A Neural Model for the Adaptive Control of Saccadic Eye Movements

Sohrab Saeb, Cornelius Weber and Jochen Triesch

Abstract— **Several studies have suggested different cost functions to explain the kinematic characteristics of saccades. However, these studies do not present any neural implementation of the optimization procedure they use. Instead, they are based on optimal control theory approaches that provide a global analytical solution rather than a local adaptation scheme. In this study, we propose a model comprised of an open-loop neural controller and an adaptation unit. The neural controller receives the initial target position as input. The adaptation unit, which is the neural interpretation of a simple cost function, evaluates the optimality of this controller and induces weight changes in the controller via a local learning rule. Realistic saccades are obtained with the proposed model. We speculate that the superior colliculus and the cerebellum behave quite similar to our model's neural controller and adaptation unit.**

I. Introduction

THE relationship between the duration, peak velocity and the amplitude of saccadic eye movements is known as the "main sequence". The main sequence of saccades is stereotyped: The duration increases linearly with saccadic amplitude, and the peak velocity increases linearly for low amplitudes and then undergoes a soft saturation for larger amplitudes [1]. The velocity profiles of saccadic eye movements are smooth and symmetric for low saccadic amplitudes, while they become skewed for larger amplitudes [2].

The kinematic and dynamic characteristics of saccades are determined by the oculomotor plant properties and the neural control signals. Most studies consider a linear plant as a model of the oculomotor system, since a study by Van Opstal and colleagues [3] showed that it is not necessary to use a complicated plant model in order to obtain realistic saccades. Instead, an appropriate neural control signal is enough. This study exploited a second order linear oculomotor plant, and by utilizing the Fourier deconvolution method, calculated the necessary control signals to obtain a biologically realistic response from the plant. The general form of the optimal neural control signals achieved by this method is compatible with neuronal firing patterns observed in the medial superior temporal (MST) area of the cortex and the oculomotor neural integrator (NI) neurons of the brainstem [4][5]. This suggests that the choice of a linear plant is plausible for modeling the oculomotor system.

Sohrab Saeb, Cornelius Weber and Jochen Triesch are with the Frankfurt Institute for Advanced Studies (FIAS), Johann Wolfgang Goethe University, Frankfurt/Main, Germany (email: {saeb, cweber, triesch}@fias.uni-frankfurt.de).

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Much effort has been put forth in order to identify the optimality principles which underly the kinematic characteristics of saccades. To this end, several studies have suggested different cost functions. Early studies have proposed that the saccade trajectories are optimized in such a way that they minimize the time to reach the target [6]. This assumption alone, however, leads to a bang-bang control solution [7], for which the resulting velocity profiles are not biologically plausible [8].

Another suggestion was made by Harris and Wolpert by assuming that there exists additive white noise in the neural command, whose instantaneous power (variance) is proportional to the power of the command signal [9]. Based on this assumption, the variance of the eye response increases when one tries to decrease the saccadic duration by recruiting larger command signals. As a result, a trade-off emerges between the speed and the accuracy of saccades. The optimal solution to this trade-off is a trajectory that is biologically plausible [10].

Chen-Harris and colleagues extended the model of Harris and Wolpert by introducing an internal feedback which consists of two forward models [11]: A forward model of the oculomotor plant that predicts the state of the eye, and a forward model of the target motion that predicts the state of the target. This feedback is used to generate the neural control signal when the input is the target position. The feedforward controller is optimized in a similar way to the study of Harris and Wolpert [10], which requires re-optimization for every saccadic duration.

In a different approach, Kardamakis and Moschovakis exploited the minimal-effort principle in order to obtain an optimal control signal for coordinated saccadic eye and head movements [12]. In this way, the squared sum of the eye torque signals integrated over the movement period is minimized to obtain the optimal control signal. This approach achieves unimodal velocity profiles with shorter acceleration and longer deceleration phases, which is compatible with the main sequence characteristics.

