Dual Path Revisited : membrane integration in the saccadic burst generator

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Abstract

Key words:

1. Introduction

Generalities about gaze orientation. Posture and movements control is at the core of the nervous activity, and the understanding of its basic principles is great challenge for it involves many intricately connected cortical and sub-cortical regions. Lots of observations have been done, and lot of conceptual models have been proposed in the last decades. Among various motor skills, gaze orientation, that moves the eyes (or the mouth) toward the direction of interest, is an example of choice for its wide-spread genericity and reproductibility among species, from early vertebrates to man.

Saccade overview. Saccades are the high velocity displacements of the eye produced to align the greatest accuracy zone of the retina, the fovea, with targets of interest. Saccadic eye movements allow to very quickly align the fovea with visual target of interest initially detected in peripheral visual field.

Metrics and basic figures. The saccadic part of the oculomotor system has been studied since decades through animal and human experiments [1, 2, 3]. [attention ref Robin75 est plutot de la modelisation d'apres le titre] In human, the amplitude of these movements ranges from a few degrees to 40-45 deg and their velocity profile corresponds to bell shape curves with peak values up to 800 deg.s-1.

The brain stem saccadic circuit. Being present in most of vertebrate species, the neural network controlling the production of motor commands for these movements is located in the brainstem. It has been the subject of numerous studies since last 60 years. This large data body gives a very precise knowledge of the organization and functionning of this network (for review: [4, 5, 3, 6]). The very fine and precise observations of the brain stem neural circuit that produces saccades has been attractive for modelling. Some of the key mechanisms at stake for the control of movement are indeed expected to implemented by this system, such as the transformation from topographic coding to frequency based coding ("spatiotemporal transformation" [7]), feedback control of the amplitude [1], adaptivity and robustness to change.

Fine-grain modelling. If the general pathways from the decision to the motor command start to be well understood, a more fine-grain description of the parallel mechanisms regulating the command acuracy in the reticular formation are still far from understanding.

The models are mostly defined by a scheme on a paper, and they are very useful for designing experiments on human or animals, but they are seldom implemented in a way that may show how they work and behave as time passes.

In particular, the description of a motor control in terms of spiking neuronal activity is still at the very beginning, despite the rise of computational power that allows to simulate the dynamics of interactions between neural populations in a realistic way. Many models of the saccadic system have already been proposed [8] but none of them has been implemented since with spiking neuron networks.

A gap is thus induced between models and experiments, the latter accounting more and more for temporal recordings.

[All these computational models have brought both informations and hypotheses about the interactions of the underlying neural networks].???

Implementation. The model has been implemented as a modular SNN and its dynamics has been simulated with the DAMNED simulator.

Plan. Section 2 contains an overview of the physiology of saccadic eye movements Section 3 presents an overview of existing models. Section 4 presents a 2D motor map model of the superior colliculus intermediate and deep layers. Section 5. Section 6. Section 7. Section 8.

2. Neurophysiology of saccadic eye movements

2.1. The MotoNeurons (MNs)

The activity of the extra-ocular muscles responsible of saccadic movements are controlled by the activity of several motoneuron nuclei (see **??** for reviews). The characteristics of the motor command for a saccadic eye movement is now well known: motoneurons emit a brief high frequency burst of spikes that induces the muscle contraction. This muscle activity allows to overcome the orbital viscous drag and to displace the eye with a high velocity. Then the eye is maintained in its new position through a tonic discharge of motoneurons. As a whole this saccadic command has been called "the pulse-step pattern".

2.2. The Short Lead Burst Neurons (SLBNs) : The source of the pulse signal

The motoneurons burst (pulse) has its origin in a category of neurones called Short Lead Burst Neurons (SLBNs). An on-direction can be defined for these SLBNs: organization in Left / right / up / down systems. Each SLBN discharge for every saccade in its on-direction. Indeed, some of these neurons are excitatory (EBNs), project ipsilaterally toward motoneurons and display a burst of spikes just before saccade ($10ms$). The rest of this population is inhibitory (IBNs). These cells inhibit contralateral motoneurons (and also contralateral EBNs and IBNs) hence preventing non adapted activities when a saccade has to be produced. The amplitude of the saccade is a function of the duration of this burst and of the frequency of this burst ([9, 6]). At this level, the saccade amplitude depends on the number of spikes in this burst and the velocity profile of the saccade depends on the frequency profile of this burst. Hence, at the level of the SLBNs, the coding of saccade characteristics is a temporal coding (frequency, duration).

