Neuronal networks working at multiple temporal scales as a basis for amphibia's prey-catching behavior[1]

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ABSTRACT

We analyze a model of neuronal mechanisms underlying amphibia's prey-catching behavior, integrating hypotheses generated within different areas of Neuroscience and studying how the efficacy of visual prey-like *dummies* to release toad's prey-catching actions depends on *parallel distributed processes* occurring at multiple levels of temporal abstraction. First, in the scale of 100's of *msecs*, changes in neuronal activity caused by the stimulus characteristics and its current spatial-temporal relationship with the toad, as well as nervous signals related to actions' expected consequences (e.g., mouth mechanoreceptors activation after a *snapping*); second, signals generated during learning events happening at a temporal scale of minutes to hours; third, signals related to the *course of actions*, within an undetermined time scale that may last for several hours; and fourth, signals generated by changes in motivational factors (e.g., hunger, daily and yearly cycles) occurring at a much slower time scale. In addition, we analyze how in this *knowledge representation*, the course of actions (*plan*) is *episodic, goal-oriented* and can be modulated by *learning*, or by changes in the *agent*'s *motivational state*. This modulation is the outcome of accommodating information of new situations (a non catchable prey-like stimulus) into the dynamics of underlying neuronal mechanisms, in order to change the way the toad (*agent)* normally responds to that *domain of interaction* (stop yielding prey-catching behaviors towards that specific stimulus), without affecting its performance when similar situations appear in its immediate surroundings (prey-catching behaviors to real prey remain unchanged).

Key words: Prey-catching Behavior, Reinforcement Learning, Stimulus Specific Habituation, Neuronal Networks, Behavior-based Robotics, Distributed Artificial Intelligence.

Running title: Multiple temporal scales in neural net models.

1.- *INTRODUCTION*

Knowledge representation at different levels of temporal abstraction, and its relationship to planing and learning are key issues in Distributed Artificial Intelligence, and in behavior-based robotics (Sutton and Barto, 1998; Beer *et al*, 1990). In particular when the aim is to build *agents* that could incorporate ("*learn*") into their control (*planing*) system different aspects ("*knowledge*") of the relationship they maintain with other *agents* or with an uncertain environment, i.e., their "*ecology*" (Arkin *et al*, 2000). Some work in the field of behavior-based robotics have shown the benefits of exploiting models of behavior proposed by neuroethologists (Beer *et al*, 1990; Mataric, 1990), or computational neuroscientists (Rosenblath and Payton, 1989; Arkin, 1989).

In our previous work (Arkin *et al*., 2000), we used Schema Theory as an interlingua to implement a model of the agent-environment interactions of the Praying Mantis (Cervantes-Pérez *et al*, 1993a) into the control system of a robotic hexapod, Hermes II, that displays visually guided behaviors, such as prey-acquisition, mating, predator-avoidance, obstacle-avoidance, and the "*chantlitaxia*" behavior.

Continuing our interplay of modeling biological data and robotic implementations, in this paper, we analyze a series of theoretical models of those neuronal mechanisms underlying the activation and modulation of the amphibia's prey-catching behavior. Our aim is to show how the amphibia's visuomotor system is a good example of a biologically implemented control system of a *goal-oriented* behavior, where *knowledge* of environmental characteristics, and of the course of actions needed to be executed to achieve the goal, is *represented* within the structural and functional complexity of neuronal networks carrying out processes that occur at multiple temporal scales. Of course, in a future stage these models would be used to continue our efforts to provide the basis for determining the means by which a robot could "*discover*" its own *ecological niche* within the world where it "*lives*" (McFarland, 1994).

Frogs and toads have been valuable biological models to the study of visomotor coordination, not only for getting an in-depth understanding of the neurobiological basis of behavior (Grüsser and Grüsser-Cornehls, 1976; Ewert, 1980; Ingle, 1982; Fite, 1973), but also for inspiring control schemas in mobile robotics (Arkin, 1989; Arkin *et al*., 2000). They live within complex and uncertain three dimensional environments rich in different modes of sensory signals, however, their behavior is mainly guided by visual information. They have a limited behavioral repertoire, and their survival chances depend on their ability for displaying, at every moment, the most appropriate action to cope with diverse environmental situations. Grüsser and Grüsser-Cornehls (1976) classified the amphibia's visually guided behaviors in seven categories: a) orienting, in general; b) prey-catching; c) avoidance and hiding; d) eye responses (e.g., accommodation and ocular movements); e) vegetative responses of short latency (e.g., breathing and heart beating); f) vegetative responses of long latency (e.g., circadian rhythms); and g) changes on skin color to changes in illumination, or in the background.

Among these behavioral patterns, prey-catching ("*food ingestion*") is essential for survival, and it can be studied under lab conditions at different levels of analysis. In ethological studies, using different types of *dummies*, it has been shown that these animals' response to *domains of interaction* representing potential prey is determined by different factors whose processing runs at multiple temporal scales:

a) *stimulus characteristics*, e.g., form, size and the spatial temporal relationship between the amphibia and the prey (Grüsser and Grüsser-Cornehls, 1976; Ewert, 1976; Ingle, 1982);

b) *previous experiences with the stimulus*, e.g., learning and conditioning, (Ewert and Ingle, 1971; Ewert and Kehl, 1978; Finkestädt, 1989); and

c) *motivational factors*, e.g., season of the year, food deprivation and maintenance within the lab prior to the experiments execution (Cervantes-Pérez, *et al*., 1993b; Ewert and Siefert, 1974; Ewert, 1980; Ingle, 1983).

In addition, anatomical studies have shown topographical maps among Retina, Optic Tectum and Pretectum, as well as great connectivity between these two structures receiving direct projection from retinal cells and the rest of the animals' brain (Grüsser-Cornhels, 1984; Fite and Scalia, 1976; Székely and Lázár, 1976); whereas electrophysiological analyses have shown great correlation between the levels of activity in tectal cell T5(2) and pretectal cell TH-3, and the probability that the current visual stimulus fit into the prey or predator categories, respectively (Ewert, 1984). Moreover, Ewert (1976, 1980) has shown that in animals with pretectal ablation, T5(2) cells activity also resembles the toads' overall behavior, responding indiscriminately to any moving object traversing their visual field.

Our own group has developed, based on the leaky integrator neuron model, a series of neuronal networks models to study how the retino-tectal-pretectal interactions might underlie some characteristics of prey-catching facilitation (Lara *et al*, 1982; Cervantes-Pérez and Arbib, 1990), prey-predator discrimination (Cervantes-Pérez *et al*, 1985), and prey-catching stimulus specific habituation (Lara and Arbib, 1985; Cervantes-Pérez *et al*, 1991).

In here, we analyze extended versions of our models, where we incorporate other hypotheses of how stimulus specific habituation and changes in motivational factors modulate the efficacy of visual prey *dummies* to elicit toads' prey-catching behavior. The aim is to seek an in-depth understanding of the computational properties associated to those neuronal mechanisms subserving toads' overall behavior, in terms of interacting dynamical systems processing events that occur at multiple temporal scales rather that in terms of neuronal plasticity. In addition, as a second goal, we set forth the basis for building an abstract model of an *agent* (toad) that stores information of certain domains of interaction (visual prey-like stimuli), coming from an uncertain environment (its *ecology*), within the dynamics of neuronal processes that occur at *multiple temporal scales*. Moreover, in this *knowledge representation scheme*, we study the course of actions (*plan*) as *episodic*, *goal-oriented* and plastic enough to be *modulated by learning*, or by changes in the agents' *motivational state*. Finally, we also aim to establish a link between the neuroethological studies of toad's visuomotor coordination and the Theory of Reinforcement Learning, seeking to gain access to the mathematical framework associated to the Markov Decision Processes to better analyze the temporally extended actions (planing) embedded in the toad' prey-catching ethogram.

2.- *ACTIVATION OF PREY-CATCHING MOTOR ACTIONS: A FAST TEMPORAL SCALE*

In the wild, at the twilight time of the day toads begin to *explore* the immediate surrounding in search of potential prey; thus, prey-catching can be looked at as a *goal-oriented* behavior, where the goal is *being fed*. These animals present their whole prey-catching repertoire under laboratory conditions; that is, when a prey-like stimulus is presented in the toad's visual field, they display one of the following prey-catching actions:

- *Orienting*.- If the stimulus is located at the lateral visual field, the toad orients towards the stimulus, and brings it into the binocular part of its visual field.

- *Approaching*.- When the stimulus rests in the binocular visual field, but a little bit too far to be caught, the toad moves forward trying to bring it into reaching distance.

- *Following*.- If the stimulus is in the frontal visual field but getting away, the toad moves in the same direction as the stimulus, trying to maintain it within reaching distance.

- *Binocular Fixation*.- Once the stimulus is within the *snapping zone*, the toad carries out a binocular fixation process to better estimate its three-dimensional relative position.

- *Snapping (Attacking)*.- When the stimulus remains within reaching distance, and binocular fixation has taken place, the toad snaps at it, and brings it into its mouth.

- *Swallowing*.- If a prey is captured, and hence inside the toad's mouth, the corresponding mechanoreceptors get stimulated, signaling the completion of a sub-goal: *food has been ingested*.

This ethogram description is similar to that of Ewert's (1980), but it includes the *Following* motor action, and we left out the *wiping-mouth* response (see also Roche-King and Comer, 1996). It does not represent a fixed sequence of actions, rather, it is a repertoire flexible enough to allow the animals to properly interact with moving prey-like objects in a complex three-dimensional environment full of uncertainty. All toad's prey-catching responses are episodic, occur in a time-scale of *hundreds of milliseconds*, and their outcome generates new situations with high probability of eliciting another prey-catching response; that is, they conform a sequence of temporally extended actions that leads towards the completion of the overall goal of *being fed*.

In Cervantes-Pérez *et al.* (1985), we developed a neural model of the interactions among elements of the Retina, the Optic Tectum and the Thalamic Pretectal region (Tectum and Pretectum henceforth), to study how these neural mechanisms could underlie, among other things, prey-recognition in amphibians. The main hypotheses embedded in the model were:

a) Topographical maps from the Retina to Tectum and Pretectum, and from Pretectum to Tectum. Retinal ganglion cells project retinotopically to Tectum (R2, R3 and R4), and to Pretectum (R3 and R4) (Witpaard and Keurs, 1975; Fite and Scalia, 1976; Székely and Lázár, 1976, Ewert, 1976; Grüsser and Grüsser-Cornehls, 1976), whereas pretectal cells project, also retinotopically, to Tectum (Fite and Scalia, 1976; Ewert, 1980, Ingle, 1982).

b) The Optic Tectum (principal visual center in the amphibian's brain) is organized in columns, comprised by different types of processing elements (see Figure 1): the Large

Pear-shaped cell (LP), the Small Pear-shaped cell (SP), the Stellate neuron (SN), the Pyramidal cell (PY), the only one having projecting axons outside the tectal circuit, and, finally, the glomerulus (GL), representing the convergence in the superficial tectal layers of axon terminals coming from the Retina and other brain structures, and dendrite arborizations of tectal cells (Székely and Lázár, 1976; Schwippert *et al*, 1995). GL receives signals from ganglion cells R2, and activates LP and SP cells, which, in turn, receive inputs from ganglion cells R2, and send axons back to GL forming positive feedback loops that tend to recruit tectal activity when a prey-like stimulus is present in the animal's visual field. LP also excites the SN cell, postulated to be the only inhibitory tectal cell (Székely and Lázár, 1976), which forms negative feedback loops with the LP and SP to keep tectal activation in control, specially when the stimulus disappears from the animal's visual field. Finally, the efferent PY cell integrates the overall tectal activity with the inputs it receives from ganglion cells R2, R3, and R4. PY cell is equivalent to the T5(2) cells of Ewert's (1980), whose level of activation correlates quite well with the probability of a toad displaying a prey-catching response towards a visual stimulus in its visual field.

c) Prey-recognition is due to tectal integration of retinal excitation and pretectal inhibition. Animals with pretectal ablations loose their capability of avoiding predators, attacking any stimulus that moves into its visual field. Even the hand of the researcher that feeds them becomes a potential prey (Ewert, 1980). Thus, it has been postulated that pretectal cells (TP in Figure 1) modulates tectal activation through an inhibitory effect upon LP, SP and PY cells, specially when the animal is confronted with predator-like stimuli. According to data from Schwippert *et al.* (1995), about superficial tectal layers receiving a direct projection from pretectum, in here we also consider a pretectal inhibition upon GL. This interaction is the basis for prey-predator discrimination; that is, stimuli that produce low tectal activation with high pretectal responses signal "*danger*" (predators), whereas the ones producing the opposite effect signal "*food*" (prey) (Ewert, 1976, 1980, 1984; Ingle, 1973, 1980).

PLACE FIGURE 1 HERE

In modeling the neural mechanisms underlying these phenomena we considered the *weighted spatial-temporal summation* property of a neuron, and its nonlinear capability of mapping changes in its membrane potential to the corresponding changes in its firing rate. We used the *leaky-integrator* model for the dynamics of the membrane potential *m(t)*:

$$\tau \dot{m}(t) = -m(t) + \sum_{i=1}^{N_E} v_i x_i - \sum_{j=1}^{N_I} w_j y_j + M_0$$

where,

τ – membrane time constant;
x_i and v_j – excitatory inputs and weights, respectively;
y_i and w_j – inhibitory inputs and weights, respectively; and
M_0 – resting potential.

The cells' firing rate were related to their membrane potentials through a nonlinear function of either *threshold*, *linear*, or *saturation* form:

$$f(m(t),\theta) = \begin{cases} 1 & if\ \ m(t) \geq \theta \\ 0 & if\ \ else \end{cases}$$

$$h(m(t),\theta;\beta) = \begin{cases} \beta m(t) & if\ \ m(t) \geq \theta \\ 0 & if\ \ else \end{cases}$$

$$s(m(t),\theta_0;\theta_1) = \begin{cases} \theta_1 - \theta_0 & if\ \ m(t) \geq \theta_1 \\ m(t) - \theta_0 & if\ \ \theta_0 \leq m(t) < \theta_1 \\ 0 & if\ \ m(t) < \theta_0 \end{cases}$$

Where, β, θ, θ_0, θ_1 are threshold parameters.

