Motivational modulation of endogenous inputs to the superior colliculus

Jason Satel
Faculty of Computer Science
Dalhousie University
Halifax, NS B3H 1W5
E-mail: satel@cs.dal.ca

Thomas Trappenberg
Faculty of Computer Science
Dalhousie University
Halifax, NS B3H 1W5
E-mail: tt@cs.dal.ca

Raymond Klein
Department of Psychology
Dalhousie University
Halifax, NS B3H 4J1
E-mail: klein@dal.ca

***Abstract*— Proper initiation of saccadic eye movements depends on an intricate balance between exogenous and endogenous control mechanisms. The superior colliculus (SC) is a major site of signal integration that has been shown to drive the initiation of saccades in the brainstem. Previous work has shown that a winner-take-all mechanism implemented with a continuous attractor neural network (CANN) can explain and reproduce a multitude of behavioural findings, including the gap effect and the production of express saccades [1], [2]. This investigation advances the CANN model of saccade initiation in several important ways in order to account for trial by trial adaptation of saccadic reaction times in a biologically plausible manner. A key hypothesis is that endogenous inputs to the intermediate layer of the SC can be adapted through motivationally-based feedback from other areas of the brain such as the basal ganglia or higher cortical areas.**

I. Introduction

Rapidly directing gaze to targets in response to external cues such as unexpected noise is crucial for responding to potentially dangerous situations. However, reflexive behaviour under exogenous control can sometimes be undesirable so there must be a mechanism to balance this with voluntary, goal directed behaviour under endogenous control. The superior colliculus (SC) is a major site of signal integration [3], [4], [5] that drives the initiation of saccades in the brainstem [6], [7]. Kopecz and colleagues have shown how the dynamics of this integration, based on a continuous attractor neural network (CANN) model which implements a winner-take-all mechanism, can explain several behavioural findings, such as the gap effect [1], [8]. Our group has advanced this research by integrating the model with physiological findings of the SC and extending it to additional behavioural findings [2], [9]. In this paper we advance the model with a reward-based, adaptive control mechanism. This mechanism performs the necessary empirical tuning of parameters in the model and is consistent with experimental findings of trial-by-trial modulations of saccade reaction times [10], [11].

In the previous study we were able to adjust some parameters such as the interaction profile from physiological studies, while other parameters, such as the strength of input signals, had to be chosen empirically to account for the different behavioural findings. Here, we argue that the effective strength of information converging on the SC can be modulated to accomplish a better situation-based response of the oculomotor system. We demonstrate that a simple adaptation mechanism based on reward signals can account for some behavioural findings, in particular the motivationally-dependent balance between exogenously driven express saccades and endogenously driven regular saccades. In particular, we give a mechanistic explanation of findings similar to the ones by Juttner and Wolf [11] that, following a catch trial, saccade reaction times (SRTs) are increased and the probability of an exogenously driven saccade is reduced. We also extend the simulations to an antisaccade task [12], [13] that can demonstrate related effects more clearly.

II. Biological background

The SC receives convergent input from a variety of subcortical and cortical areas. While the SC is a multimodal integration area [14], here we concentrate entirely on visual processing. There is a direct pathway from the retina to the intermediate layer of the SC, as well as two main cortico-collicular pathways along which saccade related activity can be recorded [15]. The first cortico-collicular pathway proceeds through the frontal eye field (FEF) [16], [17], supplementary eye field (SEF) [18], dorsolateral prefrontal cortex (DLPF) [19], and the lateral intraparietal area (LIP) [20], while the more indirect, second pathway proceeds through the FEF, SEF and DLPF to the caudate nucleus (CD) [21], [22] and then the substantia nigra pars reticulata (SNr) [23], [24] before converging on the SC [15]. Furthermore, inputs to the intermediate layer of the SC from other areas such as the oculomotor thalamus (OcTh) [25], posterior cingulate cortex (CGp) [26], orbitofrontal cortex (OFC) [27], and the cerebellum [28] all contribute to the initiation of saccadic eye movements. The SC itself projects to the brainstem saccade generator which in turn directly drives the eye muscles [6], [7].

