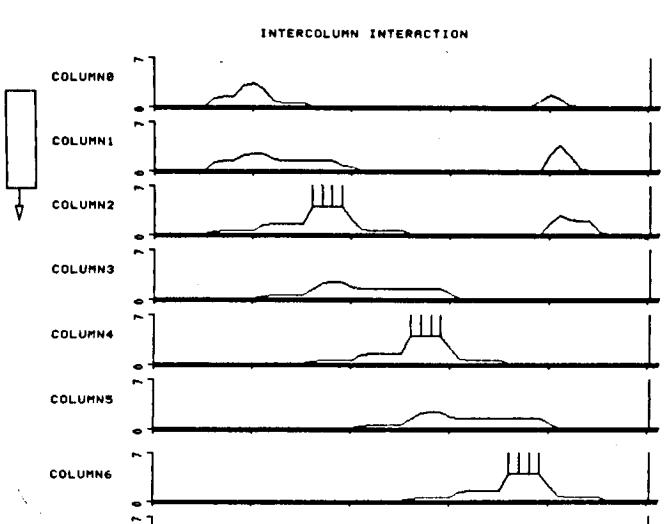

Fig. 7 – Here the stimulus covers 2 glomeruli simultaneously. The results show that the strength of activation increases when the size of the object is elongated. The latency of response is also faster.

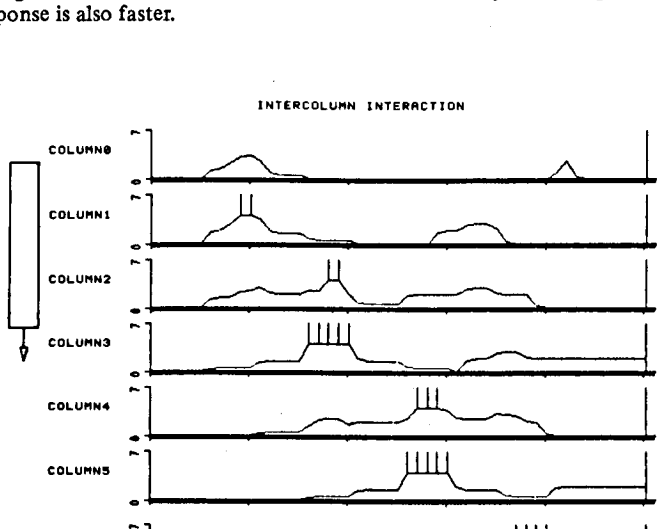

Fig. 8 – In this figure the stimulus simultaneously covers 3 GL. It can be seen that the latency of response is shorter and the total activity is greater than in Figs. 6 and 7. Notice that all columns fire with this stimulus.

4. A MODEL OF PREY-SELECTION

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. He 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. The model is a refinement of one developed by Didday (1970, 1976) while working with me at Stanford, 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 to provide yet a third stage in the evolution of *Rana computatrix*. Given that a pair of stimuli may fail to elicit a response even when either stimulus alone would have produced one, we conceptualized prey-selection in terms of 'competition' between the neural representations of the stimuli. (We cannot here be content with a simple program to search for the maximum from a list of stimulus strengths; our task in Brain Theory is to distribute prey-selection over a neural network conforming to the available constraints from anatomy and neurophysiology.)

The Didday model started from two postulates: (i) There is available as input a retinotopic array of activity which encodes the loci of 'food-like' movements in the environment. (In this simple exposition, we ignore the possible role of visual accommodation and stereopsis in providing a third dimension to this representation.) This 'foodness layer' corresponds to the glomerular input to a spatial array of the tectal columns modelled in section 2. (ii) The output of the tectum is again a retinotopic array, and sufficient activity in this array will, when played down through efferent structures, cause the animal to snap at the spatial locus corresponding to the 'centre of gravity' of activity in this output array. This output layer was referred to as the 'relative foodness layer', since high activity there should, in general, represent relatively high activity in the foodness layer. Thus, this activity corresponds to the pyramidal activity (PY) in our spatial array of tectal columns. We seek to explain, then, how competition amongst multiple peaks in the foodness array (input to the glomeruli) can lead to the suppression of all but one of them in the output array (PY activity), with consequent snapping at but one of the 'prey'.