A neural network, based on the *leaky integrator model*, is represented by a set of first order differential equations with non-linearities. Thus, taking into account the connectivity shown in Figure 1, the model of the Retino-Tectal-Pretectal (RTP) Column is:

$$\tau_{gl}\,\dot{gl}(t) = -gl(t) + w_{r2\bullet gl}R2 + w_{sp\bullet gl}f(sp) + w_{lp\bullet gl}f(lp) - w_{tp\bullet gl}f(tp)$$

$$\tau_{lp}\,\dot{lp}(t) = -lp(t) + w_{r2\bullet lp}R2 + w_{sp\bullet lp}f(sp) + w_{gl\bullet lp}h(gl) - w_{sn\bullet lp}h(sn) - w_{tp\bullet lp}h(tp)$$

$$\tau_{sp}\,\dot{sp}(t) = -sp(t) + w_{r2\bullet sp}R2 + w_{gl\bullet sp}h(gl) - w_{sn\bullet sp}h(sn) - w_{tp\bullet sp}h(tp)$$

$$\tau_{sn}\,\dot{sn}(t) = -sn(t) + w_{lp\bullet sn}f(lp)$$

$$\tau_{py}\,\dot{py}(t) = -py(t) + w_{r2\bullet py}R2 + w_{r3\bullet py}R3 + w_{r4\bullet py}R4 + w_{lp\bullet py}f(lp) + w_{sp\bullet py}f(sp) - w_{tp\bullet py}h(tp)$$

$$\tau_{tp}\,\dot{tp}(t) = -tp(t) + w_{r3\bullet tp}R3 + w_{r4\bullet tp}R4 + H(t)$$

In Figure 2, we show a computer simulation of the dynamics of the RTP neural circuit when a prey-like stimulus is presented in the corresponding visual field. GL is the first element in increasing its level of activity (it is modeled as a functional unit with zero threshold), followed by LP an SP, being LP the one that first reaches membrane potentials above threshold. GL level of activity is recruited every time LP or SP get activated, through the positive feedback loops. Once LP output gets activated, SN membrane potential begins to increase, and when it goes above threshold its inhibitory effect upon LP and SP diminishes the overall tectal activity. When LP and SP cells are active, and the stimulus is still in the visual field, PY membrane potential goes above threshold, signaling that the visual stimulus fits the prey-category. Because the *dummy* is a prey-like stimulus, the pretectal TP cell shows a low level of activity, which will be increased in habituated animals through *H(t)* (see below).

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Having a model expressed as a set of first order nonlinear differential equations allowed us, in Cervantes-Pérez and Arbib (1990), to conduct stability and parameter dependency analyses that, in combination with computer simulations, served to identify a set of parametric

conditions under which the retino-tectal-pretectal interactions might underlie Prey-Catching Facilitation. A behavioral phenomenon studied by Ingle (1973, 1975), where frogs were stimulated once with a small visual object during a sub-threshold duration of 0.3 seconds (time period during which the stimulus did not produce a frog's prey-catching response), followed by a resting period of 3.2 seconds, and a second presentation of the *dummy,* where the frogs displayed an attack within the sub-threshold duration period.

Assuming a resting state when no stimulus is present (all tectal cells membrane potential at their resting level) and neuroethological correlates (Ingle, 1973, 1975; Ewert, 1976, 1980), we identified a set of relations among tectal neural parameters under which a tectal column model would follow an appropriate trajectory (state sequence), in order to underlie prey-catching facilitation. In Figure 3, we show how a sub-threshold presentation of a prey-like stimuli starts a process of cooperation/competition between positive (i.e., R2→GL↔LP; R2→GL↔SP) and negative (i.e., R2→LP↔SN; R2→SP→LP→SN→SP) feedback loops, and generates an LP decaying oscillation that, in turn, produces a GL and SP residual activity (a short-term memory process) that is responsible for facilitating PY response during the second stimulus presentation. Here, we follow Ewert's (1980) results of PY response being highly correlated with the probability of a stimulus to yield a prey-catching response. It was also observed that when the excitatory effects of the positive feedback loops were increased, keeping the ones of negative feedback loops constant, all cells in the tectal column model could present: much longer oscillations (see Figure 4a), or continuous responses of all cells, which means that prey-catching facilitation for sub-threshold stimuli could even occur for longer time periods between the first and second stimulus presentation. When these oscillations are much stronger, there will be a kind of "*epileptic*" response when the PY cell gets activated in the absence of a visual stimulus (see Figure 4b). On the contrary, if negative feedback loops are dominant, prey-catching facilitation does not occur and both stimulus presentations are processed as independent events (see Figure 5).

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Thus, based on these analysis, we may conclude that information about prey and predators, in the amphibians' visuomotor system, is distributed among different structures (e.g., Retina, Tectum, and Pretectum), and that its processing is carried out in parallel. Also, that the *knowledge* used during prey and predator recognition is *represented* within these neuronal circuits in a *spatial-temporal* way; that is, to signal whether the visual stimulus fits into the prey or predator category, it is important not only what neurons got activated, but also what was the temporal course of their activation. Finally, It should be pointed out that that PY cells response, related to the activation of a prey-catching action, is generated in a temporal scale of 100's of *milliseconds* (see Figure 2).

3.- *LEARNING DYNAMICS: TIME-VARYING TEMPORAL SCALES*

Toads are capable of modifying the way they interact with some of the stimuli that appear in the *uncertain environment* where they live. As a good example of that, we have the stimulus specific prey-catching habituation, one of the simplest form of learning where the tendency to respond to a specific prey-like stimulus declines, and eventually disappears, when the stimulus remains in the visual field despite the toad's efforts to catch it (Ewert and Kehl, 1978; Cervantes-Pérez *et al*., 1991). That is, the probability, $\pi(s',a')$, assigned to prey-catching action, *a'*, in an environmental situation, *s'*, is not a time-invariant mapping, but rather it has its own dynamics. It depends on the stimulus characteristics and the spatial-temporal relationship between toad and prey, and changes according to information processes working at multiple temporal scales (e.g., the efficacy of a specific stimulus to yield a prey-catching response decreases as the habituation experiment progresses). This learning process has adaptive significance in that animals should not respond forever to non-catchable prey; rather, they should be able to learn to "*abandon*" a given task when their efforts do not produce the proper result ("*reward*"), so they would be able to properly interact with other key-stimuli that may appear in the environment, specially with those signaling food or danger.

In Cervantes-Pérez *et al*.(1991), we presented data from a series of behavioral experiments, conducted with a group of toads *Bufo marinus horribilis* under a setup that allowed the animals to display their whole repertoire when interacting with a *worm-like* dummy, a horizontal black rectangle, in a white background, with its longest axis parallel to the direction of motion). Toads were placed within a plastic cage, in front of a TV set where the image of the stimulus was played back. In Figure 6 we show the results obtained with six animals, trained with a *worm-like* stimulus of $1.5 \times 0.4 cm$, moving at $4.1 cm/sec$, and at a height of $2 cm$ from the substrate where toads were located. The main hypotheses derived from results obtained under this paradigm were:

a) Prey-catching response to visual prey-like objects decreases with repetitive stimulation, until it eventually disappears. There are differences in the temporal evolution of the response displayed by individual toads; however, as the experiment progresses, all animals showed, first, quantitative changes (prey-catching response frequency decreases), and, second, at the end of the training a qualitative change takes place (toads stop attacking the stimulus).

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b) Prey-catching habituation is stimulus specific. Once toads had been habituated to a given parametric configuration (i.e., *worm-like* stimulus of 1.5 x 0.4 *cm*, moving at 4.1 *cm/sec*), and after a 40*min* resting period, a change in the horizontal dimension, from 1.5*cm* to 5.5*cm*, was enough for leading the toad to elicit prey-catching actions towards the new configuration of the stimulus (see Figure 7).

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c) Spontaneous recovery takes longer than the acquisition of habituation (see Table 1). All toads trained under this paradigm took different times to habituate, ranging from few minutes to almost 2 hours (see Figure 6), but it took them days to "*forget*" the training. Some animals were retrained after the 11th day of the first training event. The first four

animals, retrained after 11, 13, 20, and 45 days respectively, did not respond to the same stimulus configuration used during the habituation process; whereas another one, re-trained after 49 days, responded during 28*min* with a maximum frequency of 22 *responses/min* (for a detailed description refer to Cervantes-Pérez *et al.*(1991)). In Ewert and Kehl's (1978) experiments on the habituation of the orienting behavior, toads habituated in 5 minutes, and recovered in half an hour.

PLACE TABLE 1 HERE

To explore how habituation might be implemented in the amphibia's visuomotor system, in this section the RTP model is extended to include an Habituation Column (HC) model (see Figure 8).

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We start by taking into account Lara and Arbib's (1985) habituation model, where they explained prey-catching habituation, following Sokolov's (1960) original proposition, in terms of building a model of the activity produced by a stimulus presentation that, when it becomes "*familiar*" due to repetitive exposure, acts to suppress its efficacy to elicit prey-catching responses. In addition to their MO unit, we postulate, and here we hypothesized, that the beginning of this modeling process is triggered by a Failure Unit (FU) that gets activated when the expected consequences associated to the response generated by the RTP column at time, *t*, do not appear in the situation detected at, *t+1*, (e.g., mouth mechanoreceptors activation after a *snapping* response has been elicited). In case the expected consequences occur, the FU controls the time period in which the model of the stimulus is "*forgotten*". The FU could be modeled as a neuronal circuit storing all patterns resulting from combining environmental situations and the actions they could possibly activate, as well as the corresponding expected consequences of executing such actions, according to the animals' ethogram description, and used them as a content addressable memory. However, in here, it is enough to model it as a logic unit:

$$z = \begin{cases} 1 & if \ \ py > 0 \ \& \ NC > 0 \\ 0 & if \qquad otherwise \end{cases}$$

where, *NC* means "*No expected consequence occurred*". Thus, the MO unit dynamics is represented as:

$$\tau_m \dot{m}(t) = [\alpha_m (1 - z) + z](-m(t) + m_0 + zr(t))$$

and the output,

$$f_M(m(t); \theta_m) = \begin{cases} m(t) & if \qquad m(t) \geq \theta_m \\ 0 & otherwise \end{cases}$$

where,

z — FU output,

θ_m — MO unit threshold,

$r(t)$ — input from the retina, here it is considered as constant for a given stimulus, which when multiplied by z forms an activity-gated input, instead of a direct input,

τ_m — acquisition time constant, and
α_m — a variable that regulates the recovery time constant.

FU is active (z=1), when a prey-catching response was executed and its expected consequences failed to occur. Thus, the modeling process starts and, $m(t)\rightarrow r+m_0$ as $t\rightarrow\infty$, with a time constant τ_m. Otherwise, in case everything goes well (z=0), $m(t)\rightarrow m_0$ as $t\rightarrow\infty$, with a bigger time constant, $\frac{\tau_m}{\alpha_m}$, that accounts for a slower evolution of $m(t)$ to its resting level (i.e., "*forgetting*" the model of the stimulus takes longer than its acquisition). This mechanism might account for some aspects of stimulus specific habituation in successive training sessions separated by intermediate recovery intervals, described by Ewert (1984) and modeled in a similar way by Wang (1993). That is, during subsequent trials toads will take less time to habituate, because the MO level of activity will be closer to the retinal signal level, thus, the build up phase of habituation will start sooner.

The habituation process does not initiate with a single failure trial, rather it requires that the output of the MO unit resembles the retinal signal yielded by the stimulus presentation. So, it is required that the toad attempts several times to catch the stimulus without success, before the building up of habituation begins. This task is accomplished by the Comparison Unit (CO), which is also modeled as a logic unit:

$$c=\begin{cases}0 & \text{if } m(t)=r(t)\\ 1 & \text{if otherwise}\end{cases}$$

That is, the CO is maintained activated until the MO output is equal to the signal coming from the retina, and the dynamics of the habituation unit is modeled similarly to the MO unit:

$$\tau_h\dot{h}(t)=[\alpha_h(1-c)+c](-h(t)+h_0+ch_{\max})$$

and,

$$f_H(h(t),\theta_h)=\begin{cases}h(t) & \text{if } h(t)\geq\theta_h\\ 0 & \text{otherwise}\end{cases}$$

where,
c — CO output,
θ_h — habituation unit threshold,
$h(t)$ — habituation intensity,
τ_h — acquisition time constant, and
α_h — a variable that regulates the rate of *spontaneous recovery*.

When CO becomes inactive ($c=0$), the output of MO, $m(t)$, matches the retinal signals, $r(t)$, it means that a prey-like stimulus has been in the animal's visual field for certain time, despite its efforts to catch it. Thus, the building up of prey-catching habituation starts: $h(t)\rightarrow h_{max}$,, as $t\rightarrow\infty$, with a time constant τ_h. Here, h_{max} is a value big enough to ensure that the level of pretectal inhibition acting upon tectum highly decrease the probability of the stimulus fitting the prey-category. In this way, the release of any prey-catching response will be stopped. Otherwise, with CO active ($c=1$), meaning that a new potential prey has arrived into the

animal's visual field, *spontaneous recovery* begins to take place; that is, the habituation unit moves towards its resting potential, $h(t) \rightarrow h_0$, as $t \rightarrow \infty$, with a time constant, $\frac{\tau_h}{\alpha_h}$, that accounts for a slower evolution.

Finally, because the habituation process acts upon the pretectum only after the model created by MO matches the retinal input (i.e., $c=0$), the output unit, H, of the habituation column model is represented as a logic unit:

$$H = \begin{cases} h(t) & if \quad c = 0 \\ 0 & if \quad c = 1 \end{cases}$$

In Figure 9 we show a computer simulation of a stimulus specific habituation process. Each stimulus presentation activates the Failure Unit modifying the MO unit membrane potential. During this period CO remains active, $c=1$, keeping the habituation unit membrane potential at its resting level, h_0. Once the MO unit reaches the same activation level as the signal coming from the retina, the CO output becomes inactive, $c=0$, the habituation unit membrane potential starts increasing towards its maximum level, h_{max}, and every time there is a stimulus presentation the H unit sends an excitation to the pretectal TP neuron, increasing its inhibitory effect upon tectal neurons to reduce the probablity of the stimulus fitting the prey-category.

PLACE FIGURE 9 HERE

To show the dynamics of the habituation tectal column when after an habituation process we present a different prey-like stimulus in the animal's visual field, in Figure 10 we show a computer simulation of such an experiment. A non-catchable stimulus is presented (the dynamics is similar to that described for Figure 9) until the habituation unit activity reaches its maximum level, h_{max}, then, the stimulus is replaced by a stimulus that resembles a catchable prey. At this point, the Failure Unit stops responding, so the MO membrane potential starts going down to its resting level, although with a much slower time constant controlled by α_m. CO becomes active once again and inhibits the H unit, thus, the level of activation in the cells of RTP column model would be closely related to the stimulus efficacy to yield prey-catching responses. Finally, the habituation unit membrane potential, $h(t)$, starts also decreasing towards its resting potential, h_0, in a similar fashion as the MO unit; that is, with a much slower time constant controlled by α_h. It must be pointed out that the dynamics of those cells in the RTP model at the begining of the habituation experiment would be similar to that described in Figure 2; whereas, once the habituation unit activation makes pretectal inhibition strong enough to stop tectal neurons from getting activated by the visual stimulus, these elements dynamics would be similar to that described in Figure 14 (see next section).