2.3. The source of the step signal

To hold the eye in its new position, the required MNs tonic discharge is the result of an integration (in a mathematical sense) of the velocity signal present in the SLBNs burst. This velocity integration allows to obtained the tonic activity corresponding to the new eye position after the displacement due to the pulse. This integration process has been showed to be performed by the Nucleus Prepositus Hypoglossi and ajdacent regions of vestibular nucleus ([10, 11])

2.4. The Omnipause Neurons (OPNs)

Another important cell class belonging to the brainstem network controlling saccades is formed by the OmniPause Neurons (OPNs). These OPNs inhibit SLBNs. In fact, these neurons are tonically active during gaze fixation and hence prevent SLBNs to be activated. Their activity has to be turned off to allow a saccadic eye movement. This organization is known under the term of the OPNs "gate". As there is only one pool of these OPNs in the brainstem and as these neurons present a pause in their activity for saccade in every directions, they have been called omnipause neurons. OPNs recover their activity at the end of the saccade. At least for natural saccades (versus electrically evoked saccades) it seems that their activity recover is not the sole determinant of the saccadic termination ([12, 13, 14]).

2.5. The central Mesencephalic Reticular Formation (cMRF)

The cMRF is another part of the reticular formation that revealed to be involved in saccadic control. Three functions have been proposed for for this structure: - feedforward conduction of motor commands from the SC to SLBNs (and possibly a participation in the decomposition of a vectorial command in its horizontal and vertical components) - opening of the OPNs gate - feedback mechanisms through connections with SLBNs area and SC. Electrical stimulation of this region produces saccades ([15, 16]), its inactivation leads to major deficits in saccadic control ([17]) and recordings of cMRF cells show precise relationships between neuronal activity and saccade characteristics ([18, 19, 20, 21]). Additionally, anatomic data revealed its importance in the saccadic system: the cMRF and the SC are strongly and reciprocally inter-connected (Moschovakis et al 1988a,b; Chen and May 2000). Neurons of the cMRF project to the OPNs areas (Edwards 1975; Langer and Kaneko 1984, 1990; Bttner-Ennever and Bttner 1988; Horn et al. 2006). Nevertheless, possible projections from cMRF neurons toward the contralateral pool of premotor neurons (SLBNs) remains discussed (Bttner-Ennever and Bttner 1988; Scudder et al 1996). Importantly, the cMRF seems also to benefit from feedback information as the EBNs project to the cMRF, but this projection is an ipsilateral one (Edwards 1975). Finally, also demonstrated are relationships between the two cMRFs (in the fish: Luque et al. 2008; in the monkey: Warren et al. 2008, 2009). Neurones of cMRF show a burst of activity for contralateral saccades. This discharge begin at least 30 ms before the movement. Hence the latency between peak discharge and the start of the saccade was either the same or slightly shorter than that

noted for saccadic cells in the SC. In cMRF, very few neurons with closed movement fields were described [cMRF: 4% - PPRF: 0%] (Waitzman, Silakov and Cohen 1996). Majority of PPRF and cMRF neurons best correlated to component velocity (only 20% cMRF and 30% PPRF related to vectorial velocity) (Cromer and Waitzman 2007). Importantly, some cMRF neurons have activity that best correlate with saccade duration, whereas this best correlation was with saccade amplitude for other cMRF neurons (Cromer and Waitzman 2006). Finally, Waitzman et al. (1996) showed that around 70% of recorded cells were high-background activity which were inhibited during ipsiversive saccade. Around 30% were low-background cells, some of which displayed a burst just before the end of ipsiversive saccades (Waitzman et al. 1996). Finallty, the majority of cMRF cells were found to be clipped to the end of the saccade (60-75%) whereas the remaining 40-25% were only partially clipped to the saccadic end.