In behavioural terms it is useful to divide input to the SC into two categories, namely exogenous signals that are mainly driven by direct, only marginally processed visual signals and endogenous signals that are highly processed cortical inputs that might include instructional processing leading to more voluntary control of eye movements [29]. Based on physiological findings, we assume that the exogenous input is mainly driven by the direct pathway with a time delay

of about 70 ms after target presentation. This delay can be approximately broken down into 20 ms for transduction in the retinal ganglion cells, 10 ms for transmission up to the striate cortex, an additional 20 ms to arrive at the SC, with only 20 ms left for processing [30]. A longer time delay of 120 ms is associated with endogenous input, which presumably is due to an additional 50 ms of transmission and processing time in higher cortical areas. We further assume that the time course of exogenous signals are mainly transient, while endogenous input could be maintained longer if necessary [1], presumably based on working memory. The spatial distribution of signals on the SC map is presumed to be location specific with input to the rostral pole effecting fixation related activity and peripheral input effecting saccades to specific eye directions [31].

The strengths of the various input signals were empirically adjusted in our previous work to account for a variety of behavioural findings [2]. In this paper, we study the situation-based alterations of the signal strength in a systematic way. Exogenous signal strength is thereby kept constant as the form of reward-based adaptation discussed in this paper is likely not effected in the direct pathway. However, the endogenous signal strength to the SC is altered in a systematic way. It is possible that this signal strength alteration is supported cortically, for example through attentional, intentional, and working memory mechanisms. However, we specifically discuss the strength modulation in terms of reward mechanisms, in which the basal ganglia has been implicated [32], [33]. For example, Hikosaka's group has elegantly demonstrated the rapid changes of caudate responses after changing rewards to different saccade targets [34], [15]. Houk and colleagues [33] have described an intriguing possibility for the role of the basal ganglia in the reward-based learning of motor actions, and it has been demonstrated [35] that this scheme is consistent with the findings of Kawagoe et al. [34]. Direct output from the substantia nigra to the SC does indicate that such reward-based mechanisms can influence saccade programming. In the model studied in this paper we use a simplified generic reward adaptation scheme to illustrate the basic consequences of reward-based modulations of endogenous inputs to the SC.

III. The Model

A. Dynamics of the superior colliculus

The basic model of the intermediate layer of the superior colliculus is based on the neural-field model with lateral inhibition studied by Amari [36] which can function as a winner-take-all network (see [37] for a review). The implementation in this paper is unchanged from our previous implementation [2]. Namely, it consists of a recurrent network in which the firing rate $r_i(t)$ of a node i is given by

$$\tau \frac{du_i}{dt} = -u_i(t) + \sum_j w_{ij} A_j(t) \Delta x + I_i^{in}(t) - u_0 + \eta \quad (1)$$

$$r_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)}, \quad (2)$$

where τ is a time constant, w_{ij} represents the synaptic efficiency (weight) from node i to node j, Δx is a scaling factor, $I_i^{in}(t)$ describes the external input to the system, η is a noise term introduced to represent stochastic processing in the system, u_0 is a global constant set to zero except for burst nodes during active fixation, when it is set to 100, and β and θ are parameters of the sigmoidal gain function. If a node's firing rate reaches 80% of maximum, then a signal is sent to the brainstem, initiating a saccade. The parameters used in the following simulations are summarized in Table 1.

Category	Parameters
Architecture	N = 501; Δt = 1 ms
SC dynamics	$\tau = 10$ ms; $u_0 = 0$ (buildup)
	$u_0 = 100$ (burst during fixation)
Transfer function	$\beta = 0.07$; $\theta = 0$
Weight matrix	$a = 180$; $b = 60$; $\sigma_a = 0.6$ mm; $\sigma_b = 3\sigma_a$
Noise η	Normal distributed random variable $N(1, x)$

TABLE I
PARAMETERS OF THE MODEL

The weight matrix w_{ij} describes the lateral interaction in the collicular map, and their center-surround form is parameterized as a difference of two Gaussians

$$w_{ij} = a * exp(\frac{-(j-i)^2}{2\sigma_a^2} + b * exp(\frac{-(j-i)^2}{2\sigma_b^2}) - c \quad (3)$$

The parameters of this profile have been adjusted to fit physiological data from a distracter experiment in monkeys [2]. A similar interaction profile was used by Kopecz [1] and was also found by Arai et al. [38] when training a network based on electrical recordings of movement fields in monkeys. A graphical illustration of the interaction profile is shown in Figure 2.