PLACE FIGURE 10 HERE

Prey-catching stimulus specific habituation is a *learning from interactions* process whose characteristics seem to be universal, they appear in studies:

a) *with different species*, including humans, e.g., habituation in the turning behavior of nematodes (Rankin and Broster, 1992), habituation of a defensive withdrawal reflex in Aplysia (Carew *et al*., 1972), escape reactions habituation in ground living birds

to the sight of other birds flying above (Tingbergen, 1951), rats short-term and long-term startle habituation to different tones (Leaton, 1976), and habituation in baby's orienting response to a face (MacKenzie, Tootell, and Davis, 1980);

b) *at different levels of analysis*, e.g., behavioral (see a)), lesion experiments of brain structures (Ewert, 1980; Finkestäd, 1989), and at the cellular level (Kandel, 1976; Byrne, 1982); and

c) *experimental* (see a) and b)), *and theoretical approaches*, e.g., Lara and Arbib (1985) posed a neuronal model of amphibia's prey-catching orienting response; Wang (1993) developed a model of the possible neuronal mechanisms subserving short-term and long-term habituation in toads prey-catching orienting response; Staddon and Higa (1996) developed a two-unit cascade-integrator model to study how the interactions of two inhibitory processes, with different time scales, might be the basis for habituation, recovery, rate-sensitivity and the effects of variable inter-stimulus intervals in the nematode *Caernorhabditis elegans.*

Habituation is in itself a phenomenon that offers a rich framework, in behavioral complexity, experimental data and theoretical models, with phases that occur in different neuronal circuits working at multiple temporal scales: the acquisition phase takes from *minutes* to *hours*, whereas the spontaneous recovery may take from *minutes* to *days* (Leaton, 1976; Ewert, 1984; Cervantes-Pérez *et al*, 1991; Wang, 1993). It also must be pointed out that, in many cases, there seems to be a *time-varying dynamics* involved:

a) in Cervantes-Pérez *et al* (1991), it took different time periods for each animal to habituate, and to recover (see Figure 6 and Table 1);

b) toads trained under different scenarios required different times, e.g., under our paradigm toads took from minutes to hours to habituate, and days to recover; in Ewert and Kehl's (1978) experiments toads habituated in 5 minutes and recovered in half an hour; and

c) the number of trials also varies when using different type of stimuli, e.g., when a toad catches a bee, its sting at the toad's tongue is enough to prevent it from catching bees in the future (Ewert, 1980).

Thus, and here we hypothesized, it seems that the *expected*, and the *unexpected*, *consequences* of some behaviors (e.g., *snapping* in our case, and the bee sting in Ewert's case) might have a bigger effect on the building up of habituation than others (e.g., prey-catching orienting in Ewert and Kehl's experiments).

4.- *COURSE OF ACTIONS DEVELOPMENT: TEMPORALY EXTENDED ACTIONS*

When toads *search for food*, there seems to be a *plan* embedded in the prey-catching ethogram: "*explore the immediate surroundings until a prey appears in the visual field, then modify the spatial-temporal relationship with the stimulus so it gets and stays within the reaching zone, and catch it*", and the course of actions to fulfill this *plan* develops in a time-scale that involves the execution of many basic prey-catching actions, without specifying a particular time at which it should stop (that will depend on when the toad has caught enough prey).

In Figure 11 we show all processing elements involved in the *agent-environment* interaction during the elicitation of the amphibia's prey-catching behavior. The *agent* is the animal's visuomotor system (Retina, Habituation Column, HC, Pretectal-Tectal interactions, PTC, Motor Centers and Muscular System); whereas the *environment* is formed by the immediate surroundings, and those brain structures, not belonging to the visuomotor system, carrying out information processes whose outcome modulates the way in which amphibia respond to visual stimuli (failure of expected consequences, FU, and motivational state, MOT). The basis for this being that these processes can not be directly modified by the visuomotor system, but rather they are affected by changes in environmental conditions (e.g., seasonal cycles, lack of prey, etc.).

PLACE FIGURE 11 HERE

Considering that toads interact with visual prey at a discrete time-scale, $t = 0,1,2,\ldots$, at each step, the current environmental situation, $s_t \in S_{pc}$ (where, $S_{pc} \subset S$, the set of all situations related to prey-catching behavior being a subset of all possible environmental situations *S*), activates a motor action from the animal's repertoire, $a_t \in A_{pc}$, (with, $A_{pc} \subset A$, all actions in the prey-catching ethogram being a subset of the animal's global repertoire *A*), according to a *mapping*, π, where a given situation, s_t, might activate an action, a_t, with probability, $\pi(s_t, a_t)$. Toads not always perform when placed in a lab scenario, sometimes they just sit there and do nothing. Each action, a_t, is associated to an *expected consequence* that, if everything goes well, will be reflected in the environment's transition into, s_{t+1}. That is, in the case of prey-catching behavior: *orienting* brings the stimulus into the frontal part of the visual field; *following* keeps the stimulus at a giving distance; *approaching* reduces the distance between the toad and the prey; *binocular fixation* generates the estimation of depth; and *snapping* takes the prey into the toad's mouth stimulating the mechanoreceptors. In addition, some structures in the toad's brain (represented by FU in Figure 11), and here we hypothesized, are monitoring for these expected consequences to occur (e.g., activation of the mouth mecanoreceptors, *F*), in order to generate the proper "*reward*" signal, r_{t+1}.

According to the prey-catching ethogram, each action's expected consequences define a new situation with high probability of eliciting another prey-catching action, in such a way that the temporally extended course of actions ("*Plan*") ends up by executing a *snapping* and a *swallowing*, to signal that, partially, the *goal* has been achieved. That is, capturing one prey does not mean that *been fed* has been completed, but rather that an *episode* towards its completion has been carried out. Thus, an *exploring* phase starts all over again to initiate the next episode. This *episodic* behavior keeps on going until the nutrients level (an accumulative "*reward*" signal, represented by, *Q*, in Figure 11) reaches a certain level that would indicate the completion of the goal. At this point the probability of displaying prey-catching responses

would decrease, so the elicitation of other behavioral patterns can be favored (e.g., *in search of a hiding place*, which would allow the toad "*to go back home*").

Thus, the course of actions comprises the execution of a non determined number of prey-catching responses, and the completion of a series of short-term expected consequences (e.g., orienting to a prey bring it into the frontal part of the visual field), which should lead the toads to reach intermediate-term expected consequences (e.g., *food ingestion*), until a longer-term expected consequence is achieved (e.g., *being fed*). Thus, the course of actions during toads prey-catching behavior occurs in a temporal scale slower than that described for the prey-catching actions activation.

Additionally, the course of actions can be modulated by learning. When there is a sequence of situations activating the animal's prey-catching actions, $(s_t, a_t), (s_{t+1}, a_{t+1}), ..., (s_{t+n}, a_{t+n})$, with a sequence of *rewards*, $r_{t+1}, r_{t+2}, ..., r_{t+n+1}$, signaling the failure of expected consequences, then, a build up of habituation takes place. This learning phenomenon changes the course of actions (*plan*), highly reducing those situations' probability of eliciting a prey-catching response. The outcome of these changes keep the animal from attacking a non-catcahable prey, and allows its interaction with other key-stimuli that may appear in the immediate surrounding. In this way, an environmental situation, s_t, including both external and internal signals, contains all relevant information to activate a toad's prey-catching response, a_y. That is, our state description has the *Markov property* (Sutton and Barto, 1998); thus, given that, S_{pc} and A_{pc} are finite, the dynamics in the toad's visuomotor system might be described as using a *one-step model* of the environment, where the transitions dynamics can be represented by one-step state-transitions probabilities,

$$p_{ss'}^{a} = \Pr\{s_{t+1} = s' | s_t = s, a_t = a\},$$

and one-step expected "*rewards*",

$$r_s^a = E\{r_{t+1} | s_t = s, a_t = a\},$$

for all, $s, s' \in S$, and, $a \in A$, and assuming here that, $p_{ss'}^{a} = 0$ for $a \notin A_{pc}$.

Identifying this property in the amphibia's prey-catching behavior, opens the possibility of a fruitful exchange between scientists working in understanding the neuroethological basis of this behavior, and those developing the Theory of Reinforcement Leraning (Sutton and Barto, 1998). The former ones gain access to a mathematical framework (i.e., Markov and Semi-Markov Decision Processes) for analyzing, and testing, hypotheses related to sensorimotor coordination and temporally extended behaviors; whereas for the later ones, there is a corpus of experimental data and theoretical models (e.g., Schema-Theoretic, and neuronal networks) of how an *agent* (amphibian) represents knowledge of its world (prey-recognition as spatial-temporal patterns of neuronal activity within networks working at multiple temporal scales), and how it uses it to select the most appropriate action to cope with dynamic environmental situations (learning to stop responding to non-catchable prey).

5.- *CHANGES IN MOTIVATIONAL FACTORS: SLOWER TEMPORAL SCALES*

Prey-catching behavior elicitation is also modulated by signals coming from changes in internal processes attributable to the animal's motivation (i.e., changes due to an alteration in the animal's internal state). Some of them work like *internal clocks*, e.g., Ewert (1980) described seasonal effects on toads' prey-catching behavior; while others result from the animals' interactions with the *environment*. Ewert (1980) describes how prey-odors increase the animals response towards prey-like objects. Cervantes-Pérez *et al.*(1993b) reported that the way animals are handled in the lab, before the experiments take place, modifies the efficacy of visual *dummies* to yield toads' prey-catching responses; in one hand, toads with a high level of motivation to display predatory actions discriminate well between *worm-like* and *square* stimuli when they are moved at medium velocities (4.4 *cm/sec*); however, they present similar responses to both stimuli when moved at lower (2.2 *cm/sec*) and higher velocities (8.8 *cm/sec*). On the other hand, both stimuli were as effective to elicit prey-catching actions in animals with low level of motivation when moved at any of these velocities.

In addition, it has been postulated that these motivational effects may influence the activation dynamics produced by the presence of a prey-like stimulus over the neuronal elements of the retino-tectal-pretectal interactions (Ewert, 1980; Ingle, 1983; Ewert, 1991; Cervantes-Pérez *et al*, 1993b). Here, we analyze how these signals varying in a much slower temporal scale might modulate the RTP model dynamical characteristics, in order to show how changes in motivational factors may affect the nonlinear mappings embedded in the model to associate prey-like stimuli with proper prey-catching actions (e.g., toads yield prey-catching behaviors towards a prey when they are hungry, but not when they are satiated).

To simplify the analysis, we include a variable, $Q=\xi(tp(t),M_j)$, to represent the overall outcome of integrating the pretectal activation dynamics, $tp(t)$, due to the presence of a visual stimulus at time t, with changes in the animal's motivational state, M_j $(j=1,2,\ldots)$, formed by the responses of different sources of motivational changes, e.g., hunger, circadian cycles, etc. (see Figure 12). The hypothesis being that changes in motivational factors would modulate the pretectal inhibitory effect upon Tectum when a visual stimulus appears in the toad's visual field; sometimes increasing it (e.g., during the animal's periods of sexual activity of hibernation, or in satiated animals), and others doing just the opposite (e.g., during the summer, or in hungry animals).

It must be pointed out that neither of these effects is observable at the temporal scale of executing basic prey-catching actions; rather, they vary at a much slower multiple temporal scales (e.g., seasonal changes occur as yearly cycles, whereas the effects described by Cervantes and coworkers happened within periods of 14 and 17 hours). Thus, when analyzing visuomotor coordination experiments, the values of the variables associated to these modulating sources would be considered as constant parameters. In addition, we also outline a Dependency Analysis on Q, to show how it affects the efficacy of a visual prey-like stimulus to yield toads' prey-catching actions varies depending on the integration of the retinal level of activation it produces (R in Figure 12) with internally generated signals due to changes in motivational factors (i.e., toads Central Nervous System internal state), represented by M_j in Figure 12. In this way, neuronal circuits working at different multiple temporal scales (i.e., the RTP model and those generating M_j values) would be offered as the basis for a plausible explanation of why at different times toads respond differently to the very same environmental situation (e.g., hungry animals would catch a worm, while satiated animals would not).

PLACE FIGURE 12 HERE

Considering *Q*, and that PY cell does not participates in modifying the tectal internal activity, that is, considering only the neuronal elements (*GL*, *LP*, *SP*, *and SN*) responsible for producing and maintaining the tectal level of activation, we may define the following two vectors:

$$\mathbf{x} = (x_1(t), x_2(t), x_3(t), x_4(t))^T = (gl(t), lp(t), sp(t), sn(t))^T$$

$$\mathbf{y} = (y_1, y_2, y_3, y_4)^T = (h(gl), f(lp), f(sp), h(sn))^T$$

with *h* and *f* as defined in Section 2, so that the RTP model, in the absence of retinal input, could be expressed as:

$$\begin{pmatrix} \tau_1 \dot{x}_1(t) \\ \tau_2 \dot{x}_2(t) \\ \tau_3 \dot{x}_3(t) \\ \tau_4 \dot{x}_4(t) \end{pmatrix} = -\begin{pmatrix} x_1(t) \\ x_2(t) \\ x_3(t) \\ x_4(t) \end{pmatrix} + \begin{pmatrix} 0 & w_{21} & w_{31} & 0 \\ w_{12} & 0 & w_{32} & w_{42} \\ w_{13} & 0 & 0 & w_{43} \\ 0 & w_{24} & 0 & 0 \end{pmatrix} \begin{pmatrix} y_1 \\ y_2 \\ y_3 \\ y_4 \end{pmatrix} + \begin{pmatrix} w_{q1} \\ w_{q2} \\ w_{q3} \\ 0 \end{pmatrix} Q$$

in matrix notation,

$$\tau \dot{\mathbf{x}}(t) = -\mathbf{x}(t) + \mathbf{w_{ij}}\mathbf{y} + \mathbf{w}_q Q\,.$$