The Superior Colliculus (SC)

The SC is a paired and layered structure situated at the roof of the brainstem (see Scudder et al 2002; May 2006; Moschovakis et al. 1996; Sparks and Mays 1990; Isa 2002 for reviews). Neurons located in superficial layers are visual neurons and those contained in this intermediate-deep layers present multi-sensory activities (visual, auditory and somatosensory) and pre-motor activities. The SC is organised in neuronal maps: visual map for superficial layers; multi-sensory and motor map for deeper layers. For these deeper layers, the amplitude of the movement is coded along the antero-posterior dimension of the map [rostral sites = small amplitudes and caudal sites = large amplitudes] whereas its direction is coded along the medio- lateral dimension [lateral = downward saccades and medial = upward saccades]. Hence, saccade related cells of these deeper layers show movement fields: a given neuron discharges for a finite set of saccade amplitudes and direction (Sparks et al. 1976). Hence, a spatial to temporal transformation must be performed between the SC level and the SLBNs level: the topographic representation of the target location with respect to the current position of the fovea has to be transform in a temporal code (frequency and duration) for required SLBNs (organization of projections toward the different pools of SLBNs).

Burst activities / Motor activities: control of saccadic eye movements. But in addition, to control the amplitude of saccades, the duration of all these activities have to be controlled. Activities in the SC also participate in target selection. Visuo-motor activities / prelude activities: target selection [it is known now that these processes of selection could be shared by several

types of eye movements : saccade and smooth pursuit + even by putton press with the hand] Importantly, axons of the output neurons of the SC have two mains target: the premotor neurons (the EBNs) and the cMRF. Several schemes have been proposed to take into account these projections (Soetedjo et al. 2002; Paul and Gnadt 2006; see also the dual path scheme of Sparks and Nichols 1996). Nevertheless, to the best of our knowledge, no implemented models have been proposed that incorporate this dual pathway organization.

Schemes proposed for the functionning of the brainstem neural network controlling saccadic eye movements [Proposed models / The concept of the Feedback Loop]

It is now well accepted that the control of saccadic eye movements relies on a feedback organization according to the seminal proposition of Robinson (Robinson, 1975). He proposed that the command to displace the eye (the burst) is controlled through the functionning of a feedback loop. In his model, signals carried out by the feedback loop corresponded to eye position. A refinement of his feedback loop model was to consider that signals are rather displacement signals (Jrgens et al. 1981).

NI functionning with displacement signal. Experimental evidence suggest that this feedback loop closes downstream the SC (Kato et al 2006). This does not preclude the existence of other larger feedback loops that would comprize the SC (see Discussion). From the target location with respect to the fovea (notably coded in deep layers of the SC), a desired displacement signal is computed. From this reference/desired displacement, the network would initiate the eye displacement. The network would monitor the already performed displacement that would be subtracted to the desired displacement to obtain a motor error (the displacement that remains to be done to align the fovea with the target) ̆2013 the motor error would be the drive that feed the SLBNs. When the motor error reaches zero, the SLBNs would stop their activity and the saccade would be terminated. Several experimental evidences now strongly support this scheme (Jrgens et al. 1981; partial inactivation of SLBNs region: Barton et al. 2003; brief electrical stimulations: Keller 1977, King and Fuchs 1977, Keller and Edelman 1994) The burst of spikes discharged by the SLBNs corresponds to a velocity signal. It entails that an integration is required to obtain an estimation of the actual eye displacement. It has been demonstrated that this integration is not performed by the same structures as those involved in the generation of the step component of the pulse-step pattern (Cannon and Robinson 1987; Cheron and

