# A NeuroRobotic Model of Infant Looking Behavior

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#### Abstract

Very young human infants demonstrate visual exploration behavior. The behavior is modulated by habituation as stimuli are experienced multiple times. Primate studies have shown that when neural structures responsible for habituation are lesioned, the visual exploration behavior is retained while while the habituation (learning) component is abolished. This paper presents an anatomically-inspired neuro-robotic model of the visuomotor (oculomotor) system that can accomplish looking behavior similar to that observed in non-learning infants or in primates with lesioned parahippocampal regions. The neuroanatomical basis for the different parts of the model and their interaction are discussed.

### 1 Introduction

Newborn human infants are capable of astounding behaviors with which they are not commonly attributed. For example, visual habituation, a type of visual category learning, is present at least as early as birth (Slater, Morison, & Rose, 1984). The typical way of testing these (visual) behaviors is to measure the aggregate looking time towards a visual stimulus over multiple encounters with that stimulus (Fantz, 1964). This is compared to the response to a never-experienced visual stimulus that is matched for some properties such as complexity and salience. If the looking time to the "familiar" stimulus is different than the looking time to the "novel" stimulus, then the ability of the infants to discriminate between the stimuli, and even the ability to learn to recognize the stimuli (at least as being "previously encountered vs. not"), is inferred.

In these preferential looking experiments, looking time is a measure aggregated over many discrete behaviors of the infant, such as steady gazes towards a given point ("fixations"), which occur in between shifts between fixations ("saccades"). An infant's gaze is thus constantly moving at a much faster timescale than the one generally reported in habituation studies (Bronson, 1990). This micro-level looking behavior (the order, length, etc.) of fixations is interesting because it gives more insight into the mechanisms that are giving rise to the aggregate looking times. They also are informative for building artificial systems to mimic desirable capabilities of the human infants (e.g. developmental learning ability).

Even in the absence of habituation (e.g. with exceedingly simple stimuli such that habituation is instantaneous, or in darkness, or with certain brain areas lesioned) infants and primates demonstrate a visual search behavior. With habituation removed, when aggregated over time, the looking times for stimuli will be proportional to properties of the stimuli such as complexity or salience. It seems there is some intrinsic visuomotor mechanism/dynamics in place running in a constant loop which ensures that gaze will be allocated around the visual environment in a way that is roughly proportional to (probably low-level) properties of the components of the visual environment. Theories have been suggested (regarding e.g. minimizing uncertainty, entropy, learning rate, etc.) as to "why" this might be the case (evolutionarily), but this is not the question addressed in this paper. This paper rather attempts to build a (robotic) model of what this visuomotor system might look like neurally, and how it functions to produce the looking behavior observed in infants. Habituation's interaction with the visuomotor system is addressed, but a model of habituation is not explicitly built nor tested in this paper. The focus is entirely on the "inside loop" of the system, i.e. the loop which keeps the eyes moving around the visual environment, fixating on components of the environment in turns roughly proportional to their properties.

The model presented is a "prototype" model containing the "large" pieces of a more complex model of the same phenomenon that is exhaustively based on neuroanatomical evidence. The present model for the sake of prototyping on the robot and demonstration of concept takes some shortcuts anatomically. It does not, however, take shortcuts mechanistically – all mechanisms are accomplished via biologically realistic continuous neural circuit models, and the shortcuts can all be demonstratably implemented using more complex circuit models.

# 2 Target Behavior

The target behavior for the model will be to match fixation times of very young human infants (less than 2 months postnatal, preferably newborns). The overall looking behavior should be matched qualitatively (i.e. the infant should not only look perfectly between the two most salient objects, it should also look away at e.g. random wall point for some short period of time every now and then). In addition, the proportion of looking time should increase as a function of the stimulus complexity (Lewis, Kagan, & Kalafat, 1966). This function will be simply linear in this model, but as implemented it can be exchanged for any function of the complexity. Thus, it should be matchable to actual infant looking data for e.g. some set of objects. "Stimulus complexity" in this paper and model are just a placeholder for some unknown properties of visual stimuli that draw or hold looking in infants (those properties that experimenters try to control for between stimuli).