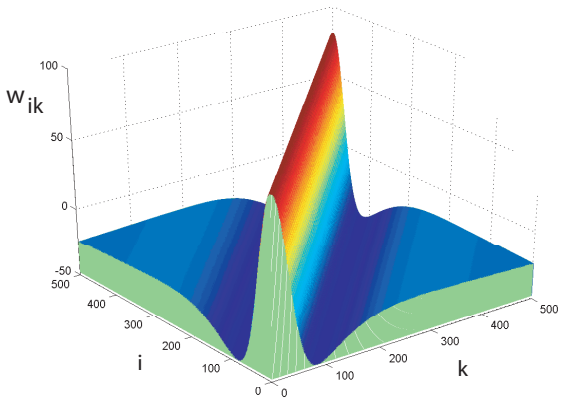

Fig. 1. Graphical illustration of the Mexican Hat-type lateral interaction profile in the collicular map. The center-surround form of this weight profile is parameterized as a difference of two Gaussians.

The input signals are modeled using a Gaussian spatial shape, that is, the external input to a node i in the network is given by

$$I_i = a * exp(\frac{(k-i)^2}{2\sigma^2}) \quad (4)$$

when a visual cue is centered around location k. The parameter a corresponds to the strength of the input signal. The strength of exogenous inputs were kept constant in all simulations, reflecting the relatively direct pathway from the retinal ganglion cells to the SC. Target appearance was modeled as an exogenous input at the target location with a strength value of 60, presented 70 ms after target onset. Removal of the fixation point and the corresponding off-set effect was modeled as an exogenous input to the center node with a strength value of -10, presented 70 ms after fixation point removal.

Endogenous inputs include location specific anticipation (a_s), presented to the anticipated target node, global fixation disengagement (a_g), presented to the center node, and the move signal. The move signal was kept constant in all simulations, with a strength of 10, and was presented 120 ms after target presentation. Location specific anticipation and fixation disengagement were independently modulated in the different simulations as described below, and were presented to the system 120 ms after fixation point removal.

B. Reward adaptation of endogenous signals

In typical saccade experiments with monkeys, the animals are rewarded with a small amount of liquid after each correct trial. Kawagoe et al. [34] found that saccades to rewarded targets are faster than saccades to non-rewarded targets even though the task requires saccades to all targets. They also found that cell activity in the basal ganglia reflected a change in reward contingencies soon after the change occurred (within one or two trials). We captured these findings by a small increase of anticipation after a correct trial, either by an increase of endogenous input at the target location (local effect) or an increase in the endogenous fixation disengagement (global effect), which is equivalent to a reduction of fixation activity after fixation offset.

$$a_{\{s,g\}} = a_{\{s,g\}(previous-trial)} + 0.1. \tag{5}$$

Juttner and Wolf [11] found that the probability of express saccades following a incorrect saccade is highly reduced. We capture this finding by a large decrease of endogenous anticipation (either local or global) after an incorrect saccade

$$a_{\{s,g\}} = a_{\{s,g\}(previous-trial)} - 2. \tag{6}$$

An analogy of this algorithm is the following: Some people (not including the authors) tend to increase their driving speed on highways over time until they are caught by police. The fine is typically sufficient to slow down the drivers for a while, though the average speed is typically increasing again over time.

It is possible to model the reward machinery in much more detail. For example, the involvement of the basal ganglia in reward-based learning of motor responses has been widely discussed in recent years [32], [39] and some detailed models have been developed [33], [35], [40], [41], [42]. While the involvement of the basal ganglia in modulating saccade activities is likely given that the substantia nigra is projecting to the SC, it is also possible that anticipatory signals are modulated in other brain areas. We are here more interested in exploring the consequences of the alterations of endogenous anticipation, and the simple algorithm specified above is sufficient to outline some experiments that could help investigate the important question of global versus local anticipation effects.

IV. Simulations

A number of simulations were performed in order to test the idea that the strength of endogenous input signals to the SC can be modulated based on local reward optimization after each trial.