Godaux 1987; Cheron et a. 1986; Kaneko 1997). Thus the estimation of gaze displacement has to be performed by a second integrator. The physiological basis of this second integration remains debated (review: Scudder et al. 2002). Ž01cNeurons with the predicted discharges have been seen only rarely. Such a neuron would be silent at the onset of a saccade, gradually increase firing as the saccade progressed, [and decrease firing after the saccade with a time constant of 45-55ms.]Ž01d (Scudder et al. 2002). Nevertheless, some neurons with suitable discharges have been described in the vertical system: the inhibitory feedback cells (IFN) (Moschovakis et al. 1991a,b; Moschovakis et al. 1996). These cells are bursters that do not project to motoneurones but rather project back to the other bursters (riMLF Ž192 NIC Ž192 back to riMLF). This type of cells was already preoposed for the horizontal system in a modelling paper by Scudder (1988). We make another proposition in the current modelling study. The current displacement would be coded in the evolution of membrane potential of particular neurons (in the cMRF). Complementary aims of the present study was to implement a dual path scheme based on the projections of the SC to the cMRF and the EBNs. Modelling the spatio- temporal transformation [Only component neurons in EBN and cMRF areas].

3. General models of the saccadic system

Here we consider the saccadic system at a very general level : what are the necessary elements for producing precise saccades.

3.1. The spatio-temporal transformation

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According to the main sequence [REF], a stereotypic relationship can be observed between amplitude and duration, as well as between amplitude and velocity.

It is also observed that a very strong correlation exists between premotor bursting activity and eye velocity [9, 6]. Consequently, the amplitude of the saccade is expected to be mostly dependent on the level and duration of the burst of the pre-motor neurons in the burst generator, under the control of the colliculus, others parts of the brain stem, and the cerebellum.

It is generally admitted that the location of the focal activity that develops on the colliculus codes for the eye displacement [22], knowing the burst neurons activity depends initially on three aspects of the collicular activity :

- the location of the bubble on the SC map
- the radius of this bubble (number of neurons activated)
- the frequency of the neurons on the colliculus

The so-called spatio-temporal transformation [7] is the complex neuronal process that transforms the focal collicular activity into a bursting pre-motor command of limited duration, precisely driving the eye displacement whatever the radius and frequency of the collicular activity.

First to consider and central to the spatio-temporal transformation is the pattern of synaptic projections from the colliculus to the burst neurons. This pattern defines the graded synaptic influence from the rostral to the caudal part of the map. Also crucial are the initiation and termination mechanisms controlled by other sub-collicular groups of neurons, implying a gate opening mechanism (silencing the Omni-Pause Neurons - OPN) and a feedback signal having the role to facilitate the termination of the burst according to the estimated displacement done so far. Also implied the cerebellum sending monitoring signals to the burst generator and allowing the global system to adapt to varying conditions. The saccadic generator is finally composed of a surprisingly complex ensemble of interacting neuronal populations, the role of all of them not being deciphered so far.

3.2. Basic principles

The first generic model of the saccadic command was proposed by Robinson in the 70s. Inspired by the ideas of control theory, it is composed of a forward excitatory track (from the colliculus to the motoneurons) and a feedback inhibitory track which integrates the command and substracts it to the forward signal (see figure 1).

Let us introduce some notations first that will be used further on. In the Robinson feedback model, briefly presented, the output of the system is the frequency of premotor linear-threshold units having a bursting activity (Burst Neurons i.e. B.N.): $u_{BN}(t) = \Theta(h^+(t) - h^-(t))$ *where* Θ *is the Heaviside function and h+ and h− are respectively the excitatory and the inhibitory signals.* $h^+(t) = \int_{x,y} W(x,y) u_{SC}(x,y,t) dxdy$ *represents the influence of the*

