Bimodal saccadic latency distributions can be generated by reward-based adaptation of endogenous inputs to the superior colliculus.

July 7, 1999

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Abstract

Saccadic reaction times (SRTs) are typically distributed unimodally when subjects are asked to move their eyes from a central fixation to a target as quick as possible after its appearance. If, however, the fixation cue disappears shortly before target onset, then SRT is reduced, and the reduction is often accompanied by splitting of the unimodal distribution into two or more modes. The fastest mode of these rapid eye movements has been termed 'express saccades'. The origin of these fast saccades is under considerable discussion. In this paper we show that bimodal distributions with a fast peak of express saccades can have their origin in the way information is processed in the intermediate layer of the superior colliculus.

Keywords: saccade, oculomotor, express, endogenous preparation, reward adaptation
1. Introduction

Primates scan a visual scene with rapid eye movements called saccades. The saccade reaction time (SRT) is defined as the time a subject needs to initiate a saccade when instructed to move his eyes from a central fixation to a target as fast as possible after the target appears. Typically SRTs are distributed around mean values in the range of 200ms in humans; monkeys are typically slightly faster than human subjects. SRTs are reduced when the fixation is removed prior to the target appearance in what has become to be known as the gap effect (Saslow, 1967; Reuter-Lorenz, Hughes & Fendrich, 1991; Kingston & Klein, 1993). In addition, Fischer and colleagues (Fisher & Boch, 1983; Fisher & Ramsberger, 1984) discovered that some subjects display bimodal SRT, and some-times even trimodal, distributions in the gap paradigm (see also Schiller, Sandell & Maunsell, 1987). The fast components of these distributions are often termed "express" as they have much shorter SRTs compared to those of the slower modes representing regular saccades (Saslow, 1967; Fisher & Ramsberger, 1984; Schiler et al., 1987; Fischer & Weber, 1993; Kingston & Klein, 1993; Parè & Munoz, 1996).

The origin of express saccades has been under considerable discussion. Fisher (1987) (see also Fischer & Weber, 1993) argued that there have to be different processing pathways, and he proposed a simple loop model to explain the observed data. The proposed scheme of his model is that each pathway has a different processing time and that the winning pathway leading to the initiation of the saccade is selected randomly due to some noise in the process. In this paper we propose a different functional explanation for the origin of bimodal SRT distributions which does not require different processing pathways. Indeed, we show that the mechanisms realized within the intermediate layer of the Superior Colliculus (SC), a midbrain area instrumental in the generation of eye movements, can lead to bimodal distributions and to express saccades when endogenous input to this structure is manipulated to optimize reward. This is demonstrated with simulations of a simple yet powerful model of the SC.

A proposed principle role of the intermediate layer of the SC that is captured by our model is that of the dynamic integration of multiple inputs via inter- and intra-collicular interactions. Such a model was first proposed by Kopecz (1995) and Kopecz & Schöner (1995) to describe some behavioral effects related to saccadic eye movements. We have recently advanced this model in several important ways and were able to reproduce a variety of behavioral SRT effects as well as corresponding typical discharge pattern of neurons in the intermediate layer of the SC in a variety of well studied oculomotor phenomenon such as the gap/overlap effect, distractor influences, antisaccades, and the effects of target probabilities (Trappeenberg et al., 1999). This model also realizes, with an explicit functional and physiological faithful quantitative implementation, the competitive inhibition (integrative layer, level 2) of the schematic model proposed by Findlay and Walker (1999). We outline our model in the next section before we discuss in this paper in more detail how the mechanisms in the SC can generate bimodal SRT distributions.

Express saccades depend strongly on training (Fisher & Ramsberger, 1986). Jüttner and Wolf (1992) have shown that the probability of an express saccade decreases as the percentage of catch trials increases. They also found that the SRTs of saccades
following a catch trial are longer, and the probability of an express saccade lower, than following a trial with a target. To our knowledge, however, no suggestion for the functional basis of these findings has been proposed. In this paper we show how the strength of endogenous input to the SC can be adjusted adaptively to maximize reward monkeys would receive in typical saccade experiments. We also show that such an adaptation does explain the occurrence of bimodal distributions as well as the experimental findings of Jüttner and Wolf (1992). Finally, our framework can also be used to provide an explanation for the frequent failure to observe bimodality in untrained human subjects (e.g. Kingstone & Klein, 1993; Reuter-Lorenz et al., 1991).