Although the two latter approaches have been successful in explaining the kinematics of saccades in terms of satisfying some optimality criteria, they do not propose any neural implementation for the optimization procedure they use. In fact, the optimization procedures used by these approaches are based on Pontryagin's extremum principle, which requires boundary conditions at the initial and the final time of the saccadic movement, and provides a global analytical solution rather than a local adaptation procedure. It may be speculated that such a solution is the result of evolution, however,

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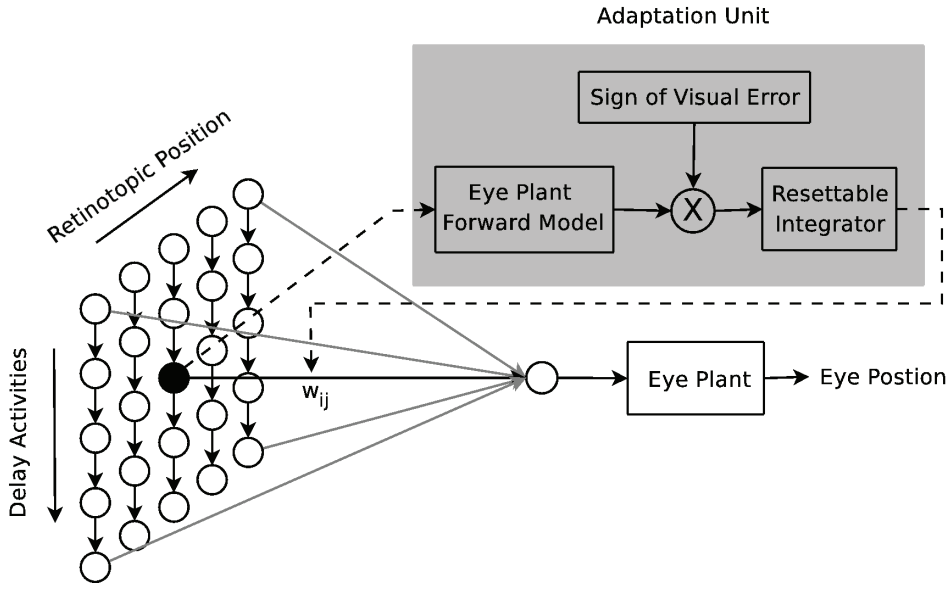

Fig. 1. Model Architecture. There is one delay line per retinotopic position of the target. The read-out neuron is linear and each weight parameter w_{ij} is adapted locally by the adaptation unit. The dashed lines account for the adaptation signals pathway.

numerous experimental results indicate that saccadic eye movements are constantly adapted [13].

In this study, we introduce a model based on an open-loop neural controller. We try to obtain an adaptation mechanism which on one side is local and biologically realistic, and on the other side minimizes a cost function. The neural controller receives the initial target position as the input and does not rely on the predicted states of the eye and the target in order to generate the neural control signal. The adaptation mechanism evaluates the optimality of the controller with respect to the cost function and induces parameter changes via a local learning rule.

II. Model Description

The model architecture consists of two pathways: "control" and "adaptation". The control pathway is a neural network comprised of a number of delay lines and one read-out neuron, as shown in Fig. 1. There is one delay line per target position. The activity of the neurons in delay lines is only dependent on the initial position of the target object in the scene and the progress of time. These activities are integrated by the read-out neuron to create the neural control signal that drives the oculomotor plant. The second pathway comprises the adaptation unit, the structure of which is derived from the adaptation rule (see Section II-C). It combines the information that is required to adjust the connection weights of the control pathway.

A. Open-loop Controller

The model perceives the retinotopic object position, i.e. the object position with respect to retina coordinates, which is expressed as the visual error, $r_{\text{vis}}(t)$. When a target object appears, depending on the initial value of $r_{\text{vis}}(t)$, the corresponding delay line is activated (Fig. 1). A saccade is initiated when the first neuron in a delay line starts firing. The next neurons in the delay line start firing in a successive manner. The firing pattern of each neuron is defined as a Gaussian function. As a result, a Gaussian wave of activity is propagated through the delay line. Given column j is activated at time $t = 0$, this propagation can be formalized as:

$$s_{ij}(t) = A \exp\left(-\frac{(i - \frac{t}{\Delta t})^2}{2\sigma^2}\right), \tag{1}$$

where $s_{ij}(t)$ represents the instantaneous firing rate of the neuron i in line j, Δt is the sampling period, σ^2 is the variance, and A scales the height of the activity peak.