In addition, taking into account that *h(gl)*, *f(lp)*, *f(sp),* and *h(sn)* are piecewise linear functions, our analysis of the dynamical repertoire displayed by the RTM column model can be simplified by separating the overall system into a set of eight linear systems of first order differential equations, that collectively describe the nonlinear dynamics of the whole neuronal network. For example, in the situation where all neurons membrane potentials reach above threshold values (i.e., when $h(gl)=\beta_{gl}gl$, $f(lp)=1$, $f(sp)=1$, $h(sn)=\beta_{sn}sn$), the network dynamics is described by:

$$\begin{pmatrix} \tau_1 \dot{x}_1(t) \\ \tau_2 \dot{x}_2(t) \\ \tau_3 \dot{x}_3(t) \\ \tau_4 \dot{x}_4(t) \end{pmatrix} = -\begin{pmatrix} x_1(t) \\ x_2(t) \\ x_3(t) \\ x_4(t) \end{pmatrix} + \begin{pmatrix} 0 & w_{21} & w_{31} & 0 \\ w_{12} & 0 & w_{32} & w_{42} \\ w_{13} & 0 & 0 & w_{43} \\ 0 & w_{24} & 0 & 0 \end{pmatrix} \begin{pmatrix} \beta_1 x_1(t) \\ 1 \\ 1 \\ \beta_4 x_4(t) \end{pmatrix} + \begin{pmatrix} w_{q1} \\ w_{q2} \\ w_{q3} \\ 0 \end{pmatrix} Q$$

with, $\sigma_i = \dfrac{1}{\tau_i}$, this expression reduces to:

$$\dot{\mathbf{x}}(t) = \begin{pmatrix} -\sigma_1 & 0 & 0 & 0 \\ \sigma_2 w_{12}\beta_1 & -\sigma_2 & 0 & \sigma_2 w_{42}\beta_4 \\ \sigma_3 w_{13}\beta_1 & 0 & -\sigma_3 & \sigma_3 w_{43}\beta_4 \\ 0 & 0 & 0 & -\sigma_4 \end{pmatrix} \mathbf{x}(t) + \begin{pmatrix} \sigma_1(w_{21} + w_{31} + w_{q1}Q) \\ \sigma_2(w_{32} + w_{q2}Q) \\ \sigma_3 w_{q3} Q \\ \sigma_4 w_{24} \end{pmatrix}$$

which is a linear system of first differential equations:

$$\dot{\mathbf{x}}(t) = \mathbf{A}\mathbf{x}(t) + \mathbf{a}\,.$$

Accordingly, the state space of all membrane potentials can be split into eight different regions, each one described by a linear system. Each system has a unique equilibrium point, $\mathbf{e_i}$, **(1,2,…,8)** which is *asymptotically stable* (Cervantes-Pérez, 1985; Cervantes-Pérez and Arbib, 1990). Thus, we can study the RTP neuronal network dynamics around the equilibrium points. First, we determine their location in state space. Following our example (region 8):

$$\mathbf{e_8} = \begin{pmatrix} w_{21} + w_{31} \\ \beta_1 w_{12}(w_{21} + w_{31}) + \beta_4 w_{42} w_{24} + w_{32} \\ \beta_1 w_{13}(w_{21} + w_{31}) + \beta_4 w_{43} w_{24} \\ w_{24} \end{pmatrix} + \begin{pmatrix} w_{q1} \\ \beta_1 w_{12} w_{q1} + w_{q2} \\ \beta_1 w_{13} w_{q1} + w_{q3} \\ 0 \end{pmatrix} Q = \mathbf{p}_8 + \mathbf{m}Q$$

which has been calculated by solving:

$$\dot{\mathbf{x}}(\mathbf{t}) = \mathbf{A}\mathbf{e}_i + \mathbf{a} = \mathbf{0}$$

m is a constant vector that appears in all equilibrium solutions, and *Q* acts as a modulating parameter. Then, we analyze the network stability by determining the eigenvalues λ_i *(i=1,…4)* of matrix **A,** by solving the characteristic equation, $|\lambda\mathbf{I} - \mathbf{A}| = \mathbf{0}$. Thus, for region 8:

$$\lambda_1 = -\sigma_1, \quad \lambda_2 = -\sigma_2, \quad \lambda_3 = -\sigma_3, \quad \lambda_4 = -\sigma_4\,.$$

Because all membrane time constants are positive, all eigenvalues of matrix **A** are real and negative: $R_e\lambda_i<0$; so, the RTP neuronal model is *asymptotically stable* around the equilibrium solution $\mathbf{e_8}$, that is, $\mathbf{x}(t)\rightarrow\mathbf{e_8}$, as $t\rightarrow\infty$ for each initial state **x(*0*)** (Ogata, 1970; Shinners, 1978). Similar results are obtained for all other regions.

In our analysis, all equilibrium solutions, $\mathbf{e_i}$ **(i=1,2,…,8)**, and their stability are extremely important for two reasons. First, because they are the basis for defining the repertoire of dynamic behaviors that may be displayed by the RTP model. That is, when they are located within their corresponding linear region, as *actual points*, the global activity of the neuronal network can converge to it; but if they fall outside their corresponding linear region, as *virtual points*, none of the trajectories of the global nonlinear system can converge to it (Ogata, 1970). Second, because their location in state space depends on the value of *Q*, the RTP dynamic behavior elicited by a given environmental situation would be modulated by changes in motivational factors M_j, whose processing occurs in neuronal mechanisms working at multiple slow temporal scales.

In Cervantes-Pérez *et al* (1993b) we showed that toad's motivational state might modulate the level of pretectal inhibition upon tectum, modifying the efficacy of a *worm-like* stimulus to elicit prey-catching behaviors, but that it was not enough to consider only changes in the characteristics of *tp* cell's activation (e.g., changes in the threshold, θ_{tp}). Our results allowed us to suggest that there might be other modulation effects acting at the level of the tectal input. Therefore, the effects of the M_j over the pretectal inhibitory effect upon tectum could occur in any of the following forms:

1. The M_js could act as a constant input that modifies the *tp* cell resting potential, getting it closer or further from the cell's threshold, θ_{tp} (see Figure 13a). Thus, when a visual stimulus appears in the animal's visual field, there would be three characteristics in the *tp* cell activation dynamics that would change: the *latency* of the *tp* cell response, its *duration*, and its *intensity* (l_k and I_k, correspondingly in Figure 13a). This will also delay the generation of *f(tp)*, modifying in this way its synchronicity with those retinal signals at the moment of arriving at the Optic Tectum. In this case, $Q= f(tp)$, representing a inhibition upon tectal activation (i.e., $Q<0$).

2. The M_js could affect the intensity of *f(tp)* acting upon *tp*'s axon (see Figure 13b). Thinking in a possible neuronal mechanism similar to that of *Presynaptic Inhibition* (Rudomin, 1990), if the frequency of *the* M_j is too low compared to the one of *f(tp)* then, $Q=f(tp)$; but, if M_j's frequency gets bigger values then its effect would be equivalent to a multiplication by a parameter *k*, which varies between 0 and 1 (i.e., $Q= k*f(tp)$, *for* $0<k<1$). Finally, if M_j's frequency gets bigger than *f(tp)*, it would completely block *tp* cell's output, and $Q=0$. In summary, under this circumstances $Q\leq 0$, and there would be changes only in the *intensity* of the pretectal cell response.

3. The M_js might counteract pretectal inhibition at the level of tecatl input (see Figure 13c). If the interaction between the M_js and *f(tp)* takes place at Tectum then, $Q=Mj-f(tp)$. For M_j with excitatory effects, it would be possible that pretectal inhibition upon tectal elements could be counteracted, or even surpassed by the action of a motivational source M_j (i.e., $Q\geq 0$). Again, the *intensity* of the pretectal cell response would be modulated, although in this case, *Q* might even become positive, increasing the probability for a stimulus to fit the prey-category.

PLACE FIGURE 13 HERE

To understand how tectal integration of signal generated by neuronal mechanisms working at multiple temporal scales might modulate the action-selection mechanisms in the toads visuomotor system, we carried out computer simulations for $Q<0$, $Q=0$ and $Q>0$. Figure 2 shows the situation for $Q=0$, a prey-like stimulus is presented for 0.5 *sec*, the tectal elements GL, LP, SN, SP and PY become activated, in that order. LP, SN and SP present oscillatory behavior during the time they remain active. Here, we assume that, once PY gets activated, a prey-catching action is produced and the stimulus is captured, so it disappears from the toad's visual field. In Figure 14 we show a computer simulation of a situation when $Q<0$ ($Q=-0.3$), assuming a direct effect of motivational center upon Tectum, the presence of a visual prey-like object increases the level of activity in GL, LP, SP and PY without eliciting any prey-catching response. Thus, the prey-like stimulus would follow its way and will eventually disappear from the animal's visual field. Because LP is the only input to SN, this cell remains in its resting conditions during the whole process.

PLACE FIGURE 14 HERE

On the contrary, when $Q>0$, the presence of the same stimulus generates great activity among the tectal elements of the RTP neuronal circuit. In Figure 15a we show the results of a computer simulation where $Q=0.02$, and in Figure 15b when $Q=0.06$. It can be observed that the level of activation in the RTP column model increases; furthermore, there is an oscillatory

behavior presented by LP, SP and SN neurons that gets stronger for higher values of *Q*, which causes an almost sustained strong response in GL and a longer period of activity in PY(even after several time simulation units after the stimulus has disappeared fro the animal's visual field. The later being postulated as the trigger of prey-catching actions, then, this would mean that motivated animals would display faster and stronger responses towards prey-like stimuli during long periods of time. In addition, as *Q* increases, GL and SP not only reach a stronger level of activation, but they remain active during a longer period of time. These functional units act as a short-term memory mechanism: in highly motivated animals, the presence of a prey-like stimulus would facilitate toad's responses to subsequent stimuli.

PLACE FIGURE 15 HERE

Finally, it must be pointed out that the idea of sensory-motor coordination being modulated by changes in motivational factors was already put in practice in Arkin *et al* (2000), where we included three motivational variables: *hunger*, *sex-drive*, and *fear*. There, the first two were considered to increase linearly with time, and to reset to zero when the robot makes contact with a prey, or a mate; at that time it is assumed that the robot has eaten or mated. *Hunger* was also assumed to increase more rapidly than *sex-drive*. In the case of *fear*, its level remains equal to zero until a predator appears in the robot's visual field, then, the corresponding variable is set to a predetermined high value, and reset to zero once the predator is no longer visible. Thus, in order for the robot to properly cope with specific environmental situations, in the *Action-Selection* algorithm the state of the variable with the greatest current value is integrated with the level of activity produced by the current situation. If there is an associated stimulus (i.e., a prey for the *hunger* variable), then the robot executes the corresponding behavior, otherwise, this process is repeated with the motivational variable with the next greatest value, and so on. In case there is not a prey, or a predator, or a mate, or an object that signals a *hiding* place, then, the robot keeps on exploring the surroundings. Thus, as a sequel of our work, the challenge remains to design control algorithms that include more realistic models of how motivational variables, changing over time at multiple temporal scales, affect the efficacy of visual stimuli to yield proper behaviors.

7.- *CONCLUSIONS*

In one hand, we have analyzed how the neuronal mechanisms underlying amphibia's prey-catching behavior are organized into functional units of interconnected cells, distributed along different brain structures, and with retinotopic mappings and dynamical features essential for generating a set of neuronal signals at multiple temporal levels of abstraction. For example, the RTP model's *cooperation/competition* processes between positive and negative feedback loops provide a scenario for the appearance of oscillatory dynamic behaviors, which have been offered as the basis for short-term memory processes underlying the modulation of prey-catching behavior, by stimulus specific habituation and changes in motivational factors. This modulation could be described as the outcome of accommodating information of new situations (a non catchable prey-like stimulus) into the dynamics of the proper neural mechanisms (TP cell inhibitory effect upon tectal elements), in order to modulate the way amphibia normally respond to a specific stimulus (stop yielding prey-catching behaviors), without affecting its performance when similar situations appear in its immediate surroundings (prey-catching behaviors to real prey should remain unchanged). That is, we have posed that, once an amphibian is engaged into capturing prey, the elicitation of prey-catching actions not only depends on the activity caused by the stimulus characteristics and the current spatial-temporal relationship between both animals, but rather on its integration with other signals generated by inner parallel processes occurring at multiple temporal scales (e.g., learning, and motivation).

On the other hand, seeking to establish a fruitful interactive exchange between scientist working in Neuroscience and those concerned with building computer based sensory guided automata, we tried to make our analysis amenable to those communities of Distributed Artificial Intelligence and Behavior-based Robotics. We presented our analysis in terms of an *agent* (toad) that stores, within the dynamics of neuronal processes working at *multiple temporal scales*, information of certain *domains of interaction* (prey-like stimuli) that may appear in the uncertain environment where they live (its *ecology*). Specifically, we studied stimulus specific habituation in toads as a *Reinforcement Learning Problem,* where toads ethograms can be looked at as conforming a set of behavioral systems embedding *planning* processes flexible enough to allow to modulate the execution of a *sequence of episodes* (temporally extended actions), to optimize the probability of the animal achieving overall goals, and immediate sub-goals.

Thus, it is our intention that our study stimulates an exchange of data, models and analytical mathematical frameworks that may lead to a better understanding of the biological phenomena and, at the same time, to the inspiration of alternative models of knowledge representation, learning and planning that might be useful within Behavior-based Robotics and Distributed Artificial Intelligence.

ACKNOWLEDGEMENTS

FIGURE CAPTIONS

Figure 1.- Retino-Tectal-Pretectal (RTP) Column Model. This model includes the output of retinal ganglion cells R2, R3, and R4. R3 and R4 impinge upon the thalamic pretectal cell TP; R2 upon the tectal glomerulus, GL, and the large, LP, and small, SP, pear-shaped cells; and R2, R3 and R4 upon the PY cell, which is the only efferent element in the tectum. There is an inhibitory effect from TP cell upon tectum (i.e., upon all tectal elements, but the SN cell), and a cooperation/competition process among positive (i.e., GL and the LP and SP cells), and negative (i.e., SN and LP and SP cells) feedback loops. The level of tectal activity is controlled by the SN cell, the only tectal element not receiving external input, which exerts an inhibitory effect upon LP and SP cells (adapted from Cervantes-Pérez *et al*. 1985).

Figure 2.- Computer simulation of the dynamics presented by the neuronal elements of the RTP column model when processing a prey-like stimulus. GL is the first unit in getting activated, then LP, followed by SN and SP. When LP and SP are active, and the stimulus is still in the visual field, then, PY cell gets activated signaling that the stimulus fits the prey-category.

Figure 3.- Computer Simulation of Prey-Catching Facilitation in a Tectal Column model. During the first presentation of a prey-like object (square of 2×2 *deg* with a velocity of 8*deg/sec*), during a sub-threshold duration (0.3 *sec*), there is and oscillatory behavior in all cells, but no PY response. After an intervening delay of 3.2 *sec*, the residual GL and SP activity left after the first stimulation allows the network to elicit a PY response during a second sub-threshold duration presentation (adapted from Cervantes-Pérez and Arbib, 1990).