The present model (Lara & Arbib, in press) interconnects a one-dimensional array of simplified tectal columns with a layer of cells called S-cells, in retinotopic correspondence with the columns, which represent cells of the pretectum-thalamus (Fig. 9). (In the 1970 model, the S-cells were identified with the sameness cells reported in the tectum by Lettvin, Maturana *et al.*) Each S-cell is excited by activity in the relative foodness layer, save for a blind spot centred 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 & Arbib (in press), 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 conse-

quences of choosing different sites for pretectal inhibition of columnar activity. The reader is referred to their paper for details.

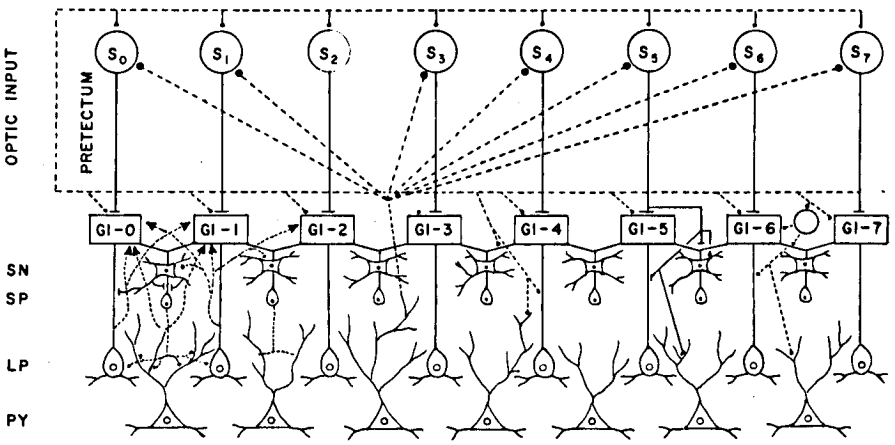

Fig. 9 – Architecture of the model for the interaction between tectum and pretectum in prey selection. Each column receives the afferents from one S (sameness) neuron; each PY (pyramidal) neuron excites all pretectal cells except the one whose blind spot is in its receptive field. The NE (newness) neurons arrive at the same site as the corresponding optic fibres.

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 via the S-cells. The model thus postulates 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. 10). However, there were cases in which the mutual supression between two peaks sufficed to hold each below a level sufficient to release behaviour (Fig. 11).

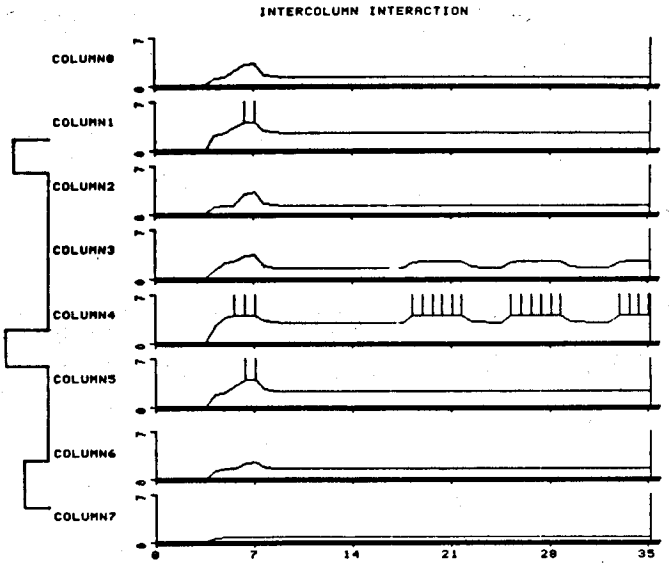

Fig. 10 – Computer simulation of the behaviour of prey selection to three stimuli of different intensities. Column 1 is excited by a stimulus of intensity 2; column 4 by one of intensity 3; and column 6 by one of value 1. After an initial brief response of columns 1, 4, and 5 the rebounding excitation converges to column 4.