The linear read-out neuron integrates the activity of the delay lines by means of weighted connections. This linear combination forms the neural command signal, $u(t)$, needed to drive the oculomotor plant:

$$u(t) = \sum_{j=1}^{N} \sum_{i=1}^{M} w_{ij} s_{ij}(t). \tag{2}$$

w_{ij} represents the weighted connection between neuron i in delay line j and the read-out neuron. N is the total number of delay lines and M is the number of neurons in each delay line. Since we allow the neural command $u(t)$ to become negative, we define it as the difference between the firing rates of the agonist and the antagonist motor neurons [14] driving the oculomotor plant.

The response of the plant is the eye position in head coordinates, $r_{\text{eye}}(t)$. It can be retrieved as the convolution between the neural control signal and the impulse response of the plant, $h(t)$:

$$r_{\text{eye}}(t) = \int_0^t u(\tau) h(t - \tau) d\tau, \tag{3}$$

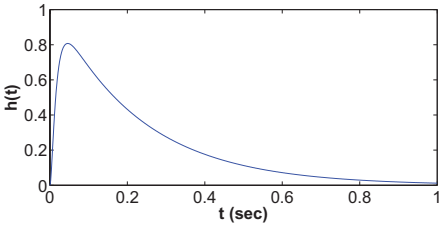

Fig. 2. The impulse response of the oculomotor plant, h(t).

where we assume $r_{\text{eye}}(0) = \dot{r}_{\text{eye}}(0) = 0$ as the initial condition.

The details of calculating the impulse response $h(t)$ are given in the Appendix. In this model, we use a linear 3-pole oculomotor plant with time constants $T_1 = 223\,ms$, $T_2 = 14\,ms$, and $T_3 = 4\,ms$, and the DC gain $K = 0.217$, according to [15]. These time constants are obtained from a mechanical model of the eye muscles that formulates the eye position, velocity and acceleration in terms of the neural activities stimulating motoneurons. The parameters of such mechanical models are estimated in such a way that the model's kinematic behavior matches the experimental data [16][17]. The impulse response $h(t)$ corresponding to these values is plotted in Fig. 2.

B. Cost Function

Assuming r_{obj} as the object position in head coordinates, we define the visual error as:

$$r_{\text{vis}}(t) = r_{\text{obj}} - r_{\text{eye}}(t). \tag{4}$$

We define a cost function that addresses the following objectives:

1) The gaze should reach the target as soon as possible and then stand still on the target position. Therefore, the cost function should depend on the absolute value of the visual error, $|r_{\text{vis}}(t)|$. This dependency can be established via any arbitrary function of $|r_{\text{vis}}(t)|$. Three examples are shown in Fig. 3. Convex functions such as the square function do not seem a good choice since they do not penalize small visual errors. We also do not choose concave functions such as the square root, but we will proceed with the absolute value function, because it results in more compatibility with neurophysiological observations, as we will see in Section IV.
2) The power of the neural control signal should be constrained. This assumption may be viewed as a regularization [18]. It also reflects the effect of signal-dependent noise [9], as it reduces the variability of the neural control signal by preventing its power from becoming too large. Since the neural control signal is linearly dependent on the weight values, the cost function should depend on the absolute values of the weight parameters. Thus, the large values of these parameters will be penalized regardless of their sign.

Accounting for these objectives, we define the cost function as:

$$E = \int_0^T |r_{\text{vis}}(t)|dt + k_{\text{reg}} \sum_{j=1}^{N} \sum_{i=1}^{M} |w_{ij}|^n, \tag{5}$$

where T has a sufficiently large value so that the integral covers the whole movement duration, and $k_{\text{reg}} \ll 1$ is a small positive coefficient, which determines the contribution of the weight limiting term to the total cost. We set $n = 4$ since this value leads to the results which have the most similarity to the experimental data.

It is worth noting that the integration duration T also covers part of the fixation period. This property of the cost function is in accordance with a study on humans [19] and another study on monkeys [20], which show that a delayed visual error signal, up to several hundred milliseconds, is still able to induce saccadic adaptation.