Figure 4.- Computer simulation of an oscillatory behavior in the Tectal Column model. When recurrent axons from LP and SP produce a strong GL residual activity: a) the network might present alternating periods of activity in LP, SP and SN, with no PY activation; and b) or, when positive feedback loop gets much stronger than the negative feedback loop, there could be sustained SP and SN responses with a strong PY activation (adapted from Cervantes-Pérez and Arbib, 1990).

Figure 5.- Computer simulation of a strong SN inhibitory effect. When SN inhibitory effect upon LP and SP cells is strong enough to avoid, after the first stimulus presentation, that GL residual activity elicits a response from either SP or LP cells, Prey-Catching Facilitation would not take place (adapted from Cervantes-Pérez and Arbib, 1990).

Figure 6.- Prey-catching stimulus specific habituation in toads *Bufo-Marinus horribilis*. Each graph corresponds to an animal's response to a *worm-like* stimulus (i.e., horizontal black rectangle moving at 4.1 *cm*/*sec*, in a white background with its longest axis parallel to the direction of motion). As the experiment progresses, the frequency (vertical axis) of prey-catching responses towards the stimulus decreases, followed by a qualitative change: toads stop eliciting prey-catching actions towards the stimulus used during the experiment (adapted from Cervantes-Pérez *et al*., 1991).

Figure 7.- Prey-catching habituation in toads is stimulus specific. See text for explanation.

Figure 8.- Neuronal Network Model for the Habituation Column. *FU* (Failure Unit), gets activated when the expected consequence of executing a prey-catching action fails to occur; *MO* (MOdeling unit), creates a model of the input coming from the Retina; *CO* (Comparison

unit), which triggers the habituation process, once the *MO* output matches the input coming from the Retina; *h* (habituation unit), builds up the level of habituation due to the presence of a prey-like stimulus despite the toad's efforts to catch it; and *H* (Habituation column output), send an excitation signal to *TP* cell, in order to modulate the level of pretectal inhibition upon tectal elements.

Figure 9.- Computer simulation of the Habituation Column Model Dynamics. It is assumed that every stimulus presentation yields a prey-catching action whose expected consequence fails to occur. This activates the Modeling Unit until its output matches the input coming from the Retina. At this point, the building up of prey-catching habituation begins, and the intensity of the signal sent by the Habituation Output unit to the TP cell (see Figure 8) increases to a maximum as the stimulus remains in the visual field despite toad's efforts to capture it.

Figure 10.- Computer simulation of the stimulus specificity of habituation. When an animal has been habituated to a stimulus A and a catchable stimulus B appears in the visual field, the Modeling Unit output differs from the retinal signal. Thus, the Comparison Unit stays active all the time, inhibiting the Habituation Output unit. Therefore, a prey-catching action can be activated once again, while the Modeling and the Habituation units begin to return to their resting levels, following a much slower time constant.

Figure 11.- The *agent-environment* interaction in amphibia's prey-catching behavior. See text for explanation.

Figure 12.- Modulation of the RTP column model. *Q* represents the modulation function that integrates signals coming from the TP pretectal neuron, already modulated by the Habituation Column, with signals coming from motivational centers M_j's. Thus, the combination of *Q* and the retinal input *R* determine whether, under current situation, the stimulus in the animal's visual field fits the prey-category.

Figure 13.- Alternative modulation schemes for changes in motivational factors. See text for explanation.

Figure 14.- Computer simulation of a situation when $Q<0$. In this case, $Q=-0.3$, only GL becomes activated, whereas all other RTP elements reach only sub-threshold membrane potentials.

Figure 15.- Computer simulations of situations with $Q>0$. The stimulus configuration is the same as in Figure 14. As motivation increases the value of *Q* (Figure 15a) $Q=0.02$, and Figure 15 b, $Q=0.06$), the oscillatory behavior of LP, SP and SN gets stronger, which yields a better PY cell response, improving the stimulus efficacy to elicit toad's prey-catching responses. In addition, GL remains active for a longer time period (4 *sec* in Figure 15a, and up to 8*sec* in Figure 15b), which may facilitate the elicitation of prey-catching responses by sub-threshold stimuli that may appear subsequently.

TABLES DESCRIPTION

Table 1. Spontaneous Recovery after prey-catching stimulus specific habituation. FR— Frequency Response; IT— Inhibition Time. See Text for explanation.

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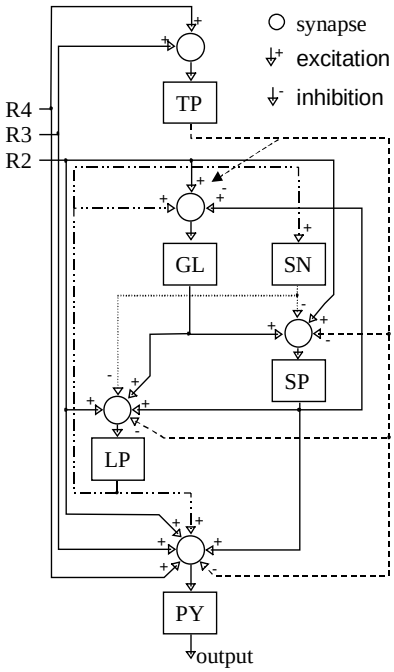
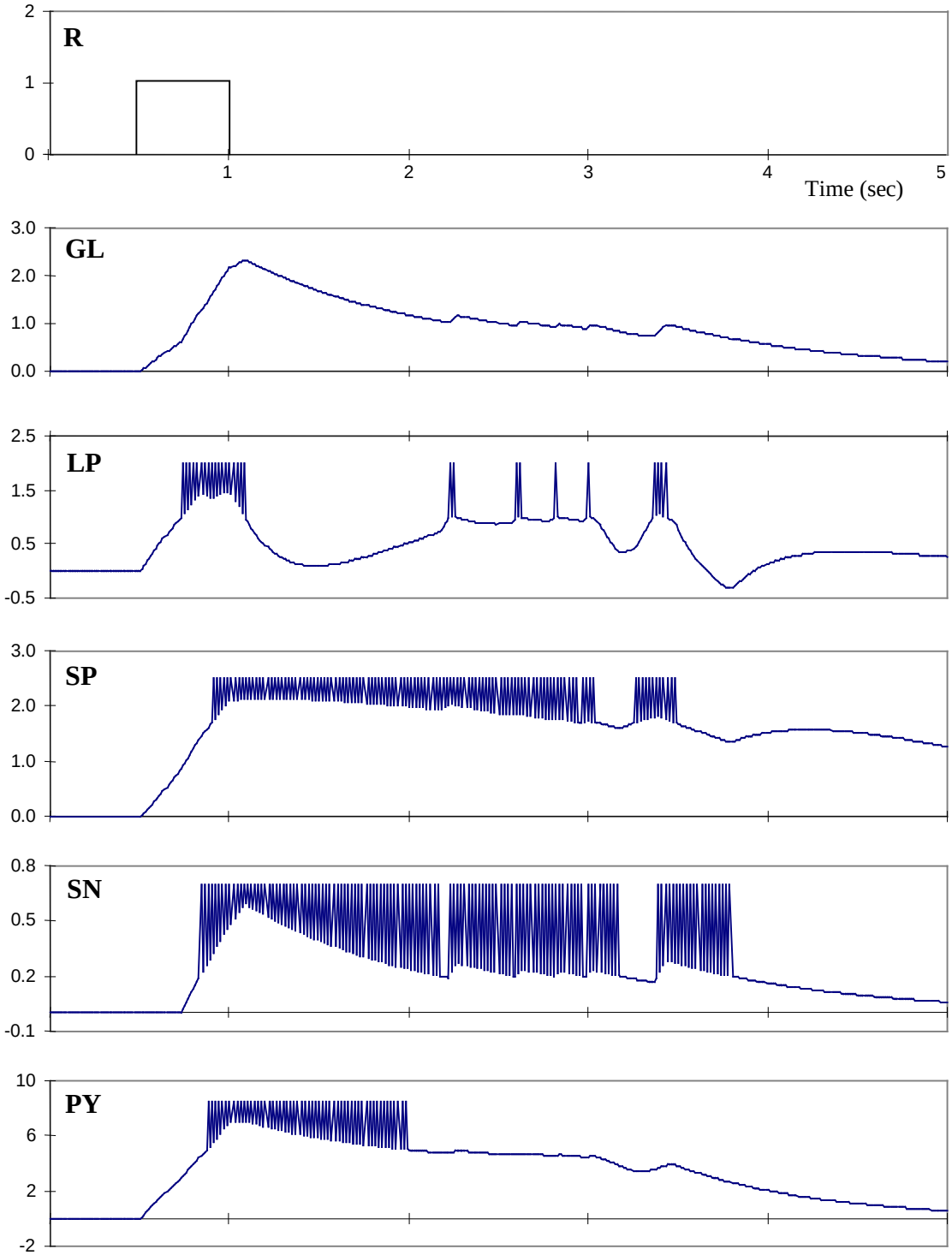

synapse
excitation
inhibition
R4
R3
R2
TP
GL
SN
SP
LP
PY
output

Figure 1.-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2
1
0
1
2
3
4
5
Time (sec)
GL
3.0
2.0
1.0
0.0
LP
2.5
1.5
0.5
-0.5
SP
3.0
2.0
1.0
0.0
SN
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0.5
0.2
-0.1
PY
10
6
2
-2