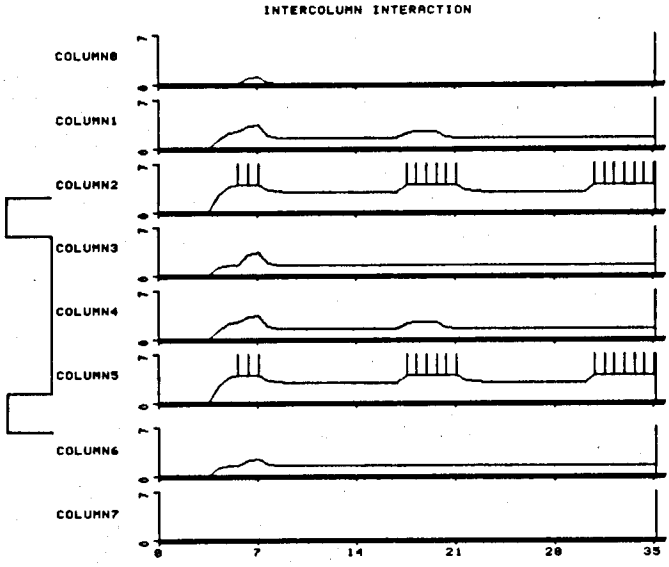

Fig. 11 – Computer simulation of the behaviour of PY neurons to 2 equally intense stimuli. The stimuli are presented in column 2 and 5. Notice that an alternation of excitation and inhibition is present without convergence to either of the stimuli.

5. CONCLUSIONS

We have exhibited an evolutionary sequence of models – tectal column, one-dimensional array of columns; array with pretectal inhibition – which explains an increasingly broad range of behavioural data on visuomotor coordination in frog 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' presented here 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 at present available. To carry out simulations, we make choices, we form explicit hypotheses (whose details are spelt out in our papers cited above) 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 current avenue for further development of *Rana computatrix*.

The model has nothing to say about the control of avoidance behaviour, nor does the basic version described here address more than a few of the prey-predator discrimination phenomena discussed in section 3. A two-dimensional array of columns will allow us to study the full range of these phenomena.

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. 12). 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 $A \leqslant B$ 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*.

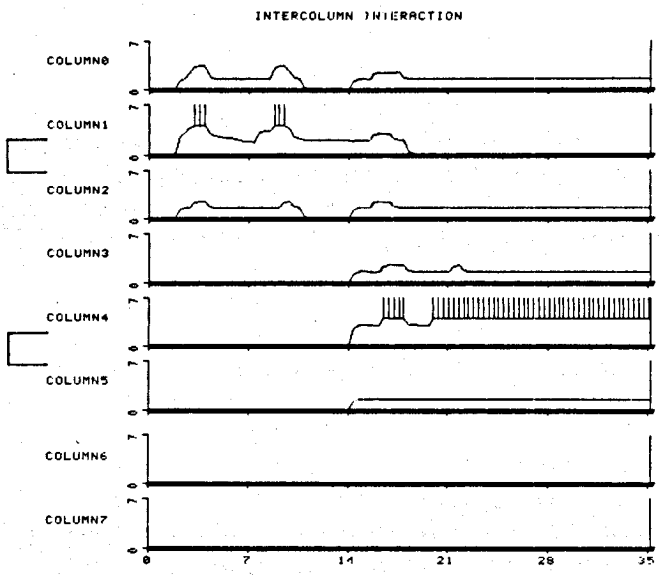

Fig. 12 – Computer simulation when the model is modified to include habituation effects on PY activity. We first present a stimulus in column 1. After a period of rest, we present 2 equally intense stimuli in column 1 and 4. The response converges to PY activity in column 4, because the pathway of column 1 is habituated.

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