C. Adaptation

We use the gradient descent method for minimizing the cost function. To this end, we first calculate the partial derivative of the cost function with respect to each weight parameter:

$$\begin{aligned}
\frac{\partial E}{\partial w_{ij}} &= \int_0^T \frac{\partial}{\partial w_{ij}} |r_{\text{obj}} - r_{\text{eye}}(t)|dt + k_{\text{reg}} \frac{\partial}{\partial w_{ij}} \sum_{j=1}^{N} \sum_{i=1}^{M} w_{ij}^4 \\
&= \int_0^T (-\text{sign}(r_{\text{obj}} - r_{\text{eye}}(t))) \frac{\partial r_{\text{eye}}(t)}{\partial w_{ij}} dt + 4k_{\text{reg}} w_{ij}^3 \\
&= -\int_0^T \text{sign}(r_{\text{vis}}(t)) \frac{\partial r_{\text{eye}}(t)}{\partial w_{ij}} dt + 4k_{\text{reg}} w_{ij}^3,
\end{aligned} \tag{6}$$

where the function $\text{sign}(x)$ is defined as:

$$\text{sign}(x) = \begin{cases} -1 & \text{if} \quad x < 0; \\ 0 & \text{if} \quad x = 0; \\ +1 & \text{if} \quad x > 0. \end{cases}$$

Using (3) we can calculate the partial derivative of $r_{\text{eye}}(t)$ with respect to w_{ij}:

$$\begin{aligned}
\frac{\partial r_{\text{eye}}(t)}{\partial w_{ij}} &= \frac{\partial}{\partial w_{ij}} \int_0^t u(\tau) h(t-\tau) d\tau \\
&= \int_0^t \frac{\partial u(\tau)}{\partial w_{ij}} h(t-\tau) d\tau,
\end{aligned}$$

and since

$$\frac{\partial u(t)}{\partial w_{ij}} = \frac{\partial}{\partial w_{ij}} \sum_{j=1}^{N} \sum_{i=1}^{M} w_{ij} s_{ij}(t) = s_{ij}(t),$$

we obtain:

$$\frac{\partial r_{\text{eye}}(t)}{\partial w_{ij}} = \int_0^t s_{ij}(\tau) h(t-\tau) d\tau. \tag{7}$$

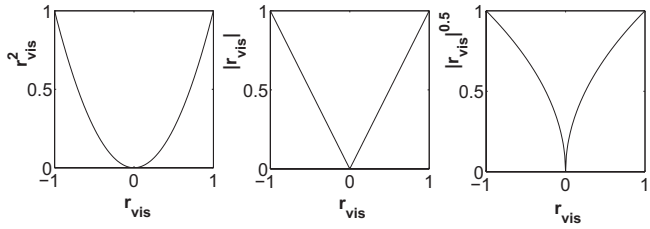

Fig. 3. The first term of the cost function can be any arbitrary function of $r_{\mathrm{vis}}(t)$. Three examples (square, absolute value and square root) are shown here.

By substituting (7) into (6), the final form of the error function gradient is calculated as:

$$\frac{\partial E}{\partial w_{ij}} = -\int_0^T \mathrm{sign}(r_{\mathrm{vis}}(t)) \left(\int_0^t s_{ij}(\tau) h(t-\tau) d\tau \right) dt + 4k_{\mathrm{reg}} w_{ij}^3. \quad (8)$$

Now that we obtained the gradients, the weight adaptation rule can be defined based on the gradient descent method as:

$$\Delta w_{ij} = -\delta_{ij} \frac{\partial E}{\partial w_{ij}}, \quad (9)$$

where Δw_{ij} represents the step change in w_{ij}, and δ_{ij} is the learning rate, which is adaptive (see Appendix).

The block diagram representation of the adaptation mechanism is shown in Fig. 1 (grey area). This representation is inspired by equations (8) and (9) in the following way: The inner integral of (8) suggests that there exists a forward model of the oculomotor plant inside the adaptation unit with the same impulse response as the oculomotor plant, which receives a copy of the neural activity in delay lines as input. The response of this forward model is modulated by the sign of the visual error and then integrated over duration T (see (8)). According to (9), the resulting signal acts on the same connection of the neuron that has stimulated the adaptation unit, which is shown by a dashed arrow in Fig. 1.