Figure 2.-

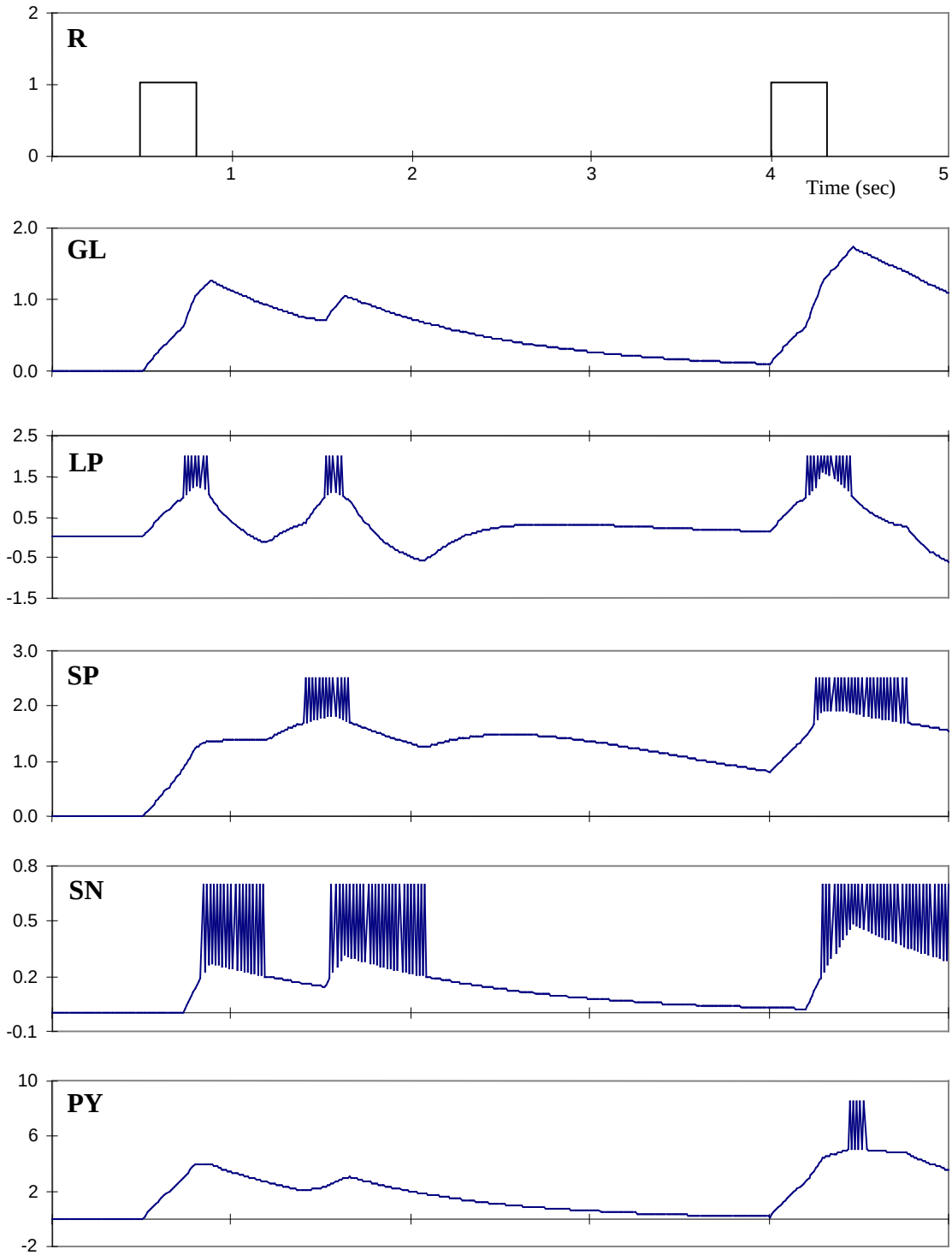

Figure 3.-

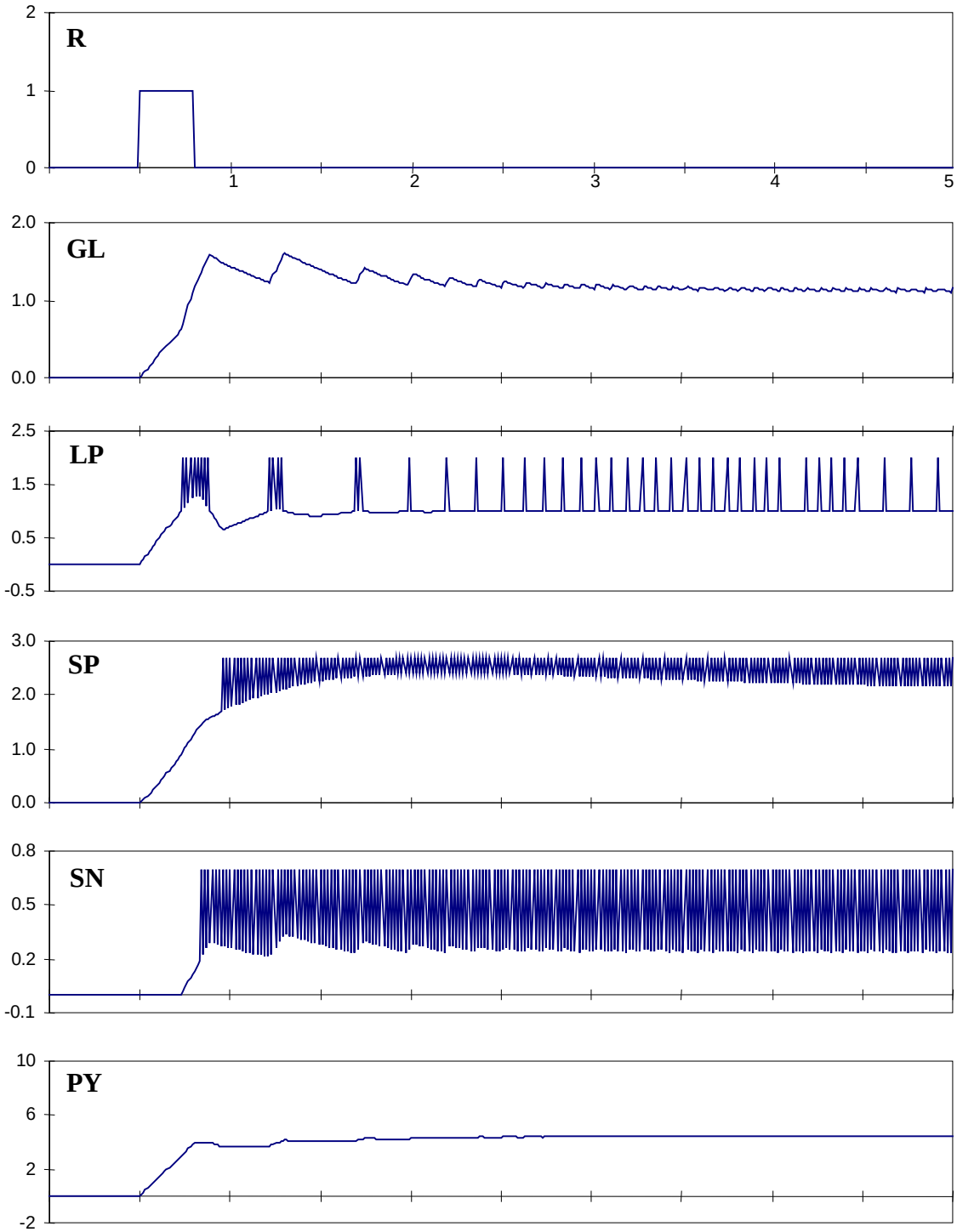

Figure 4a.-

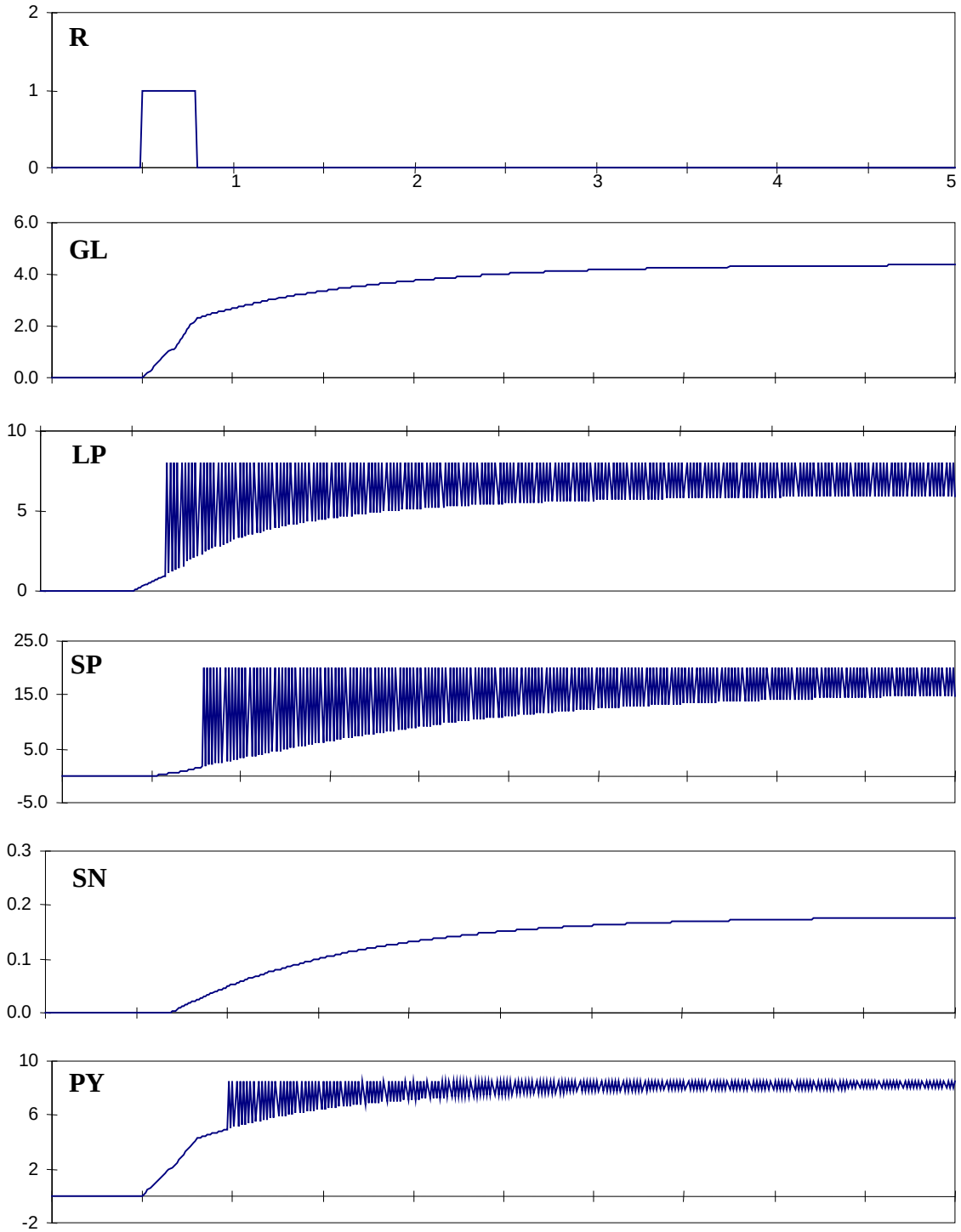
R
2
1
0
1
2
3
4
5
GL
6.0
4.0
2.0
0.0
LP
10
5
0
SP
25.0
15.0
5.0
-5.0
SN
0.3
0.2
0.1
0.0
PY
10
6
2
-2

Figure 4b.-

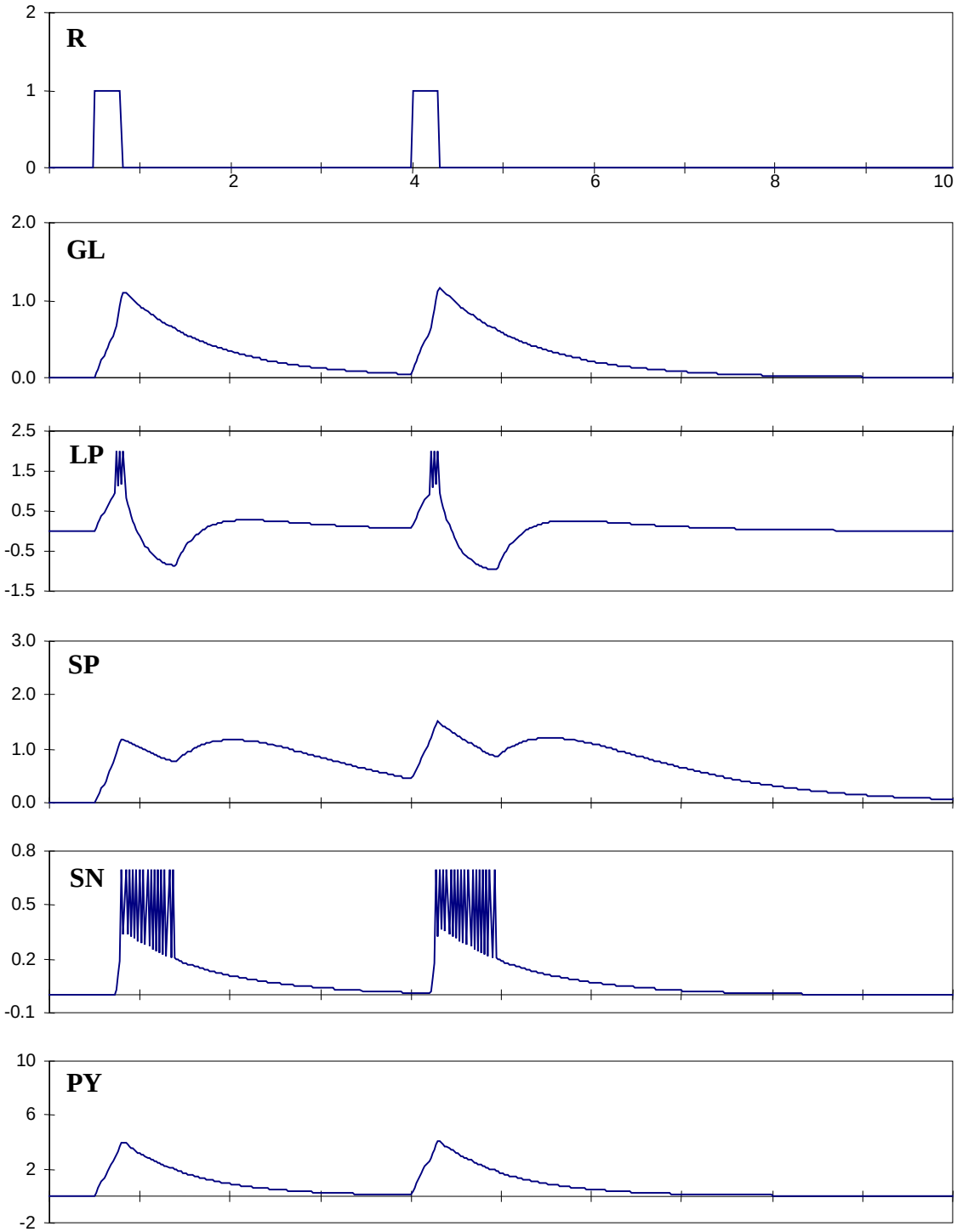

Figure 5.-

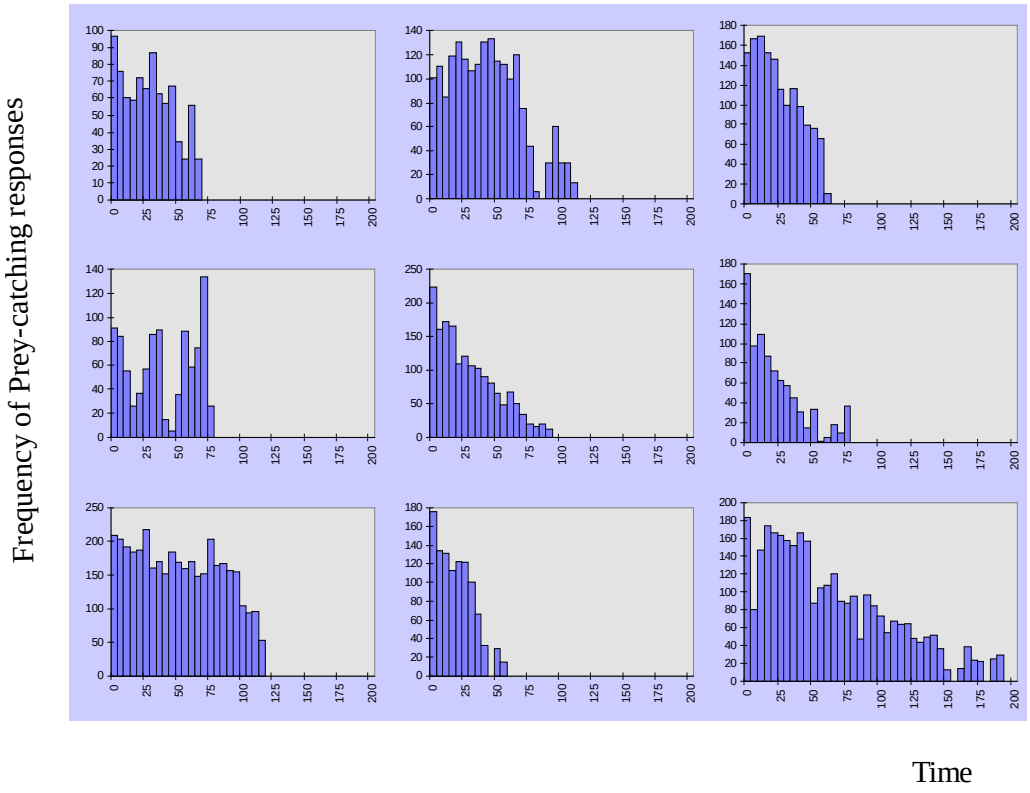

Figure 6.-

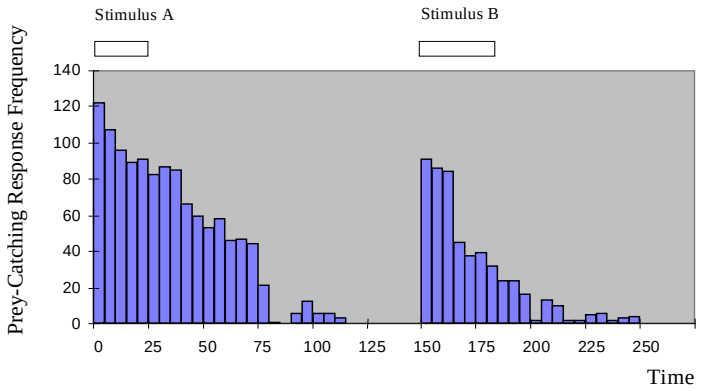
Stimulus A
Stimulus B
Prey-Catching Response Frequency
140
120
100
80
60
40
20
0
0
25
50
75
100
125
150
175
200
225
250
Time

Figure 7.-

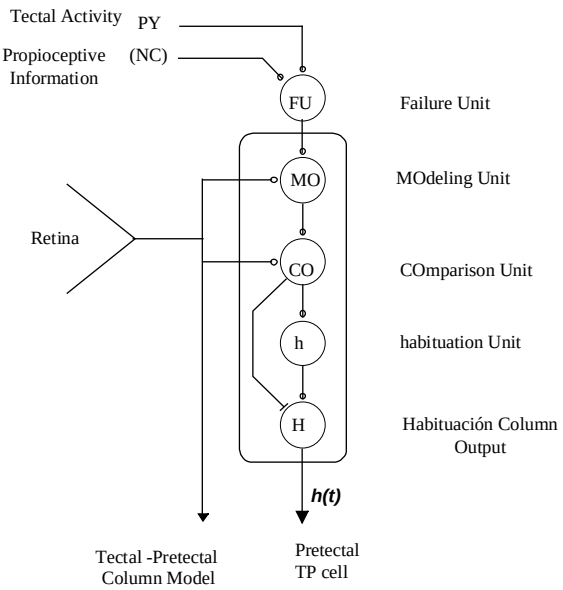
Tectal Activity PY
Propioceptive Information (NC)
FU
Failure Unit
MO
MOdeling Unit
Retina
CO
COmparison Unit
h
habituation Unit
H
Habituación Column Output
h(t)
Tectal -Pretectal Column Model
Pretectal TP cell