2. A ’neural-field’ model of the SC

Munoz and Wurtz (1995) have classified saccade-related neurons in the intermediate layer of the SC into fixation, buildup, and burst neurons based on their discharge characteristics. The structure of our model of this midbrain structure is outlined in Figure 1. The central nodes represent fixation neurons (white) in the rostral pole of the SC, whereas peripheral nodes represent buildup neurons (gray) and burst neurons (black) of the left and right colliculus respectively. All these neurons are modeled with nodes having average firing rates

\[ A_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \theta)} \quad (1) \]

\[ u_i(t) \] simulates thereby the average membrane potential of neuron \( i \), with a dynamic described by

\[ \tau \frac{du}{dt} = -u_i(t) + \sum_j w_{ij} A_j(t) + I^e_i(t) - u_0 + a_i \eta \quad (2) \]

The parameters \( \tau \) is a thereby a time constant, \( w_i \) is the synaptic efficacy (weight) from neuron \( i \) to neuron \( j \), and \( I^e_i(t) \) describes the input from other non-collicular areas onto this cell assembly. The value of the global constant \( u_0 \) is the only difference between burst nodes and buildup/fixation nodes. This constant is set to zero for buildup nodes, whereas the burst nodes receive a strong global inhibition during active fixation, which ceases only after the discharge of buildup nodes reach a certain threshold. The random variable \( \eta \), which we introduced to simulate fluctuations in the biological processes, is taken to be normally distributed, and is adjusted with the strength value \( a_\eta \). The noise term is included to represent possible stochastic processing in the motor layer itself as well as possible variability in the input signals.

The intermediate layer of the SC receives afferents from a multitude of cortical and subcortical visual and cognitive centers. Behavioral studies (Klein, Kingstone & Pontefract, 1992; Taylor, Kingstone & Klein, 1998) have shown that the sources of information driving the initiation of saccades can be categorized into two conceptually defined classes: exogenous that refers to visual inputs, and endogenous that refers to voluntary inputs which are dependent on instruction. Our model is driven by these two input streams as outlined in Figure 1. The position and the time of onset (offset) of the model stimuli depend on the experimental conditions. Both types of input are modeled using a Gaussian spatial shape with a width derived from movement fields of the cells of the monkey’s SC (Munoz & Wurtz, 1995). The precise spatial form of the input is not critical for the findings in this paper because information is also spreading laterally within the SC through the effective pathways therein. The inputs differ in the temporal domain. The exogenous input taken to
follow the onset of a visual stimulus with a 70ms delay (taken from cell behavior of monkeys (Trappenberg et al., 1999)) and decays thereafter. In contrast, endogenous target-related input requires interpretation by mechanisms outside the oculomotor system and reaches the SC only after a 120ms delay. The time course is simply assumed to be constant during the time it is applied. A central feature of our model is the form of lateral interaction captured with the weight matrix $w_{ij}$ in Equation 2. Experimental studies (see Munoz & Istvan, 1998; and references therein) have revealed evidence for short distance excitation and long distance inhibitory connections within each colliculus and between colliculi. A similar interaction profile was also found by Arai, Keller & Edelman (1995) after training a recurrent network using spatio-temporal data from cell recordings of the SC in monkeys. We have devised a distractor experiment to probe the effective interaction structure within the SC (Trappenberg et al., 1999) and found that the following parameterization described the data adequately,

$$w_{ij} = a \exp\left(-\frac{(j-i)^2}{2\sigma_i^2}\right) + b \exp\left(-\frac{(j-i)^2}{2\sigma_j^2}\right) - c \quad (3)$$

Values for the parameters were thereby chosen so that the simulated distractor effect resembled the findings of the cell recording (Trappenberg et al., 1999). The parameters of the model together with the values used in the simulations of this paper are summarized in Table 1.