A. Simulations of pro-saccades

A basic saccade experiment is the gap paradigm in which saccades are made to one of two possible targets that appear randomly with equal probability following fixation point removal. A saccade reaction time (SRT) distribution of a simulation of this paradigm is shown in Figure 2A. In this simulation we used a global fixation strength of $a_g = 7$ and allowed the location specific anticipation to change up to a maximum value of $a_s = 3$. This maximum was reached after only a few trials because no catch trials are included that would penalize express saccades. SRT distributions from a monkey performing an equivalent gap experiment are shown in Figure 2B (data courtesy of Stefan Everling; see also Tinsley and Everling [43]).

This simulation illustrates the idea that express saccades are driven by exogenous input, whereas regular saccades are under endogenous control. The cap on the strength of endogenous anticipation input has been chosen in this simulation so that fluctuations produced SRT distributions comparable to the monkey data. Thus, the strength used in this simulation can be interpreted as a maximal possible strength although this value is different for different values of a_g.

B. Simulations of pro-saccades with no-go trials

To study the effects of penalizing express saccades we propose a slightly modified experiment that includes no-go trials in a go/no-go paradigm. For example, two different targets such as different symbols or cues with different colors could indicate if a saccade should be made or not. Our adaptation scheme does predict that the presence of such no-go trials will suppress express saccades in direct proportion to the percentage of no-go trials. The SRT distribution of such a simulation with 20% no-go trials using a global adaptation algorithm is shown in Figure 2C. Note that using a location specific adaptation algorithm results in a nearly identical distribution, motivating our analysis of the differences between using these two different algorithms in the following section.

Similar findings of reduced express saccades and increased SRTs with catch trials and a reduced probability of express saccades after a catch trial was reported by Juttner and Wolf [11]. However, the catch trials in their experiment were an absence of any stimulus so that a saccade was triggered even without exogenous input. Our model does not include such purely anticipatory saccades, which is why we used a modified go/no-go paradigm rather a typical catch trial paradigm.

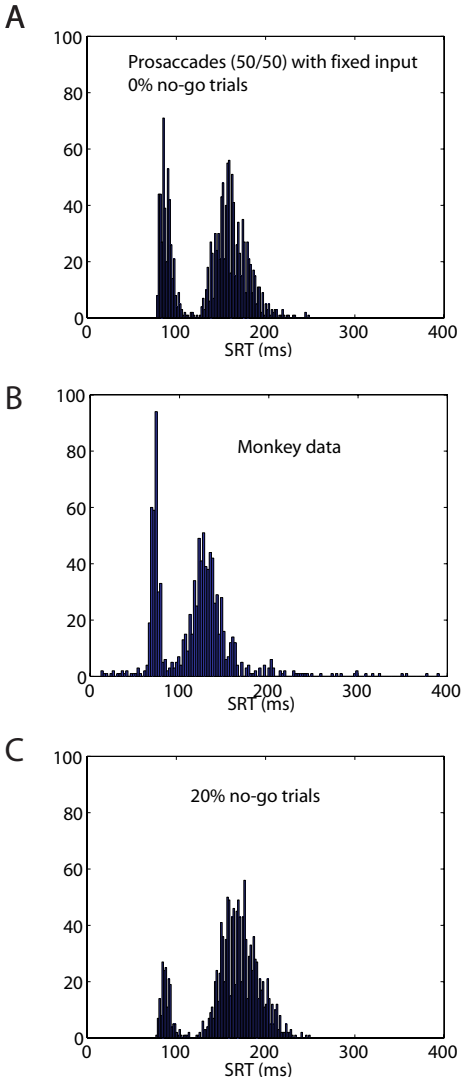

Fig. 2. SRT distribution in 200 ms gap paradigm. (A) Monkey data from pro-saccade experiments. (B) Model without no-go trials but with a cap on the maximal endogenous anticipation. (C) Model with 20% no-go trials.

C. Global versus location specific adaptation

To illustrate more generally the effect of specific modulations of the global (a_g) or location specific (a_s) endogenous signal strength we show in Figure 3 SRTs in a simulation without noise in which we vary only one specific input strength while keeping all others fixed. The solid lines in this figure represent SRTs to a high probability target, while the dashed lines represent SRTs to a low probability target on the opposite side of the visual field. In Figure 3A we set the location specific anticipation to a high probability target to $a_s = 7$ (solid line) and the location specific anticipation to an alternative low probability target to $a_s = 1$ (dashed line) while altering the global fixation disengagement (a_g). The SRTs of both targets are affected by altering a_g with larger values for the fixation disengagement resulting in lower SRTs. However, the effect in the high probability site is larger as the SRTs to this site are shorter and an increase of the fixation disengagement leads to a transition to the regime of express saccades. The adaptation mechanism is designed to find the transition point when express saccades are punished sufficiently.