III. Results

We simulated our model using MATLAB. The simulation time step was set to 1 ms. After about 500 iterations of the adaptation procedure for each target position, the model reached a stable response. We set the simulation time T to 300 ms, which was enough for learning the saccades with amplitudes up to 80°.

We trained the model for target object positions from 1° to 19° with different values of σ and k_{reg}, and we compared the results to the experimental data obtained from an infrared eye tracker system tracking horizontal eye movements [15]. The parameter values $\sigma = 0.002$ and $k_{\mathrm{reg}} = 0.016$ led to the best match between the simulation and the experimental results (Fig. 4).

The neural control signals and the correponding plant responses for three target object positions, 10°, 20°, and 30°, are depicted in Fig. 5. The neural command signals comprise two main phases: The saccadic phase in which the neural command is large; and the fixation phase where the neural command has a roughly constant but slightly oscillating positive value. The mean value of the neural command in the fixation period is proportional to the target position, and the small oscillations lead to slight eye drifts which are negligible because of their low contribution to the cost function. In fact, the eye plant filters out the high frequency inputs so that the eyes do not follow these oscillations. The decrease of the firing rate at the end of the plot is a boundary effect. No matter how long the simulation time T is, this effect is always observed at the final time.

The general form of the optimized neural control signals shown in Fig. 5 resembles the firing patterns of burst-tonic neurons responsible for saccadic eye movements [5]. In both, a fast increase in the firing rate is followed by a slow decrease (the "burst" phase); then follows an oscillatory steady state that maintains the fixation period (the "tonic" phase). It is worth noting that the neural command becomes negative for a short time when the target position is 10°, which means the antagonist force is stronger than the agonist force in this period of time. Our model integrates the activity of the agonist and the antagonist muscles by introducing a single neural command that is allowed to become negative.

For small saccadic amplitudes, the velocity profiles are smooth and almost symmetric (Fig. 6-a), which is compatible with experimental results for these target positions [2]. However, as the figure shows, the symmetry of velocity profiles is not preserved when the amplitude is increased. This phenomenon is also observed in experimental studies (Fig. 6-b). The main reason for the former symmetry is that the effect of weight updating (9) on the saccadic velocity is symmetric when the second term of the cost function (5) is small enough. This effect becomes biased against large weights when the second weight regulating term grows as a result of an increase in target eccentricity.

IV. Discussion

Using the architecture of Fig. 1 and considering a simple cost function defined by (5) we were able to reproduce the essential characteristics of saccadic eye movements. The proposed cost function does not directly penalize the saccadic duration, instead, it punishes the total visual error integrated over the saccadic and part of the fixation period. Therefore, in addtition to saccadic movement modeling, our model also explains the motor command generation immediately after

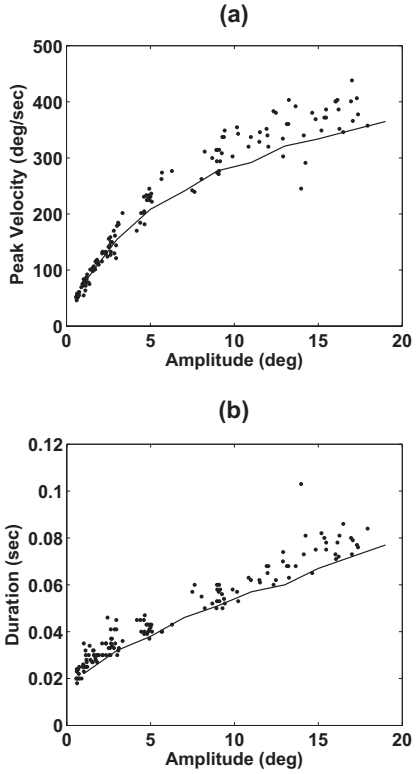

Fig. 4. Comparing the main sequence plots of the proposed model to experimental data. The solid lines represent model results after learning, with parameters $\sigma = 0.002$ and $k_{\rm reg} = 0.016$. The dots are experimental data taken from [15].