Figure 8.-

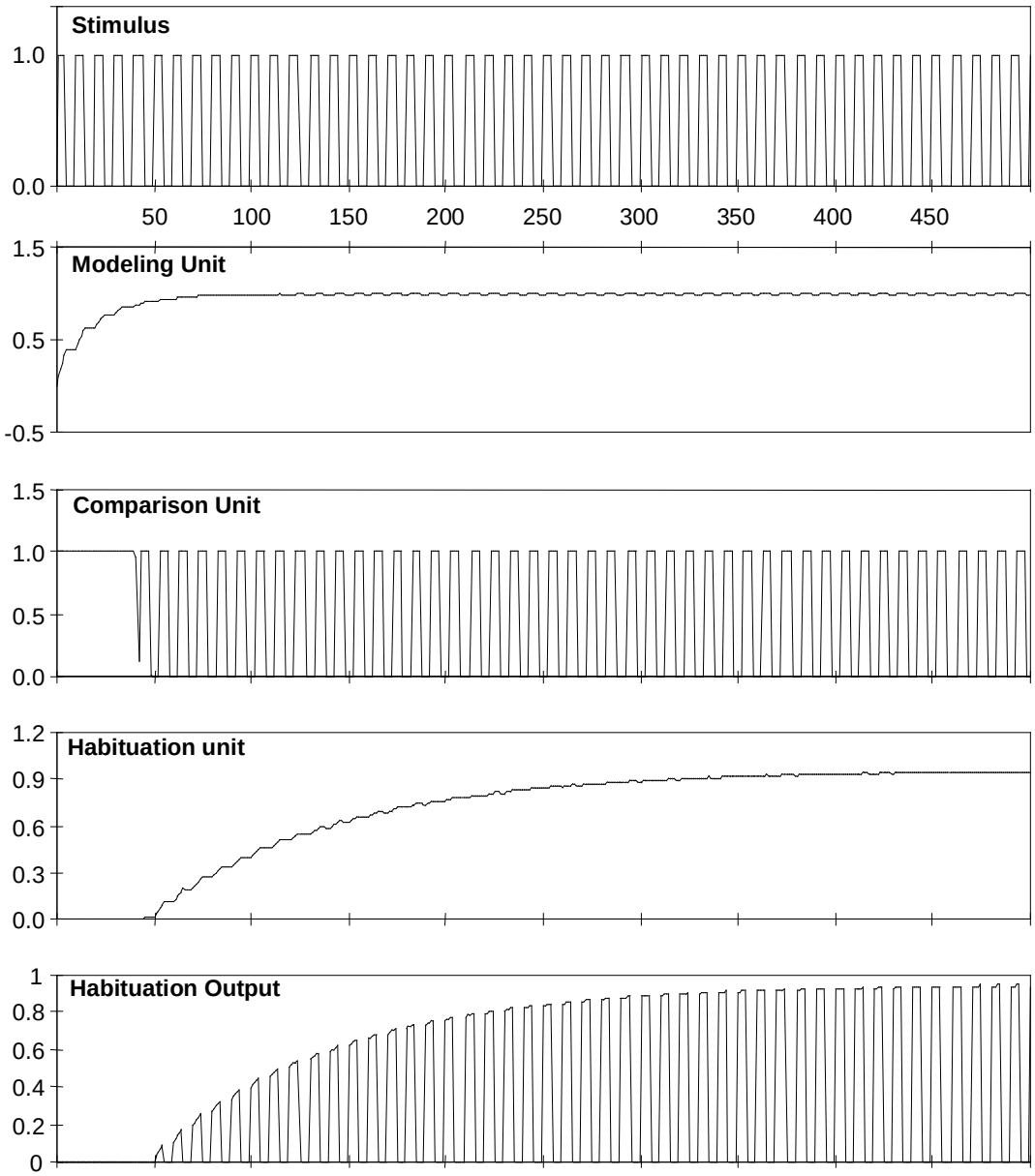
Stimulus
1.0
0.0
50
100
150
200
250
300
350
400
450
1.5
Modeling Unit
0.5
-0.5
1.5
Comparison Unit
1.0
0.5
0.0
1.2
Habituation unit
0.9
0.6
0.3
0.0
1
Habituation Output
0.8
0.6
0.4
0.2
0

Figure 9.-

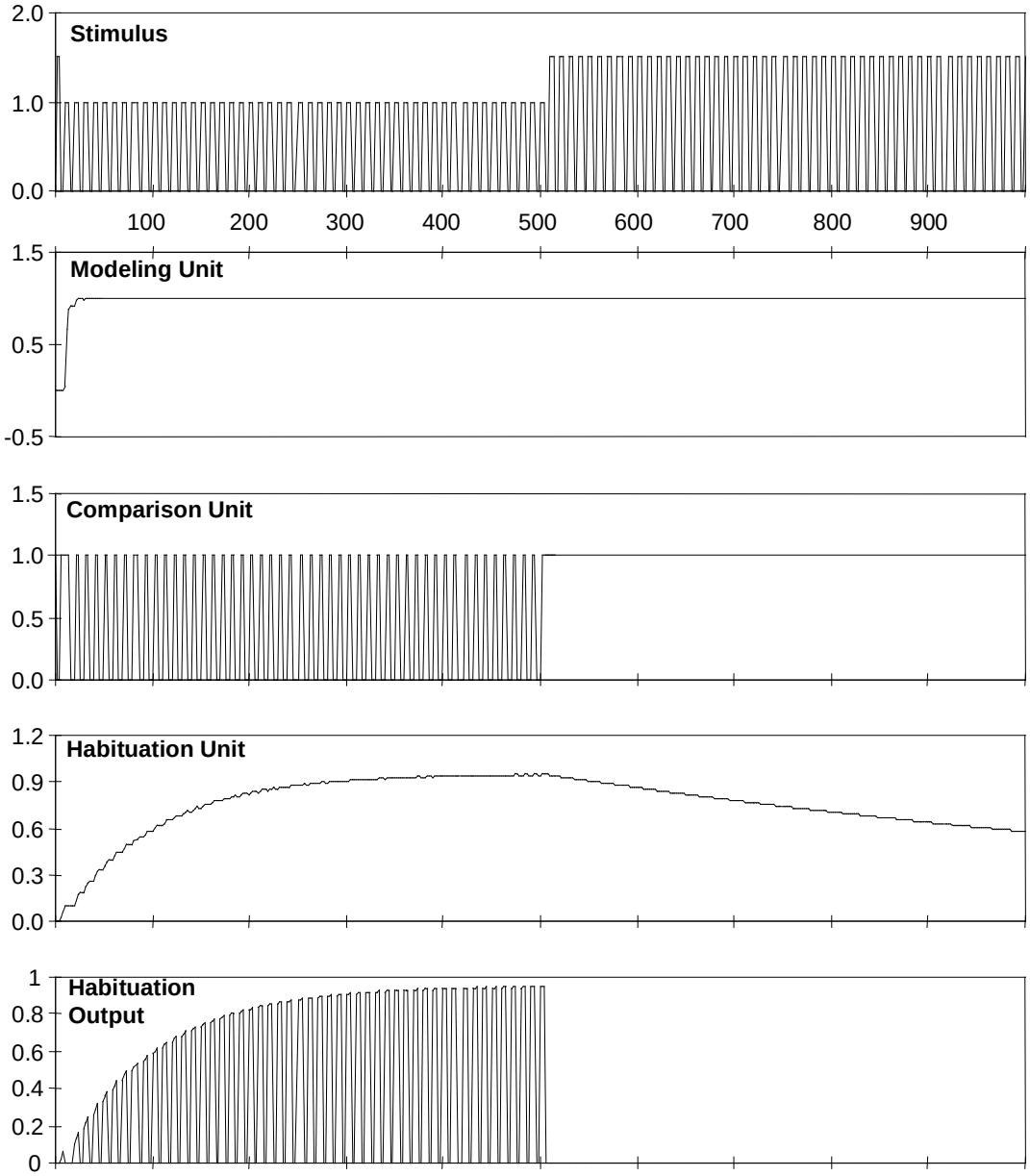
Stimulus
2.0
1.0
0.0
100
200
300
400
500
600
700
800
900
Modeling Unit
1.5
0.5
-0.5
Comparison Unit
1.5
1.0
0.5
0.0
Habituation Unit
1.2
0.9
0.6
0.3
0.0
Habituation
Output
1
0.8
0.6
0.4
0.2
0

Figure 10.-

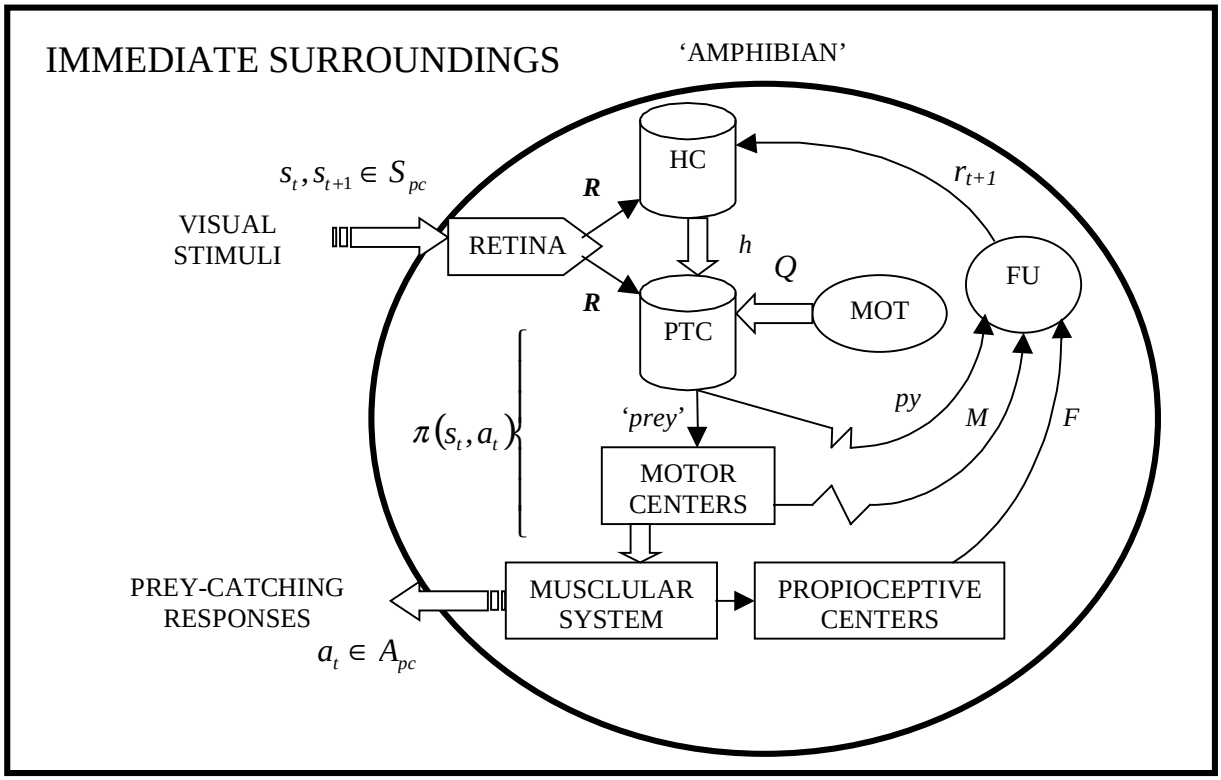
IMMEDIATE SURROUNDINGS
'AMPHIBIAN'
$s_t, s_{t+1} \in S_{pc}$
VISUAL STIMULI
RETINA
R
R
HC
PTC
h
Q
MOT
FU
r_{t+1}
$\pi(s_t, a_t)$
'prey'
py
M
F
MOTOR CENTERS
MUSCLULAR SYSTEM
PROPIOCEPTIVE CENTERS
PREY-CATCHING RESPONSES
$a_t \in A_{pc}$

Figure 11.-

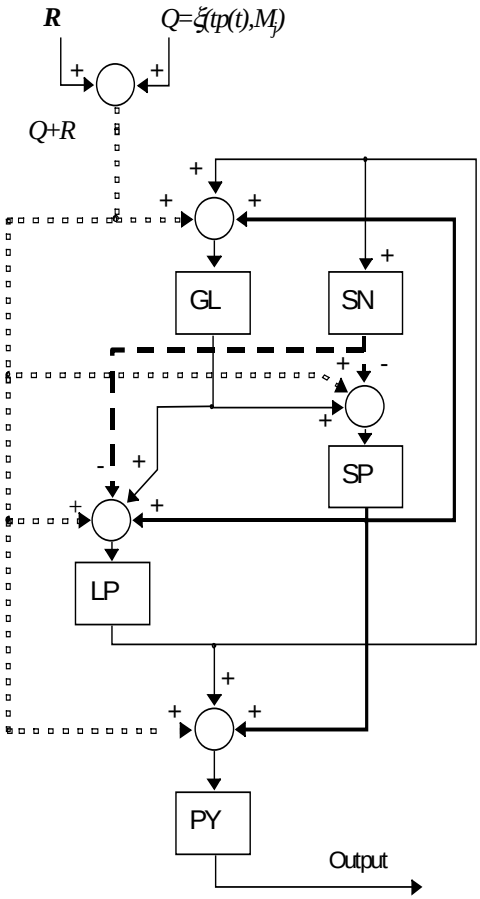

Figure 12.-

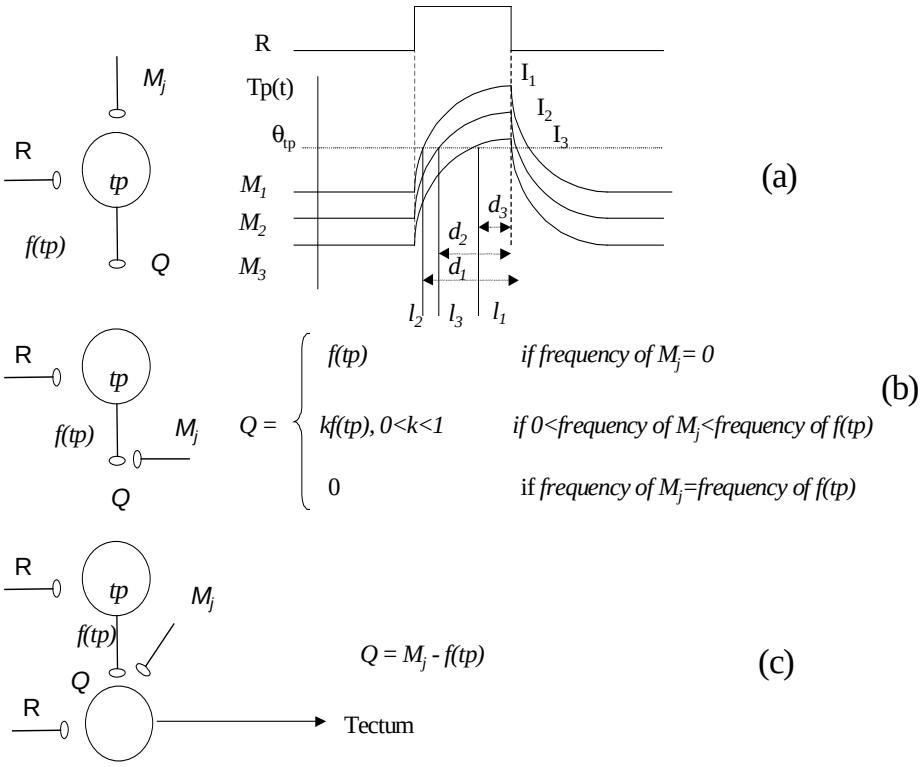
R
Tp(t)
θtp
M1
M2
M3
I1
I2
I3
d1
d2
d3
l1
l2
l3
(a)
R
tp
f(tp)
Q
Mj
(b)
Q = f(tp) if frequency of Mj = 0
kf(tp), 0<k<1 if 0<frequency of Mj<frequency of f(tp)
0 if frequency of Mj=frequency of f(tp)
Q = Mj - f(tp)
(c)
Tectum