## 3 Model Description

Instead of exhaustively enumerating the neuroanatomical, psychophysical, and neuropysiological support for the various components, the (prototype) model will be laid out, and the

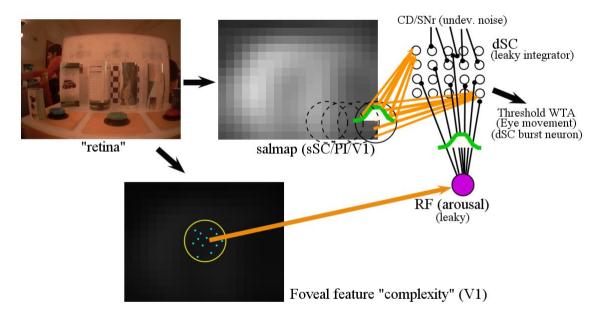


Figure 1: Graphical overview of connectivity and regions of network, and the general function of each region. Full-model anatomical correlates listed next to regions.

salient support presented briefly for the various pieces' functions, maturity, connectivity, and role in producing the behavior.

The "architecture" (i.e. implementation-biased description) of the model is laid out, rather than the mathematical formalism of the environment and solution. No analysis of the network dynamics is attempted in this paper except for informal observations of the behavior of the network in the robotic infant while it was performing.

The network is made up of several main pieces (Figure 1). The overall theory of its functionality is illustrated in Figure 2.

## 3.1 Saliency Map

Images (frames) come in (actually, one from each eye, left and right) and are put through a saliency map ((Itti, Koch, & Niebur, 1998)), which assigns a salience value to each location in the image by looking for areas of high global "uniqueness" at many spatial scales. This is accomplished by applying filters at many scales in parallel to detect feature channels such as motion, color, intensity, orientation, etc, and then subtracting and subsampling between sequential spatial scales, within channels. This is meant to represent some sort of "fast scene processing" salience, which does not care so much about the content at visual locations so much as the "salience" of locations especially in relation to the rest of the visual field. It is imagined that this type of saliency map would be used in orienting behavior, which is of course exactly what the looking behavior in this paper is about. Some of the saliency channels (color, orientation) are not ones that would be processed by the superficial layers of the superior colliculus (SC)(mostly responds to movement), nor is there evidence for

(explicit) spatial scales even in V1, so it is not clear where a salience map would actually be implemented, especially in an infant.

#### 3.2 Superior Colliculus - deep (integration, eye control)

The instantaneous salience map calculated for every frame is treated as "input" into a longer-term map made up of leaky integrating neurons (non-firing). This is considered to correspond to the intermediate/deep layers of SC, especially since this is the last "stop" before eye movements in our model. Each neurons membrane potential  $V_m$  is described by the equation:

$$\frac{\partial V_m}{\partial t} = \frac{-(V_m - V_{rest}) + R_m \cdot (I_{bg} + I_{syn})}{\tau_m} \tag{1}$$

where  $R_m$  is the membrane resistance (uniformly 1.0 M $\Omega$  for all neurons),  $I_{bg}$  the background current (uniformly 0.0 mV for all neurons) and  $I_{syn}$  the total current impinging from afferent synapses. The  $-V_m$  represents the leakage term, causing the membrane potential to decay exponentially with time constant  $\tau_m$  (uniformly 30 ms for all neurons, though since update was per-frame this has no relation to real-time). The resting potential of the membrane  $V_{rest}$  is assumed to be 0 mV.

Afferent input into a dSC neuron from the instantaneous saliency map is simply linearly summed into  $I_{syn}$ , after being multiplied by the efficacy of the synapse connecting it. The synaptic weights and connections are built initially using a 2-dimensional gaussian, with standard deviation  $\omega_w$  (1.5 for the experiments) and a cutoff value (i.e. minimum weight outside of which region there are no connections)  $\chi$  (0.05 for experiments). This has the effect of slightly blurring (low-pass-filtering) the instantaneous salmap (thus bringing out "masses" of high salience more than just isolated pixels of high salience). It also mimics the increase in receptive field size one sees when moving more towards the dSC (many pre-synaptic neurons connect to fewer post-synaptic neurons, in retinotopically defined areas).

## 3.3 Reticular Formation (arousal response to complexity)

Physiological functions (arousal, measured by heart rate) have been shown to be correlated with attentional phases in infants (Richards & Casey, 1990). Some have hypothesized the underlying circuits for these include e.g. the reticular formation (RF) which is responsible for the ascending transport of neuromodulators (horomones) to diverse regions of the brain. Such areas could become more active in response to stimulating environments (i.e. with more complex stimuli, more variation, etc.) and are also responsible for the lack of responsiveness of infants without sufficient stimulation. Though, endogenous ebb and flow guide a large part of the very young infant's arousal/attentional state at any time.