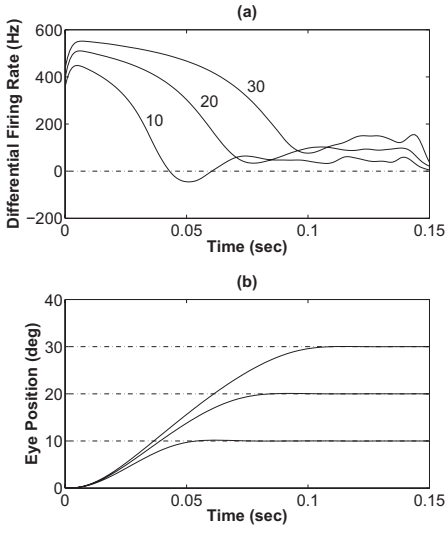

Fig. 5. Model behavior for saccades to targets at 10°, 20°, and 30°: (a) optimized neural command signals defined as the difference between agonist and antagonist neural commands; (b) eye position (eccentricity) in head coordinates. Target positions are shown by dashed lines. Learning is performed with parameters $\sigma = 0.002$ and $k_{\rm reg} = 0.016$.

a saccade, before the visual feedback from the target is re-established.

The learning mechanism of our model is derived from the proposed cost function. Since this mechanism locally adjusts the connection weights of the neural controller, it is also a biologically plausible learning rule. Simplifying equations (8) and (9), the synaptic weight adaptation has the following form:

$$\Delta w_{ij} = \delta_{ij} \int_0^T \text{sign}(r_{\rm vis}(t))\, s'_{ij}(t)\, dt - 4\delta_{ij} k_{\rm reg} w_{ij}^3, \quad (10)$$

where $s'_{ij}(t) = \int_0^t s_{ij}(\tau) h(t-\tau) d\tau$ is the response of the oculomotor forward model to the pre-synaptic activity. Since vision is impaired during saccades [13], there should be a second oculomotor forward model which provides the sign of the visual error to the adaptation mechanism using the current neural control signal, $u(t)$, as input. Therefore, $\text{sign}(r_{\rm vis}(t))$ is dependent on $u(t)$ which represents the post-synaptic activity. Consequently, the adaptation mechanism in (10) is composed of two terms: First, a term which depends on pre- and post-synaptic activities passed through forward models of the oculomotor plant; Second, a weight decay term ($-4\delta_{ij} k_{\rm reg} w_{ij}^3$) which is independent of the neural activities. Regardless of the location of these forward models, we speculate that the local adaptation mechanism in (10) might be responsible for the learning of saccadic eye movements.

The cerebellum has been widely regarded as a neural substrate where the internal models of the motor system are located (see [21] for a review), and the most convincing neurophysiological data for internal models has been obtained for eye movements [22]. Bastian suggests that the cerebellum performs feedforward correction on the movement based on the error assigned to the previous movement [23]. Interestingly, for saccadic eye movements, an experimental study by Soetedjo and Fuchs indicates that the complex spike activity of Purkinje cells (P-cells) in the vermis of the oculomotor cerebellum signals the sign (direction) but not the magnitude of the visual error during saccade adaptation [24], a finding which is in accordance with the adaptation mechanism of our model described in (10). Based on the mentioned studies, the architecture of Fig. 1 will be more realistic if we assume the adaptation unit as part of the cerebellar vermis. This assumption, however, requires several parallel implementations of the forward model for each weighted connection. The existence of parallel climbing fibers in the cerebellum, which carry error signals in motor command coordinates [25], may promise a possible neural basis for parallel forward models, but further investigation is needed.

So far we speculated on possible neural substrates responsible for the adaptation. Now we look for possible neural substrates that are managing open-loop control of saccades. Takemura and colleagues analyzed the relationship between the firing patterns of the P-cells in the ventral paraflocculus (VPFL) area of the cerebellum and ocular following responses [4]. A second order linear regression method was