In Figure 3B we set the global fixation disengagement to $a_g = -3$ and the location specific anticipation to one target $a_s = 1$ (dashed line) while varying systematically a_s to the other target. The SRTs agree for equal a_s to both targets, but the SRTs will decrease for increasing the anticipation of the target corresponding to the solid line and also transition to the express saccade domain. As expected, SRTs to the other target are not much affected. However, for large values of a_s there is a slight increase in SRTs as this strong anticipation level will produce some inhibition of the target corresponding to the dashed line.

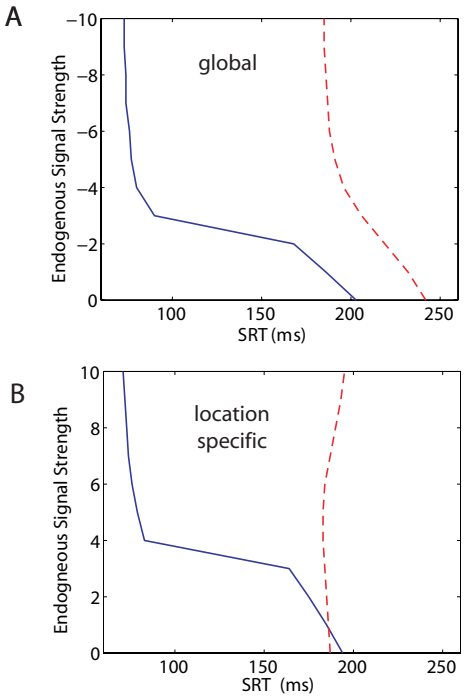

Fig. 3. SRT as a function of altering an endogenous input while keeping the other fixed. (A) Exogenous input to the high probability side (solid line) is set to $a_s = 7$ and to the low probability side (dashed line) to $a_s = 1$, while the endogenous fixation disengagement a_g is varied from $a_g = 0$ to -10. (B) Endogenous fixation disengagement input is fixed to $a_g = -3$ and the endogenous anticipation is fixed to $a_s = 1$ on one side (dashed line) while the other side (solid) was varied from $a_s = 0$ to 10.

D. Simulations of anti-saccades

A useful paradigm to explore the integration of exogenous and endogenous processing is an anti-saccade paradigm where a saccade is to be made to the opposite location of target presentation. Thus, correct saccades are completely under endogenous control so any express saccades to the target location would be deemed an incorrect response resulting in no reward being given. There is therefore no need to include catch or no-go trials in this paradigm.

We performed several simulations with an anti-saccade task. In these experiments we altered the target probabilities of the two possible target locations which was either 50/50 or 30/70. These experiments were run with the two distinct adaptation protocols, either a global protocol or a location specific protocol as outlined above. Both algorithms were able to produce SRTs as found in experimental studies [12], [13].

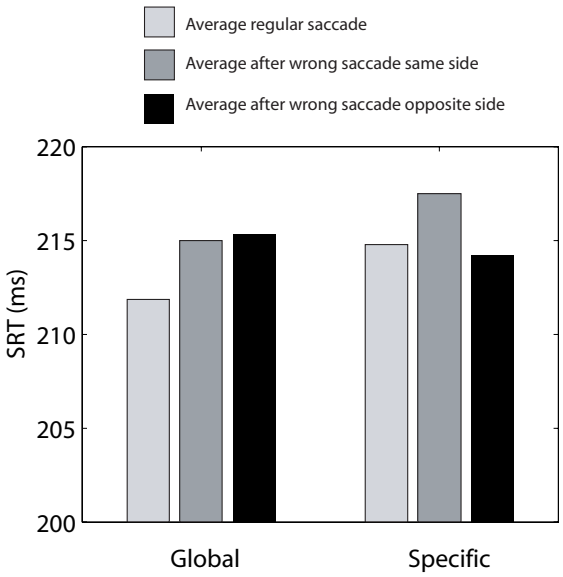

Fig. 4. Mean SRTs in an anti-saccade paradigm to illustrate global versus location specific modulation of endogenous inputs. In the global protocol SRTs are increased to both sides after an incorrectly performed express saccade. With location specific anticipation, however, SRTs are only increased to the same side as the error was performed. Saccades to the opposite side are actually decreased after an incorrect saccade.