The complexity-responsive functionality of the RF circuits are used as a mechanism for determining (soft) looking time towards an object. Really, it could be implemented via other things such as simply probabilistic microsaccades that are more likely to get "caught" in the more complex stimulus. Since usually the amount of looking time towards a stimulus during e.g. habituation experiments is postulated to be a function of "encoding" of the stimulus

(and thus, longer for more complex stimuli), yet we have explicitly lesioned those areas that are "making progress" in any sense (or learning), there must be some other mechanism than simply "learning" that drives looking time towards stimuli. Since in habituation experiments the salience etc. of stimuli is controlled, it is not easy to decouple the habituation response from the "baseline" stimulus response. Note also that more complex mechanisms, e.g. encoding into some sort of short-term memory, waiting for a non-transient response/cycle of some minimal error threshold in response to the stimulus, could be how this stimulus complexity causes longer looking times, but the current mechanism at least attempts to capture the phenomenon if not the mechanism. This is perhaps one of the least understood areas of oculomotor behavior – namely, in absense of any scene change, what causes the infant to look away? Several observations exist that correlate with the phenomenon, e.g. the dying out of fixation neurons in dSC, changes in activity in SNr, CD, and the buildup of burst neurons in dSC, but the real dynamics and mechanisms and how all these work together to move attention (fixation) are not well understood. This is one of the things this model and the more plausible one hope to offer explanations for.

In the model the RF is modeled as a single leaky neuron. One can think of it as population coding of the activity of some region that got a shot of neuromodulator/horomone cocktail in response to exciting foveal stimuli. The RF neuron inhibits every region of the dSC, so that more activation of the RF will result in more inhibition of SC (pushing the neurons there to very negative potentials), and thus it will take longer for SC neurons to recover via the normal continuous input coming from e.g. the instantaneous saliency map and the gaussian noise from SNr. However, to prevent the continuous fixation over multiple "refixations" of the central area (which might not be coded in mammals, i.e. we wouldn't have to worry about the currently foveated area ever getting burst neurons activated since there are no corresponding burst neurons there) the weighting of this inhibitory projection is topologically modulated, such that more foveal/central positions have stronger inhibitory weights than the periphery (which still have some baseline inhibitory weight).

Thus, the weight of a point in the retinotopic projection as a function of distance from the centre of the fovea is equal to the value at that point of a gaussian centred on the centre, with amplitude  $\alpha$  (6.0), variance  $\sigma$  (1.5), and baseline (i.e. y-shift)  $\beta$  (1.0).

When a gaze shift is made, the "complexity"  $c_f$  of the current foveal content is scaled and injected into the RF neuron (the RF neuron's  $V_m$  is set to  $c_f$ . In this case, as a standing, the complexity is calculated as the number of Harris feature points (thresholded 1-d gaussians LPFs run along the x- and y-dimensions) within a radius r of the centre of the image. In binocular cases the two eyes are simply summed together linearly.

The RF neuron's membrane potential  $(V_{RF})$  decays exponentially with some time constant  $\tau_{RF}$  (500ms, though again since in the experiments update of the saliency model is not locked to real time the units are arbitrary w.r.t. the experiments), i.e.

$$\frac{\partial V_{RF}}{\partial t} = \frac{-V_{RF}}{\tau_m} \tag{2}$$

The membrane potential of RF is injected directly into all (e.g. dSC) post-synaptic neurons, simply linearly scaled by the weight of the synapse connecting RF with that neuron.

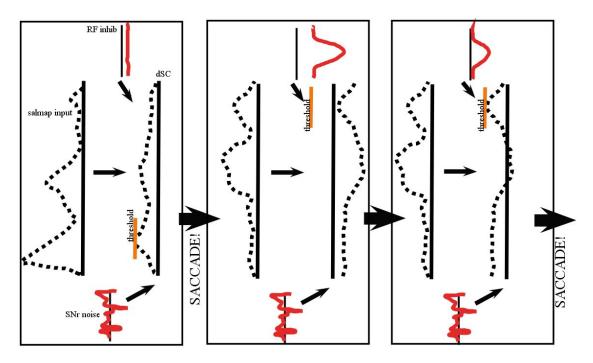


Figure 2: Intended loop dynamics of the model through several fixation-saccade iterations. Intended local circuit response properties listed where predictable.

