Bimodal saccade distributions and the origin of express saccades

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Abstract

Saccadic reaction times (SRTs) are typically distributed unimodally when subjects are ask 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 have been termed 'express saccades'. The origin of these fast modes are under considerable discussion. In this paper we show that bimodal distributions with a fast peak of express saccades can have there origin in the way information is processed in the intermediate layer of the superior colliculus.

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; monkeys are 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 [1,2]. In addition, Fischer and colleagues [3,4] discovered that some subjects display bimodal SRT, and sometimes even trimodal, distributions in the gap Paradigm (see also [5]). The fast components of these distributions are often termed "express" as they have much shorter SRTs compared to those of the longer modes representing regular saccades [2]-[7].

The origin of express saccades has since been under considerable discussions. Fisher [8] (see also [6]) 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),

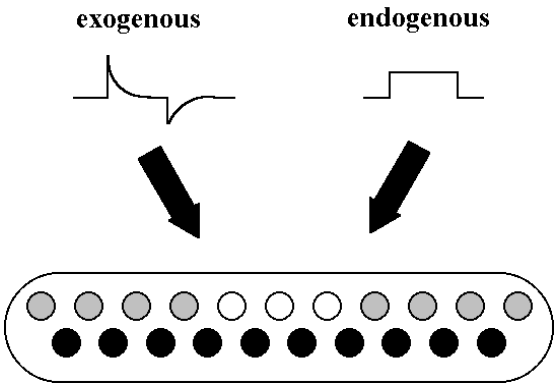

Figure 1: Outline of the model of the intermediate layer of the SC with fixation nodes (white) in the rostral pole, peripheral buildup nodes (grey), and burst nodes (black).

a midbrain area instrumental in the generation of eye movements, can lead to bimodal distributions and to express saccades. 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 and Schöner [9,10] 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 [11]. This model is described in the next section before we outline the mechanisms leading to express saccades in Section 3 and some implications of our modeling in Section 4.

2 A 'neural-field' model of the SC

Munoz and Wurtz [12] 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 (grey) and burst neurons (black) of the left and right colliculus respectively. All these neurons are modeled by nodes with average firing rates

$$A_i(t) = \frac{1}{1 + \exp(-\beta u_i(t) + \Theta)} \tag{1}$$

derived from the the average membrane potential given by

$$\tau \frac{du_i}{dt} = -u_i(t) + \sum_j \omega_{i,j} A_j(t) + I_i^{in}(t) - u_0 + a_\eta \eta. \tag{2}$$

The parameters τ is a thereby a time constant, w_{ij} is the synaptic efficacy (weight) from neuron i to neuron j, and I^{in} 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

Table 1: Parameters of the model with values used in the simulations.

Category	Parameters
Architecture	$N = 1001; \delta t = 1$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; $\sigma_b = 3\sigma_a$

ceases only after the discharge of buildup nodes reach a certain threshold. The random variable η, which we introduced to simulate fluctuations in the biological processes is taken to be normally distributed and adjusted with the strength a_η. 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 have shown [13,14] 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 [12]. 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 [11]) 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 [15] 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 et al. [16] 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 [11] and found that the following parameterization described the data adequately,

$$\omega_{i,j} = ae^{-(j-i)^2/2\sigma_a^2} + be^{-(j-i)^2/2\sigma_b^2} - c. \tag{3}$$

Values for the parameters were thereby chosen so that the simulated distractor effect resembled the findings of the cell recording [11]. 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 [17]. 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 (18,19). Similar models have also been employed to describe direction sensitive receptive fields in the visual cortex (see for example [20,21], and to model the Cortex-NRT-Thalamus loop [22]. 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 which includes noise. The fixation is removed at $t = 0$ 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 [11].

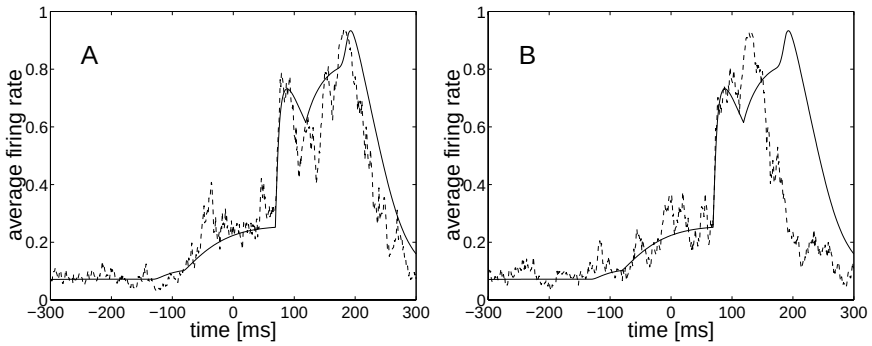

Figure 2: Solid line: Modeled average firing rate of a buildup node without noise. Dashed line: Average firing rate in trials with noise for a regular (A) and express (B) saccade.

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 relative weak and/or the location specific preparation is relatively large.

An example trial where fluctuations were strong enough to 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, which is sometimes even more pronounced than displayed here.

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 was further increased to 200ms (Figure 3C). These results compare well with the experimental findings [6].