To further illustrate the differences between a global protocol or a location specific protocol, the mean SRTs found in each protocol are compared in Figure 4. In the location specific protocol, we fix $a_g = -3$ while allowing a_s to be independently modulated according to the previously stated adaptation algorithm. In the global protocol, we fix $a_s = 3.5$ on each side, while allowing a_g to be modulated according to the previously stated adaptation algorithm. Global modulation results in a similar increase to mean SRT on the trials immediately after an error has been produced to both the same and opposite side on which the error occurred. Location specific modulation, however, results in a slight decrease to mean SRT on the opposite side as the error occurred, but an increase to mean SRT on the same side as the error occurred, as illustrated in Figure 4. Note that these effects are very small and so are very difficult to see when noise is added to the simulation. Thus, the stochastic nature of neural systems would make it very difficult to reproduce the effect experimentally.

V. Conclusions

We have previously suggested that express saccades are fast saccades that are elicited by exogenous input to the SC. In this investigation we explored further consequences of this suggestion. That is, an increase in saccade anticipation, either through a global fixation disengagement or a location specific anticipation should increase the probability of express saccades. We demonstrated this consequence in various simulations. In particular, we find that the fraction of express saccades should depend on the percentage of no-go trials in a go/no-go paradigm, a behavioural experiment that can be easily implemented.

These findings parallel the findings of Juttner and Wolf [11]. However, the catch trials in their experiments are an absence of targets that nevertheless trigger saccades to the anticipated targets. Such purely anticipatory saccades have not been studied here. It is possible to get these results with larger levels of location specific anticipation, but this would drive the model into a regime where no regular saccades are made with exogenous inputs. While buildup neurons in the SC do show a systematic activation proportional to anticipations, this activation would have to be considerably larger than has been seen in cell recordings. Future work should analyze cell activity in the SC under such conditions to further investigate such purely anticipatory saccades.

We also highlighted the behavioural consequences of modulating global versus location specific endogenous signals. Location specific anticipation could readily simulate well studied effects such as inhibition of return (IOR) and is thus worth examining further since the mechanisms of IOR are still to a large extent unknown. Behavioural experiments corresponding to the simulations in this paper should be very straight forward.

Finally, the precise mechanisms of motivational effects on the SC should be explored further. We hypothesize that motivational modulation of endogenous inputs occurs after every trial. This is a form of extremely short-term neural plasticity leading to a local optimization of reward [10]. The possible involvement of the basal ganglia can be explored with more specific simulations and contrasted with other possible sources of such modulations of endogenous inputs to the SC.

Acknowledgment

TT would like to thank Stefan Everling for useful discussions and for providing behavioural monkey data. This work was supported in part by the NSERC grant RGPIN 249885-03.

References

[1] K. Kopecz, "Saccadic reaction times in gap/overlap paradigms: A model based on integration of intentional and visual information on neural, dynamic fields," *Vision Research*, vol. 35, no. 20, pp. 2911–2925, 1995.

[2] T. P. Trappenberg, M. C. Dorris, D. P. Munoz, and R. M. Klein, "A model of saccade initiation based on the competitive integration of exogenous and endogenous signals in the superior colliculus," *Journal of Cognitive Neuroscience*, vol. 13, no. 2, pp. 256–271, 2001.

[3] M. C. Dorris, M. Pare, and D. P. Munoz, "Neuronal activity in monkey superior colliculus related to the initiation of saccadic eye movements," *Journal of Neuroscience*, vol. 17, no. 21, pp. 8566–8579, 1997.

[4] J. A. Edelman and E. L. Keller, "Activity of visuomotor burst neurons in the superior colliculus accompanying express saccades," *Journal of Neurophysiology*, vol. 76, no. 2, pp. 908–926, 1996.

[5] P. H. Schiller, J. H. Sandell, and J. H. Maunsell, "The effect of frontal eye and superior colliculus lesions on saccadic latencies in the rhesus monkey," *Journal of Neurophysiology*, vol. 57, no. 4, pp. 1033–1050, 1987.