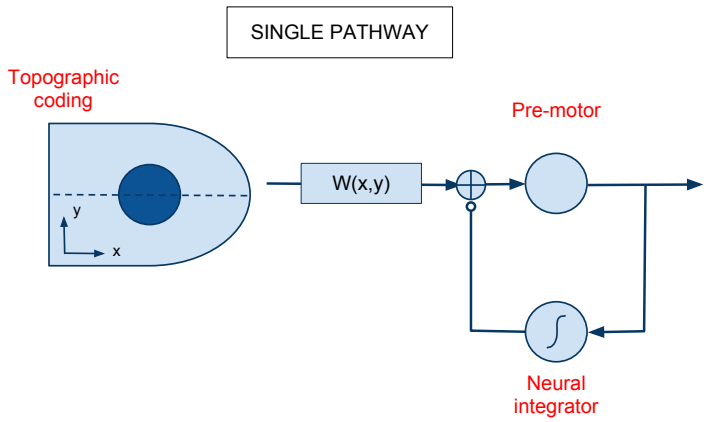

Figure 1: Single path model.

colliculus, where $W(x, y)$ *is the spatial projection and* $u_{SC}(x, y, t)$ *gives the instantaneous frequency at the locus* (x, y) *and time* t*.* $h^-(t) = k \int_0^t u_{BN}(t)dt$ *is the integral of the motor output according to gain* k*.*

This model is of course highly idealized, but interesting for the concepts and questions that raise out of it. Consistently with feedback control principles, the command relies on an error correcting mechanism, where the collicular signal h^+ gives the initially observed motor error and the feedback signal h^- gives how much has been corrected so far. The resulting command $h^+ - h^-$ is proportional to the current motor error (the difference between current and expected position). The command is expected to decrease for increasing time until equilibrium point is reached. In principle, feedback signal could be observed through proprioception, but the absence of proprioception in the case of saccadic control suggests that a feedback signal is internally estimated by specific neural integrator units that integrate the command over time. The amplitude of the resulting saccade is proportional to the integral over time of $u_B N$, where gain, time constants and projections can be adjusted in order to fit the data.

As shown by the formulas, two kinds of integrations take place in the model. Spatial integration, on one side, is a model for the collicular synaptic projections. Depending on the values of $W(x, y)$, a graded efficiency can be modelled from, e.g, the rostral to the caudal locations, consistently with biological data [7, 23]. Temporal integration, on the other side, is carried out

by a specific group of neural integrator units, whose activity is expected to increase in proportion with current pre-motor activity. Interestingly, neurons displaying this property have been observed next to the motor command in the NPH (Nucleus Prepositus Hypoglossi), maintaining the eye in the desired position until a new saccade takes place [10, 11]. However, no evidence of such an activity has been observed at the pre-motor stage so far, despite numerous attempts. The temporal integration process remains hidden, despite behavioral evidences of an effective feedback control taking place at the sub-collicular level. This absence of data being quite puzzling, it is generally admitted that a diffuse temporal integration process may nonetheless be present in the Burst Generator, not directly observable at the level of a single neuronal population. We consider this question as central for modelling studies, and present in the following several alternative hypotheses regarding the nature of this sub-collicular feedback process.

The Robinson model has been developped and enriched since in order to give a better account of the observations [24, 25, 26, 27], but the principles at stake remain essentially the same. The Robinson model and its derivatives are considered in the following as single path models for both saccade amplitude and duration are under the control of a single projection $W(x, y)$. In those models, some fine tuning or additional assumptions must be made in order to retrieve the main sequence, giving rise to intricate models which often appear quite difficult to replicate. Those models are moreover difficult to reconcile with the observations of [28] which have shown that a change in the level of the collicular activity results in slower but still precise saccades, the final amplitude being representative of the barycenter of the collicular activity (the duration being higher and the amplitude remaining unchanged when collicular activity is lowered by lidocane).