Models with such an interaction structure are often simply termed 'neural field models' and have been studied by Amari (1977). We use this 1-dimensional model throughout the simulations of this paper. However, the model can easily be extended to higher dimensions, and it has been shown that the general properties of such models do generalize to higher dimensions (Konen, Maurer, van der Malsburg, 1994; Taylor, 1999). Similar models have also been employed to describe direction sensitive receptive fields in the visual cortex (see for example Usher et al., 1996; Jancke et al., 1999), and to model the Cortex-NRT-Thalamus loop (Taylor & Alavi, 1995, 1997). We therefore believe that the mechanism of competitive integration as described in this paper is likely a widely used information processing principle in the brain.

3. Simulation results

In the gap paradigm the central fixation cue is removed before the target appears. In Figure 2A we display the typical behavior of a buildup node in this paradigm. The solid line represents the average firing rate in the case in which we have turned off the noise term in order to display more clearly the average waveform of the nodes. The dashed line represents one trial of a simulation that includes noise. The fixation is removed at $t=0$ms after which the buildup nodes display a typical buildup of activity during the gap interval. A transient visual peak follows the onset of the target at $t=200$ms, which is in turn followed by a motor peak initiating the saccade. This behavior of our model nodes captures well the average waveforms of buildup neurons found in recordings of monkeys SC (Trappenberg et al., 1999).

The global inhibition of burst neurons is removed in our model by the time buildup activity reaches a certain threshold that is set to 80% of the theoretical maximal discharge rate of the buildup nodes in our simulations. The rapid increase of burst activity will subsequently initiate a saccade. The endogenous inputs, both central and peripheral, have thereby to be chosen accordingly so that the transient visual signal of the target does not already initiate a saccade. Nevertheless, the values have to be kept
in a reasonable range to allow subsequent initiation of saccades after target related endogenous signals reach the SC. Consequently, it is possible that noise in the system, either from the processing within the SC or from fluctuations of the incoming signals, can lead to a initiation of the saccade by the transient exogenously driven activity associated with target onset. This can in particular be expected when the central endogenous fixation is relatively weak and/or the location specific preparation is relatively large.

An example trial where fluctuations were strong enough so that the visual peak was able to elicit the saccade is shown in Figure 2B. The saccade was thereby initiated before endogenous target-related information was able to reach the SC. This results in the merger of the visual and motor peak. Histograms with SRTs from 500 trial are shown in Figure 3 for three different gap intervals. In the step condition (gap interval of 0ms, see Figure 3A) the distribution is unimodal with SRTs around 240ms. However, for larger gap intervals (Figure 3B and 3C) bimodal distributions are clearly visible. Figure 3B shows simulation results for a gap interval of 100ms. The slower mode, which corresponds to regular saccades, is distributed around 200ms demonstrating the gap effect. The faster mode with SRTs distributed around 130ms, corresponds well with express saccades. The ratio of express saccades increases when the gap interval is further increased to 200ms (Figure 3C). These results compare well with the experimental findings (Schiller et al., 1987; Fischer & Weber, 1993). The same pattern is observed when the number of trials is decreased to 100.

4. Adaptation

In the previous simulations we have adjusted the strength of the various exogenous and endogenous inputs appropriately to achieve bimodal distributions. An obvious question is then how nature achieves this fine-tuning. In this section we demonstrate that such levels of endogenous input signals are indeed expected when the strength values of endogenous inputs to the SC are adjusted to maximize the reward monkeys receive in typical saccade experiments. This approach is, in particular, desirable - as it is known that the basal ganglia has a strong influence on the initiation of saccades and is in turn suggested to be instrumental in reward learning (Houk, Adams & Barto, 1995; Trappenberg, Nakahara & Hikosaka, 1998).

As mentioned above, the exogenous input is thought to reach the SC without or only indirect (e.g. via the thalamo-cortical loop) cortical processing. It is therefore a reasonable first approximation to keep the strength values of this input constant as in the previous simulations. On the other hand, the strength of endogenous input is likely to vary considerably with the motivation of the subject (Kawagoe, Takikawa & Hikosaka, 1998). In our model we can distinguish at least three endogenous signals which could have different origins and could therefore be altered independently:

1. Endogenous fixation: This is the input to the rostral pole. In the simulations in (Trappenberg et al., 1999) and in the previous simulations in this paper we have reduced the fixation activity during the gap period simulating the assumption of a reduced endogenous fixation strength without visual guidance. Alternatively, this strength can stay constant, which we will adopt in the following.
2. Location specific anticipation: This is the endogenous peripheral input during the gap interval.
3. Move Signal: This is the final endogenous input at the location of the desired target location initiating regular saccades.