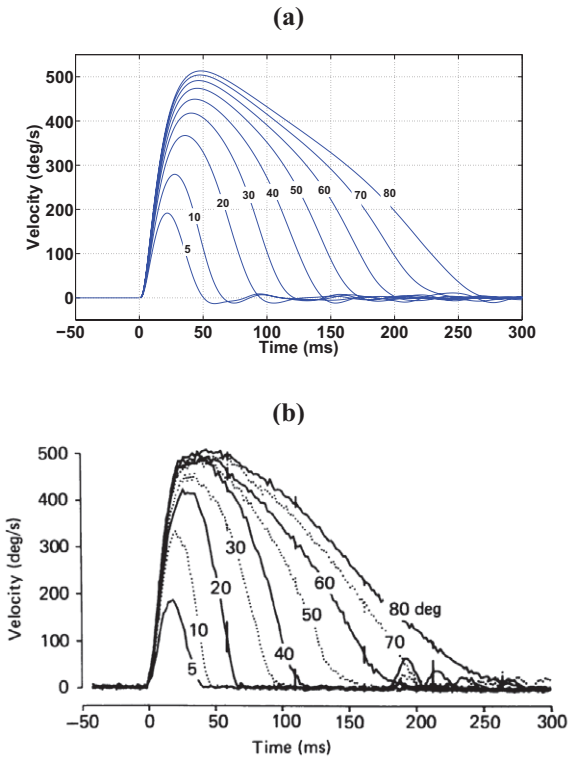

Fig. 6. Comparing model velocity profiles to experimental data. (a) Adapted velocity profiles for target positions from 5° to 80°. (b) Experimental data taken from [2]. In both plots, the symmetry degrades gradually as the saccadic amplitude increases.

used to reconstruct these signals based on three aspects of the eye movement: position, velocity and acceleration. This second-order linear method was able to regenerate temporal firing patterns of VPFL neurons. When a single set of coefficients was used for different visual stimuli in order to reconstruct the firing pattern of the cells in VPFL, the best fits were found for P-cells in this area. This observation implies that there is a linear relationship between the firing pattern of P-cells in the cerebellar VPFL and the eye kinematics. Therefore, it is plausible to assume that the oculomotor plant receives neural commands from the P-cell layer in the VPFL area of the cerebellum. Hence, the cerebellar VPFL is a strong candidate for the neural controller of our model. More specifically, we can consider the neural delay line structure as a model of the granular layer and the read-out neuron as a P-cell, in accordance with the cerebellar models which assume the granular layer as a basis for the spatio-temporal representation of the input signals and the P-cell layer as a layer that receives weighted projections from the granular layer [26][27].

Another possible candidate for the neural controller is the superior colliculus (SC). It has been widely assumed that the caudo-rostral spread of activation emerging among the build-up cells of the SC is caused by the visual error feedback signal during saccadic eye movements [28][29][30]. One of the most important predictions of these models is that interrupting this spread should delay the arrival of the activity at the rostral SC, and the eye should reach the target with delay. However, a lesion experiment performed on the SC does not support this idea: Aizawa and Wurtz observed that instead of delaying the reach time, the lesion results in a curved trajectory that does not end at the target position [31]. Motivated by this observation, Nakahara and colleagues suggested that the spread of activity is a mere epiphenomenon of the asymmetric horizontal connections within the SC [32]. This suggestion supports our assumption that the neural activity propagation in the delay lines is a self-reliant process which does not rely on any external feedback.

Fig. 7 shows the cerebellum-based part of our speculation on possible neural substrates responsible for the control and the learning of saccades. In this regard, control and learning are both accomplished by the cerebellum. However, we can not ignore the possible role of the SC in saccadic neural command gereration. Indeed, more experimental investigations are needed to clarify the contribution of the cerebellum or the SC to the open-loop control of saccadic eye movements.

V. Future Work

The present model only addresses the generation of saccades along one spatial axis, requiring one delay line for every target position along this axis. A naïve approach to generalize the model would be to introduce one delay line for every 2-D retinal location. However, this would require a very large quantity of neurons. As an alternative, one could introduce two separate 1-D controllers for the horizontal and vertical components of an eye movement. Such an approach has been successfully implemented in [33]. Furthermore, a liquid state machine (LSM) [34] may serve as a more biologically realistic alternative to our delay line structure. In addition, the LSM has been suggested as a model of the cerebellum [35]. Another open issue is a neural implementation of the forward model we are relying on, and a model that describes how the parameters of such a forward model are adapted. To this end, we can use the temporal sequence learning approach introduced in [36] to perform forward model learning. Finally, the adaptive open-loop controller model idea can be generalized to other ballistic motor control tasks beyond saccadic eye movements, by finding appropriate cost functions that underly such control tasks.