### 3.4 Substantia Nigra pars reticulata (Gaussian noise)

Caudate Nucleus (CD) and SNr neurons show visual and habituation responses (Hikosaka & Wurtz, 1983), which implies they are at play in normal visual search behavior and/or habituation learning. Assuming it's just habituation or even considering they are engaged in disgengagement and thus normal visual looking behavior ((Johnson, 1990)), these disengagement and habituation behaviors are not seen in infants or in limbic-cortex lesioned primates, respectively. This suggests that the SNr itself, it's afferents, or its efferents to dSC are undeveloped in the young infants. SNr-dSC projections are known to be strongly GABAergic and thus inhibitory, and to receive inhibitory projections from CD ("dis-inhibitory", which in turns gets input from a variety of regions as well as having interesting intrinsic dynamics involving dopamine via SNc). We model these dynamics and the product of immaturity by simply considering the baseline of SC as already receiving some amount of inhibition from the tonically firing SNr GABAergic neurons, and then add or sutract gaussian noise (gaussian random variables) from the synaptic input to each dSC neuron on every time step. The probability of drawing an input of strength of  $\delta$  is probabilistically based on the mass of the gaussian at the point, thus small numbers are likely, and very far out numbers are less likely. In the experiments, the noise is drawn from a gaussian with  $\sigma$  of 0.01 and amplitude (scale of output inputs) of 1.0.



Figure 3: The iCub head that was used for the experiments.

#### 4 Method and Results

The prototype model was implemented on an iCub robot (Figure 3). It has two pan-tilt-vergence eyes mounted in a head supported by a yaw-pitch-twist neck (see picture). Eye movements were accomplished by way of a vergence-control mechanism which ran in a closed-loop fashion with visual input (this was not realistic, but necessary because of limited time and a lack of familiarity with the iCub and with binocular systems. It is not clear how the problem of vergence is solved in infants but it is clear that saccades are not closed loop or vision-driven but are ballistic and motor-driven (Hainline, 1998)). Since the model was turned off during "saccades" this had no effect on the collected data, though from a real-time point of view it was very slow compared with actual saccades (limitations of the motors). Coordinated movement of the head and eyes is likewise probably unrealistic in such young infants. The more complete model will need to account for eye movements while either avoiding or solving these non-trivial problems.

The robot instantiated the network and was placed in front of a scene. The amount of time spent looking at objects and the "complexity" of the object was recorded. The trajectory (i.e. ordering) of looking among the objects was recorded.

#### 4.1 Results

Results will go here... Based on runs on single frames (see testonppm.cpp) the decaying-inhibition dynamics of the model work as intended. To demonstrate this, see Figure 5, plotting the results of automated experiments with the model measuring the time between first fixation and second fixation as a function of the complexity of the foveal image (x-axis).

This is the desired behavior, and would result in (at least) fixation times that follow the desired model of fixation time. For the longer-term looking dynamics, however, since foveation of the salient stimulus is assumed, it's impossible to test the looking dynamics without running on the robot or in a simulation environment that moves the visual field.



Figure 4: A picture of the experimental setup that was used during the experiments. The type of environment presented can be seen in front of the iCub head in the upper-left corner.

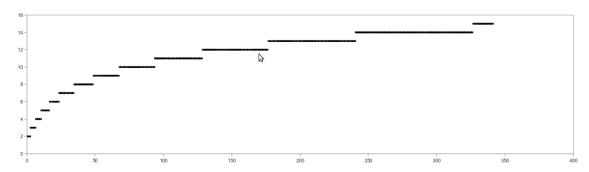


Figure 5: Plot of the time between fixations (i.e. fixation time to that foveal region) for varying levels of foveal complexity (the manipulated variable in these experiments). Note the logarithmic pattern that emerges: for very low complexities, fixation is disengaged quickly, and for slightly different complexities the looking time differs, but as complexity increases, the looking time saturates to a maximum.

## 5 Conclusion

By design, the mechanism to determine looking time towards a foveal stimulus as a function of its complexity was successful, but this is not the interesting result since these dynamics are predicted and necessitated by the simple mechanism.

The interesting bit will be the inter-object looking behavior in a real environment. Will, and how often will, the system make saccades to "noise" areas but then quickly look away because of lack of complexity?

In the future, the model will be implemented as the more complex anatomical circuit, the saliency map replaced with proper neural code from the eye-up, and the actual neurons "fudged" in most of the areas and their dynamics actually simulated. The benefit of building the more realistic model will be to actually look at the different dynamics of the model

in reaction to the changing environment, since that kind of interaction can not be easily predicted or simulated.

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