The strength of the move signal will be kept constant as before in the following simulations. However, we outline a simple scheme to manipulate either the strength of location specific anticipation, which we call \( a_{LSA} \), or, alternatively, the strength of the endogenous fixation, which we call \( a_{EF} \), to maximize reward.

In typical saccade experiments with monkeys, monkeys are given a fixed amount of liquid after each correct saccade to a target. In the following we simulate experiments where a new trial starts 500ms after a saccade was initiated. The reward over time that the monkey receives in a session of trials is therefore proportional to \( 1/(SRT+500\text{ms}) \). It follows that the monkey receives more reward when reducing SRTs. This can be achieved by reducing endogenous fixation (\( a_{EF} \)) and/or increasing the location specific anticipation (\( a_{LSA} \)). When the increase in \( a_{LSA} \) or the decrease in \( a_{EF} \) is large enough then express saccades will begin to occur. In the following experiment we included, randomly, 20% catch trials in which the monkey is not rewarded when any saccades (which would be “false alarms” on such trials) occur.

In the following simulations we realize the above considerations by the manipulation of endogenous input with the following two alternative schemes:

1. \( a_{LSA} \)-adaptation: If the reward is larger then the reward in the previous trial, then increase \( a_{LSA} \) by 0.1 until a maximum of 10 is reached. If the reward is less than the reward of the previous trial reduce \( a_{LSA} \) by 2.
2. \( a_{EF} \)-adaptation: If the reward is larger then the reward in the previous trial, then decrease \( a_{EF} \) by 0.1. If the reward is less than the reward of the previous trial increase \( a_{EF} \) by 2 until the maximum of 10 is reached.

Simulations with \( a_{LSA} \)-adaptation are shown in Figure 4A-C. The strength \( a_{LSA} \) is increased until a false express saccade is encountered for which no reward is given. At this point the level is reduced to allow endogenous control of the target location. The trials do therefore consist of regular as well as express saccades even without noise in the system (Figure 4A). However, additional noise does smooth the distribution (Figure 4B). Similar results were also achieved with the \( a_{EF} \)-adaptation. The resulting SRT distribution is shown in Figure 4D.

As can be seen from Figure 4A, the fastest regular saccades can be initiated in 158ms which correspond to an average reward of \( r=0.00152 \). Express saccades are initiated in around 81ms which correspond to a reward of \( r=0.00172 \) if a correct express saccade is made. However, with 20% catch trials express saccades give only an average reward of \( r=0.00138 \), less than with regular saccades. These three levels are indicated as horizontal dashed lines in Figure 5.

In addition we show in Figure 5 the average cumulative reward (solid line) and the average reward over the last 11 trial (dotted line) from the simulations corresponding to Figure 4C. As can be seen, the reward can locally exceed the optimal global reward. The reason for this is that the reward maximization scheme outlined here is
local in the sense, that only reward information from the previous trial is used. This is also the reason behind the fact that express saccades occur in these simulations despite the fact that the optimal global reward can only be achieved without express saccades. However, the average reward over the last 900 trial was \( r=0.00146(40) \), which is only slightly less than the optimal reward of \( r=0.00152 \). If the number of catch trials is reduced, more express saccades can be expected. This is indeed also seen in the simulations. In Figure 4C we show the SRT distribution corresponding to simulations with the \( a_{LSA} \)-adaptation scheme in which we have included only 10% catch trials (compare to Figure 4B with 20% catch trials).

Our simulation results are consistent with the experimental findings of Jüttner & Wolf (1992) who showed that the relative number of express saccades is reduced with increasing number of catch trials. They also showed that the SRTs of saccades following catch trials have the tendency to be longer than those following regular saccades. Similar to their result we show in Figure 6 the distribution of SRTs following catch trials. In agreement with their results we find that the likelihood of express saccades after a catch trial is small so that the average SRT of those trials is longer than the average SRTs of trials following target trials. This is of course also evident from the adaptation procedure. It is likely that more details of the adaptation mechanisms as realized in primates can be revealed from the time course of SRTs in experiments similar to those of Jüttner & Wolf (1992).