Appendix

Oculomotor Plant Model

The oculomotor plant in our model has the following transfer function:

$$H(s) = K[(1+T_1 s)(1+T_2 s)(1+T_3 s)]^{-1},$$

where T_1, T_2 and T_3 are the time constants and K represents the DC gain of the plant. Using the inverse Laplace transform, the impulse response of the plant can be retrieved as:

$$h(t) = K(k_1 e^{-\frac{t}{T_1}} + k_2 e^{-\frac{t}{T_2}} + k_3 e^{-\frac{t}{T_3}}),$$

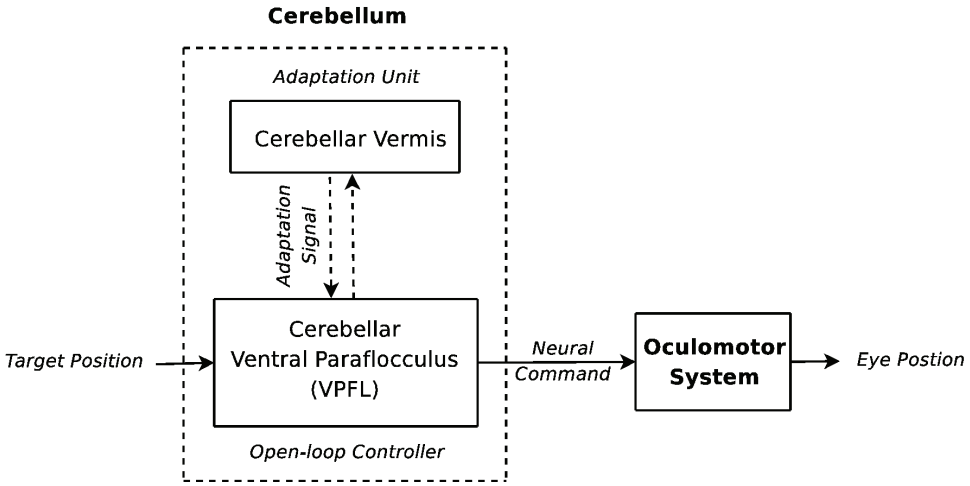

Fig. 7. Biological interpretation of the model architecture, based on cerebellum.

where:

$$k_1 = \frac{T_1}{T_1^2 + T_2T_3 - T_3T_1 - T_1T_2},$$
$$k_2 = \frac{T_2}{T_2^2 + T_3T_1 - T_1T_2 - T_2T_3},$$
$$k_3 = \frac{T_3}{T_3^2 + T_1T_2 - T_2T_3 - T_3T_1}.$$

Learning Algorithm

For fast convergence with the gradient descent approach, we use an adaptive learning rate. The method we use is quite similar to the RPROP algorithm [37] with a slight modification: Instead of using the sign of the error for updating weights, we directly use the error value. This method provides a local adaptive learning scheme where the learning rate is adapted according to changes in the sign of the gradient. If the gradient has the same sign in two successive iterations, the learning rate of the corresponding weight parameter will increase by a factor η^+; otherwise, it will decrease by a factor η^-. This adaptation process can be formalized as:

$$\delta_{ij}^{k+1} = \begin{cases} \min(\eta^+\delta_{ij}^k, \delta_{\max}) & \text{if} \quad \frac{\partial E}{\partial w_{ij}}^k \cdot \frac{\partial E}{\partial w_{ij}}^{k+1} > 0, \\ \max(\eta^-\delta_{ij}^k, \delta_{\min}) & \text{if} \quad \frac{\partial E}{\partial w_{ij}}^k \cdot \frac{\partial E}{\partial w_{ij}}^{k+1} < 0, \\ \delta_{ij}^k & \text{if} \quad \frac{\partial E}{\partial w_{ij}}^k \cdot \frac{\partial E}{\partial w_{ij}}^{k+1} = 0, \end{cases}$$

where $k+1$ represents the current and k represents the previous iteration number of the adaptation procedure, $\delta_{\max}$ is the maximum and $\delta_{\min}$ is the minimum allowed value for the learning rate.

The cost function (5) is highly non-linear and therefore expected to suffer from the local minima problem. Since the RPROP based learning method allows for larger learning rates without resulting in instabilities, it is less prone to be entangled in local minima.

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