[6] C. A. Scudder, C. R. Kaneko, and A. F. Fuchs, "The brainstem burst generator for saccadic eye movements: A modern synthesis," *Experimental Brain Research*, vol. 142, pp. 439–462, 2002.

[7] G. Gancarz and S. Grossberg, "A neural model of the saccade generator in the reticular formation," *Neural Networks*, vol. 11, pp. 1159–1174, 1998.

[8] K. Kopecz and G. Schoner, "Saccadic motor planning by integrating visual information and pre-information on neural dynamic fields," *Biological Cybernetics*, vol. 73, pp. 49–60, 1995.

[9] T. P. Trappenberg, S. Simpson, R. M. Klein, D. Munoz, M. C. Dorris, and P. McMullen, "Neural field model of oculomotor preparation and disengagement," in *Proceedings of the 1997 IEEE International Conference on Neural Networks (ICNN'97)*, vol. 1, 1997, pp. 591–595.

[10] M. C. Dorris, M. Pare, and D. P. Munoz, "Immediate neural plasticity shapes motor performance," *Journal of Neuroscience*, vol. 20, pp. 1–5, 2000.

[11] M. Juttner and W. Wolf, "Occurence of human express saccades depends on stimulus uncertainty and stimulus sequence," *Experimental Brain Research*, vol. 89, pp. 678–681, 1992.

[12] S. Everling, M. C. Dorris, R. M. Klein, and D. P. Munoz, "Role of primate superior colliculus in preparation and execution of anti-saccades and pro-saccades," *Journal of Neuroscience*, vol. 19, no. 7, pp. 2740–2754, 1999.

[13] B. Fischer, S. Gezeck, and K. Hartnegg, "On the production and correction of involuntary prosaccades in a gap antisaccade task," *Vision Research*, vol. 40, pp. 2211–2217, 2000.

[14] M. Wallace and B. Stein, "Onset of cross-modal synthesis in the neonatal superior colliculus is gated by the development of cortical influences," *Journal of Neurophysiology*, vol. 83, no. 6, pp. 3578–3582, 2000.

[15] K. Watanabe, J. Lauwereyns, and O. Hikosaka, "Effects of motivational conflicts on visually elicited saccades in monkeys," *Experimental Brain Research*, vol. 152, pp. 361–367, 2003.

[16] S. Everling and D. P. Munoz, "Neuronal correlates for preparatory set associated with pro-saccades and anti-saccades in the primate frontal eye field," *Journal of Neuroscience*, vol. 20, no. 1, pp. 387–400, 2000.

[17] D. P. Hanes and R. H. Wurtz, "Interaction of the frontal eye field and superior colliculus for saccade generation," *Journal of Neurophysiology*, vol. 85, pp. 804–815, 2001.

[18] N. Amador, M. Schlag-Rey, and J. Schlag, "Reward-predicting and reward-detecting neuronal activity in the primate supplementary eye field," *Journal of Neurophysiology*, vol. 84, pp. 2166–2170, 2000.

[19] J. D. Wallis and E. K. Miller, "Neuronal activity in primate dorsolateral and orbital prefrontal cortex during performance of a reward preference task," *European Journal of Neuroscience*, vol. 18, pp. 2069–2081, 2003.

[20] M. Pare and R. H. Wurtz, "Progression in neuronal processing for saccadic eye movements from parietal cortex area lip to superior colliculus," *Journal of Neurophysiology*, vol. 85, no. 6, pp. 2545–2562, 2001.

[21] O. Hikosaka, M. Sakamoto, and S. Usui, "Functional properties of monkey caudate neurons iii. activities related to expectation of target and reward," *Journal of Neurophysiology*, vol. 61, no. 4, pp. 814–832, 1989.

[22] R. Kawagoe, Y. Takikawa, and O. Hikosaka, "Reward-predicting activity of dopamine and caudate neurons - a possible mechanism of motivational control of saccadic eye movement," *Journal of Neurophysiology*, vol. 91, pp. 1013–1024, 2004.

[23] A. Handel and P. W. Glimcher, "Quantitative analysis of substantia nigra pars reticulata activity during a visually guided saccade task," *Journal of Neurophysiology*, vol. 82, pp. 3458–3475, 1999.