5 Discussion

We propose a possible origin of bimodal SRT distribution and express saccades from a relatively simple mechanism implemented in the SC. We argue that fluctuations in the SC could sometimes enable the exogenous signals to elicit a saccade before endogenous signals can reach the SC. This parallels the motor preparation hypothesis for express saccade generation (Parè & Munoz, 1996; Dorris, Parè & Munoz, 1997), which suggests, that increased buildup activity in advance of target presentation may allow the visual burst to surpass the threshold for eliciting a saccade. If there is not enough buildup activity in advance of target presentation the visual burst does not surpass this threshold and a subsequent motor burst must occur later in time (Dorris, Parè & Munoz, 1997). The proposed framework leading to express saccades predicts that the magnitude of splitting in SRT distribution when express saccades are present should be proportional to the difference between the visual peak and the motor peak in the behavior of neurons in the SC. This can be tested experimentally.

The proposed framework depends critically on the level of endogenous fixation and target specific anticipation. It is possible that these levels can be adjusted voluntarily by each individual which might explain both, the dependence of express modes on the training of the task as well as individual differences in that some subjects do show express saccades bimodality whereas some individuals don’t. We have shown that local optimization of reward can lead to express saccades. This does depend critically on the experimental setup. If no catch trials are included, express saccades would be dominant. This is indeed what is often found in monkey experiments. We showed that the number of catch trials does influence the ratio of express to regular saccades which is consistent with the findings of Jüttner & Wolf (1992). We strongly believe that more detailed experiments can reveal even more details of the adaptation process as realized in primates.
The reward conditions described in this paper lead to an average reward over long times when only regular saccades are made which is greater than average rewards from trials including express saccades. It seems, therefore, that a strategy of global reward optimization should lead to the omission of express saccades. However this would require more advanced optimization procedures which might be more complicated to implement in neuronal networks. Nevertheless, some subjects might be able to find the globally more rewarding mode of avoiding express saccades. This might explain why human subjects are less likely to make express saccades than monkeys. In addition, human subjects are often instructed to make only correct saccades or are conservative by nature. This would also favor the suppression of express saccades.

Acknowledgments
We would like to thank Mike Dorris and Douglas Munoz for useful discussions.
References


### Tables

<table>
<thead>
<tr>
<th>Category</th>
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<td>SC dynamics</td>
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<td>Transfer function</td>
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<td>Weight matrix</td>
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Table 1: Parameters of the model and their associated values used in the simulation of this paper.
Figure 1: Outline of the model of the intermediate layer of the SC with fixation nodes (white) in the rostral pole, peripheral buildup nodes (gray), and burst nodes (black).
Figure 2: Solid line: Modeled average firing rate of a buildup node without noise. Dashed line: Average firing rate in trials with noise for a regular (A) and express (B) saccade.
Figure 3: Histograms of SRTs in 500 simulated trials with a gap interval of (A) 0ms, (B) 100ms and (C) 200ms.
Figure 4:
SRT distributions from simulations with adaptation of strength of endogenous information. In all cases bimodal distributions are observed. In the simulations (A-C) only the strength of the location specific endogenous preparation during the gap interval was manipulated with the adaptation procedure to maximize reward. Simulation (A) did not include noise, whereas all other simulation include noise. (D) SRTs from simulations in which only the strength of endogenous fixation was adapted instead of the location specific endogenous preparation. Both adaption procedures lead to similar results. The simulations in (A), (B), and (D) contained 20% catch trials, whereas only 10% catch trials were included in (C).
Figure 5:
The cumulative average reward (solid line) and the average reward over the last 11 trials (dotted line) in the simulations corresponding to Figure 4B. The three horizontal dashed lines correspond to the reward of a correct express saccade ($r=0.00172$), the reward of the fastest regular saccade ($r=0.00152$), and the average reward of express saccades with 20% non-rewarded catch trials ($r=0.00138$).
Figure 6:
SRT distributions of all the trials following a catch trial from the experiments shown in figure 4B. The likelihood of express saccades following catch trials is small.