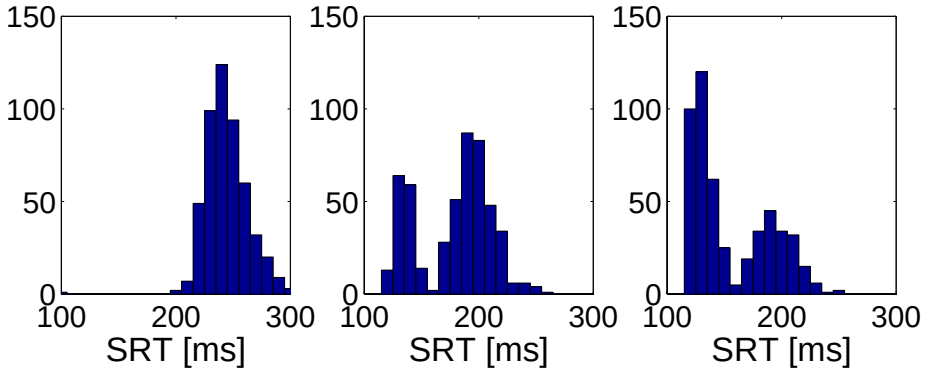

Figure 3: Histograms of SRTs in 500 simulated trials with a gap interval of (A) 0ms, (B) 100ms and (C) 200ms. The same pattern is observed when the number of trials is decreased to 100.

4 Discussion

We propose a possible origin of bimodal SRT distribution and express saccades from a 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 [23,24] 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 [24].

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 preparation. It is well possible that these levels can be adjusted voluntary 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.

Acknowledgments

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References

[1]Saslow,M.G. (1967). Saccade latency and warning signals: Stimulus onset, offset and change as warning events. *Journal of the Optical Society of America* 57:1024-1029.

[2] Kingstone, A. & Klein, R.M. (1993). What are human express saccades? *Perception & Psychophysics* 54:260-273.

[3] Fischer, B., & Breitmeyer, B. (1984) Human express saccades: Extremely short reaction times of goal directed eye movements. *Experimental Brain Research* 57:191-195.

[4]Fischer, B. & Boch, R. (1983) Saccadic eye movements after extremely short reaction times in the monkey. *Brain Research* 260:21-26.

[5] Schiller, P.H., Sandell, J.H. & Maunsell, H.R. (1987) The effect of frontal eye field and su-

perior colliculus lesions on saccadic latencies in the rhesus monkey. *Journal of Neurophysiology* 57(4):1033-1049.

[6]Fischer, B. & Weber, H. (1993) Epress saccades and visual attention. *Behavioral and Brain Science* 16:553-610.

[7]M. Parè & D.P. Munoz (1996) Saccadic reaction time in the monkey: advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *Journal of Neurophysiology* 76:3666-3681.

[8] Fischer, B. (1987) The preparation of visual of visually guided saccades. *Reviews in Physiology, Biochemistry and Pharmacology* 106:1-35.

[9] Kopecz, K. (1995). Saccadic Reaction Time in Gap/Overlap Paradigm: a Model Based on Integration of Intentional and Visual Information on Neural, Dynamic Fields. *Vision Research* 35:2911-2925.

[10] Kopecz, K. & Schöner, G. (1995). Saccadic motor planning by integrating visual information and expectation on neural dynamic fields. *Biological Cybernetics* 73:49-60.

[11] Trappenberg, T.P., Dorris, M.C., Klein, R.M. & Munoz D.P. (1999) A model of saccade initiation based on the competitive integration of exogenous and endogenous signals in the superior colliculus, *to be submitted to Journal of Cognitive Neuroscience*.

[12] Munoz, D.P. & Wurtz, R.H. (1995). Saccade related activity in monkey superior colliculus. II. Characteristics of burst and buildup cells. *Journal of Neurophysiology* 73:2313-2333.

[13] Klein, R. M., Kingstone, A. & Pontefract, A. (1992) Orienting of visual attention. *In K. Rayner (Ed.), Eye Movements and Visual Cognition: Scene Perception and Reading*, New York: Springer Verlag, 46-67.

[14] Taylor,T. Kingstone, A. & Klein, RM. (1998) Visual offsets and oculomotor disengagement: Endogenous and exogenous contributions to the gap effect. *Canadian Journal of Experimental Psychology* 52:192-200.

[15] Munoz, D.P., Istvan, P.J. (1998) Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *Journal of Neurophysiology* 79:1193-209.

[16] Arai, K., Keller, E.L. & Edelman, J.A. (1995) Two-Dimensional Neural Network Model of the Primate Saccade System. *Neural Networks* 7:1115-1135.

[17] Amari, S. (1977) Dynamics of Pattern Formation in Lateral-Inhibition Type Neural Fields. *Biological Cybernetics* 27:77-87.

[18] Taylor, J.G. (1999) Neural Bubble Dynamics in Two Dimensions I: Foundations. *Biological Cybernetics*, in Press.

[19] Konen, W.K., Maurer, T. & von der Malsburg, Ch. (1994) A Fast Dynamic Link Matching Algorithm for Invariant Pattern Recognition. *Neural Networks* 7:1019-1030.

[20] Usher, M., Stemmler, M., Koch, Ch. & Olami, Z. (1996) Network Amplification of Fluctuations Causes High Spike Rate Variability, Fractal Firing Patterns and Oscillatory Local Field Potentials. *Neural Computation* 6:795-836.

[21] Jancke, D., Erlhagen, W., Dinse, H.R., Akhavan, M., Giese, M. & Schöner, G. (1999) Parametric representation of retinal location: Neural population dynamics and interaction in cat visual cortex. *Journal of Neuroscience*, under revision.

[22] Taylor, J.G. & Alavi, F.N. (1995), A global competitive neural network, Biological Cybernetics, 72, 233-248. (1997) A global competitive model for attention. *Neural Network World* 5:477-502.

[23] Parè, M. & Munoz, D.P. (1996) Saccadic reaction time in the monkey: advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *Journal of Neurophysiology* 76:3666-3681.

[24] Dorris, M.C., Paré, M. & Munoz, D.P. (1997) Neuronal Activity in Monkey Superior Colliculus Related to the Initiation of Saccadic Eye Movements. *Journal of Neuroscience* 17:8566–8579.