[24] M. Sato and O. Hikosaka, "Role of primate substantia nigra pars reticulata in reward-oriented saccadic eye movement," *Journal of Neuroscience*, vol. 22, no. 6, pp. 2363–2373, 2002.

[25] M. T. Wyder, D. P. Massoglia, and T. R. Stanford, "Quantitative assessment of the timing and tuning of visual-related, saccade-related, and delay period activity in primate central thalamus," *Journal of Neurophysiology*, vol. 90, pp. 2029–2052, 2003.

[26] A. N. McCoy, J. C. Crowley, G. Haghighian, H. L. Dean, and M. L. Platt, "Saccade reward signals in the posterior cingulate cortex," *Neuron*, vol. 40, pp. 1031–1040, 2003.

[27] L. Tremblay and W. Schultz, "Reward-related neuronal activity during go-nogo task performance in primate orbitofrontal cortex," *Journal of Neurophysiology*, vol. 83, pp. 1864–1876, 2000.

[28] P. Lefevre, C. Quaia, and L. M. Optican, "Distributed model of control of saccades by superior colliculus and cerebellum," *Neural Networks*, vol. 11, pp. 1175–1190, 1998.

[29] R. M. Klein and D. I. Shore, "Relationships among modes of visual orienting," in *Attention and Performance: XVIII. Control of Cognitive Processes*, S. Monsell and J. Driver, Eds. MIT Press, 1997, pp. 195–208.

[30] B. Fischer and H. Weber, "Express saccades and visual attention," *Behavioral and Brain Sciences*, vol. 16, pp. 553–610, 1993.

[31] J. A. M. Van Gisbergen, A. J. Van Opstal, and A. A. M. Tax, "Collicular ensemble coding of saccades based on vector summation," *Neuroscience*, vol. 21, pp. 541–555, 1987.

[32] W. Schultz, "Responses of midbrain dopamine neurons to behavioral trigger stimuli in the monkey," *Journal of Neurophysiology*, vol. 56, pp. 1439–1462, 1986.

[33] J. C. Houk, J. L. Adams, and A. G. Barto, "A model of how the basal ganglia generate and use neural signals that predict reinforcement," in *Models of information processing in the basal ganglia*, J. C. Houk, J. L. Davis, and D. G. Beiser, Eds. MIT Press, 1995, pp. 249–270.

[34] R. Kawagoe, Y. Takikawa, and O. Hikosaka, "Expectation of reward modulates cognitive signals in the basal ganglia," *Nature Neuroscience*, vol. 1, no. 5, pp. 411–416, 1998.

[35] T. P. Trappenberg, H. Nakahara, and O. Hikosaka, "Modeling reward dependent activity pattern of caudate neurons," in *Proceedings of the International Conference on Artificial Neural Networks (ICANN'98)*, 1998.

[36] S. Amari, "Dynamics of pattern formation in lateral-inhibition type neural fields," *Biological Cybernetics*, vol. 27, pp. 77–87, 1977.

[37] T. P. Trappenberg, "Continuous attractor neural networks," in *Recent Developments in Biologically Inspired Computing*, L. N. de Castro and F. J. V. Zuben, Eds. ., 2003.

[38] K. Arai, E. L. Keller, and J. A. Edelman, "A spatio-temporal neural network model of saccade generation," in *Proceedings of the IEEE International Conference on Neural Networks*, 1993, pp. 70–74.

[39] J. O'Doherty, P. Dayan, K. Friston, H. Critchley, and R. Dolan, "Temporal difference learning model accounts for responses in human ventral striatum and orbitofrontal cortex during pavlovian appetitive learning," *Neuron*, vol. 38, pp. 329–337, 2003.

[40] P. Dayan and B. W. Balleine, "Reward, motivation and reinforcement learning," *Neuron*, vol. 36, pp. 285–298, 2002.

[41] F. Worgotter and B. Porr, "Temporal sequence learning, prediction and control - a review of different models and their relation to biological mechanisms," *Neural Computation*, vol. 17, pp. 1–75, 2004.

[42] P. R. Montague, S. E. Hyman, and J. D. Cohen, "Computational roles for dopamine in behavioural control," *Nature*, vol. 431, pp. 760–767, 2004.

[43] C. Tinsley and S. Everling, "Contribution of the primate prefrontal cortex to the gap effect," *Progressive Brain Research*, vol. 140, pp. 61–72, 2002.