Figure 13.-

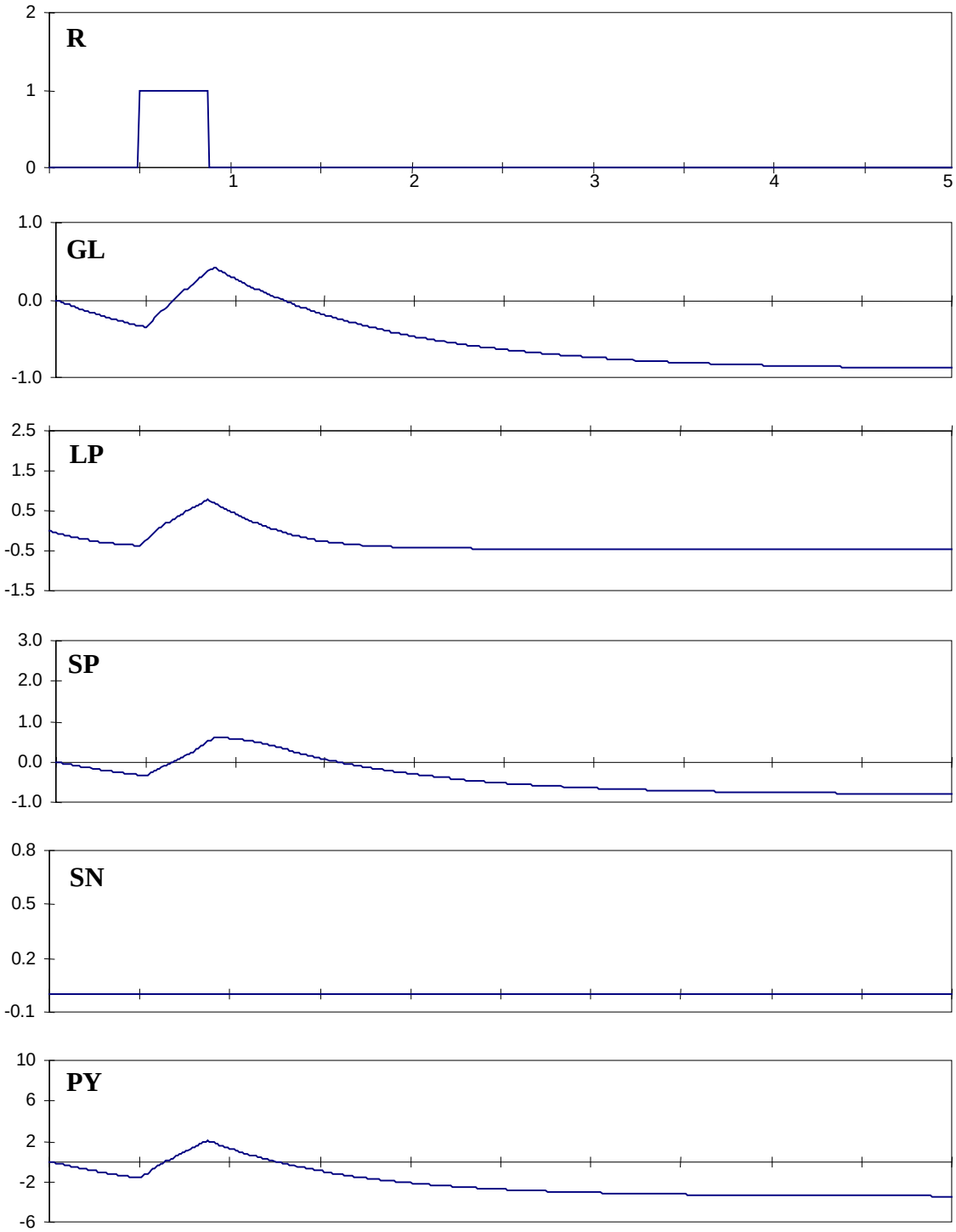
R
2
1
0
1
2
3
4
5
GL
1.0
0.0
-1.0
LP
2.5
1.5
0.5
-0.5
-1.5
SP
3.0
2.0
1.0
0.0
-1.0
SN
0.8
0.5
0.2
-0.1
PY
10
6
2
-2
-6

Figure 14.-

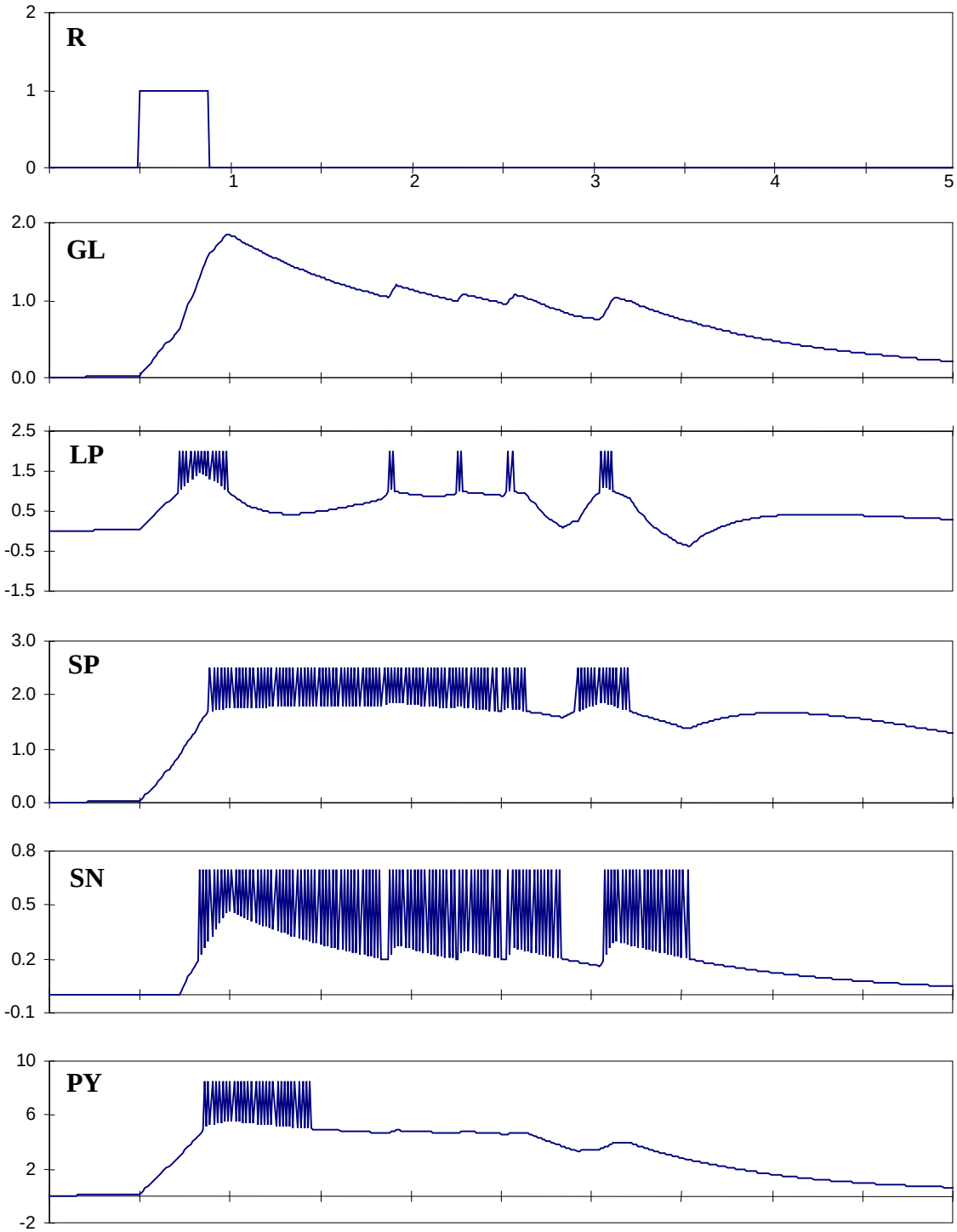

Figure 15a.-

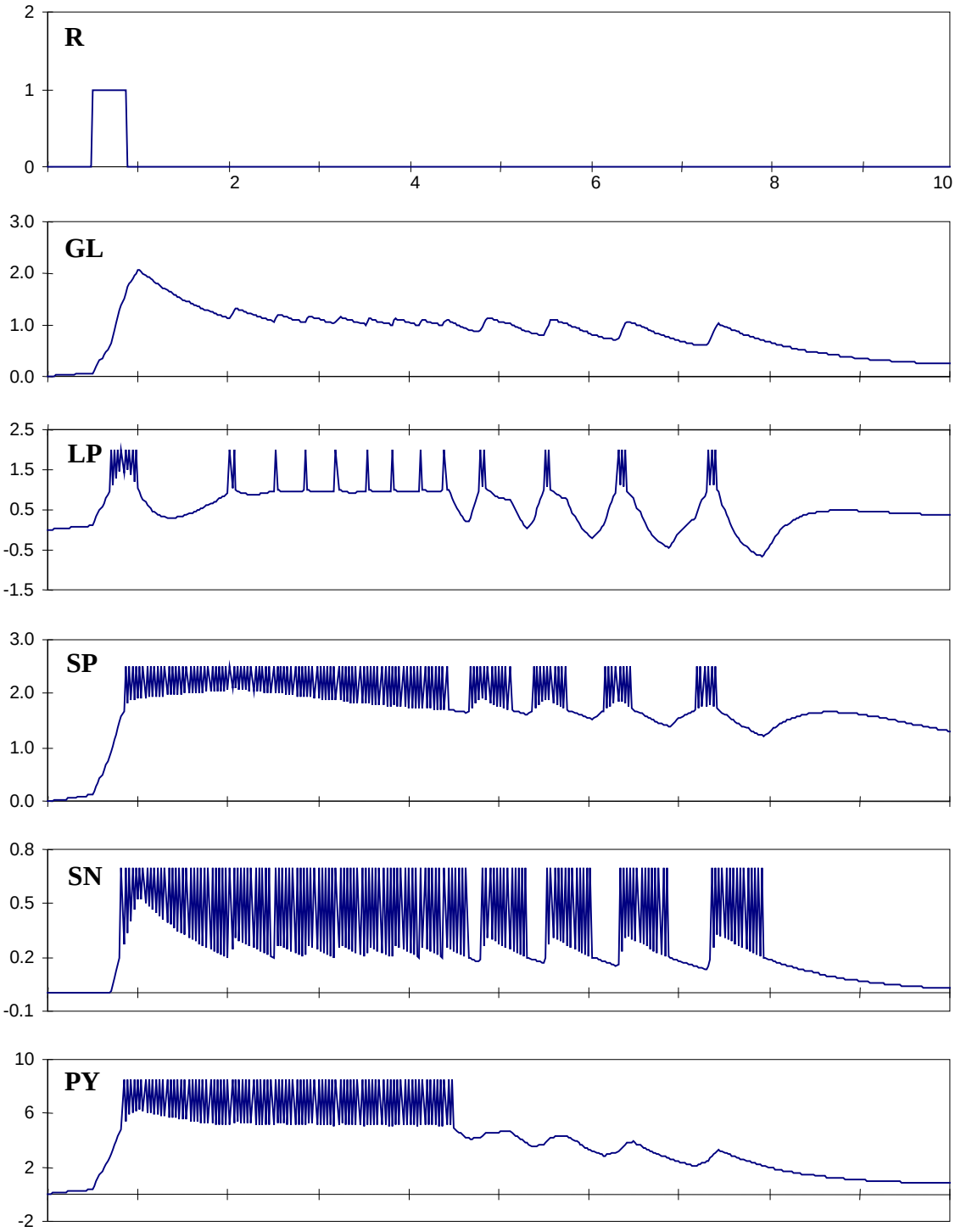
R
2
1
0
2
4
6
8
10
GL
3.0
2.0
1.0
0.0
LP
2.5
1.5
0.5
-0.5
-1.5
SP
3.0
2.0
1.0
0.0
SN
0.8
0.5
0.2
-0.1
PY
10
6
2
-2

Figure 15b.-

Table 1.- Spontaneous recovery after a Stimulus Specific Habituation training

***Worm-like* Stimulus (*cm*)**	**Inter-stimulation period (*days*)**	**Effect**	**Response Frequency**	**Time to reach Habituation**
1.5 *x* 0.4	11	No Response		
"	13	No Response		
"	20	No Response		
"	45	No Response		
"	49	Response	22 *responses/min*	28 *min*
"	56	No Response		
"	58	No Response		
"	64	Response		
"	95	No Response		
"	95	Response	1.8 *responses/min*	10 *min*
"	133	Response	7 *responses/ min*	115 *min*