3.3. Alternative models

In principle, topographic coding or locus coding observed in the colliculus means that for a given position, and whatever the level of activity (in frequency and/or diameter of the focus of activity), the final displacement will be the same. Stimulation-based observations show for instance that within certain bounds, the final displacement is essentially the same whatever the intensity or frequency of electrical stimulation. Several alternative models can be considered in order to support those observations. The most straightforward account for it is known as the averaging hypothesis. As suggested by [29] for instance, a weighted average could be calculated on the basis of

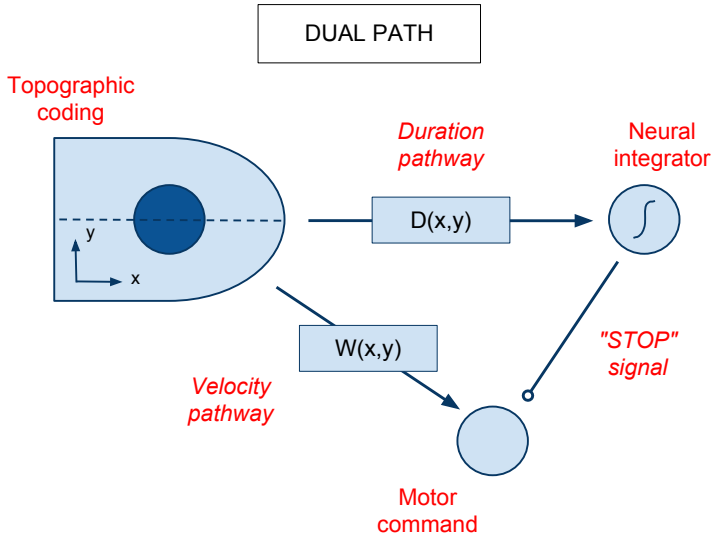

Figure 2: Dual path model.

divisive inhibitory interneurons normalizing the total activity taking place in the map. This model being quite unplausible, an alternative model using a saturation mechanism has been proposed by the same author, using a specific pathway having the role of counting the number of spikes emitted by the colliculus so far, and silencing the command when a specific number is reached. The generalization of this model is known as the dual path hypothesis [30] suggesting that duration and velocity may be coded (independently) by two independent tracks from the colliculus (see figure 2).

In the dual path model, two projections from the colliculus must be considered, so that duration and velocity can be decoded independently from the collicular activity. The first one (velocity) would depend on projection $W(x, y)$, and the second (duration) on projection $D(x, y)$. This model is appealing for it elegantly resolves the saccade normalization problem, without requiring out-of-the box divisive operation. Some questions remain, however, on the existence of a second pathway originating from the colliculus and having the property of stopping the saccade at appropriate time. This role could be devoted, for instance, to the cerebellum, under the hypothesis that some temporal integration (effectively) actually takes place in the cerebellum region . This assertion is supported by observations showing ac-

tive feedback from the caudal fastigial nuclei to the burst neurons at the saccade termination [31]. Other data suggest however that saccade termination (effectively) actually takes place without cerebellum contribution [REF : inactivation/lesion cereb], so that additional stopping mechanisms need to be considered. One possibility is to consider the bunch of projection taking place between the colliculus and the central mesencephalic reticular formation (cMRF). Neurons of the cMRF are found to respond more specifically to the horizontal components of the saccade and supposedly participate to the distribution of the horizontal saccadic command to the eye and head effectors. Some observations, however, suggest that cMRF neurons can display a very brief burst of activity at the end of the saccade, possibly having a role in the saccade termination [32].

The main objection to the dual path model is the lack of feedback signal, so that at no time the burst neurons activity is used for the control of saccade termination. This appears contradictory with observations showing that a short interruption or chemical diminution of the burst neurons activity is compensated by the saccadic system [33], giving orthometric saccades. It seems that at some point the real motor command must be considered, either for feedback error correction or for the calculation of saccade termination time. This question will be discussed in the following sections.

3.4. Saccade initiation and reset

Finally, when building a model of the saccadic controller, three components need to be considered :

- LATENCY : the time necessary to initiate a saccade
- VELOCITY the number of spikes/s emitted by the burst neurons
- DURATION : the duration of the saccade

The control of saccade velocity and duration have been described in the previous paragraphs, let us consider here the question of saccade initiation. Central to the saccade triggering mechanim is the activity of the Omni-Pause Neurons (OPN), which are inhibitory neurons that spontaneously display a tonic activity that blocks the neurons of the burst generator. The control of the saccade initiation relies on the inhibition of the OPN by a disynaptic path from the colliculus implying an inhibitory interneuron.

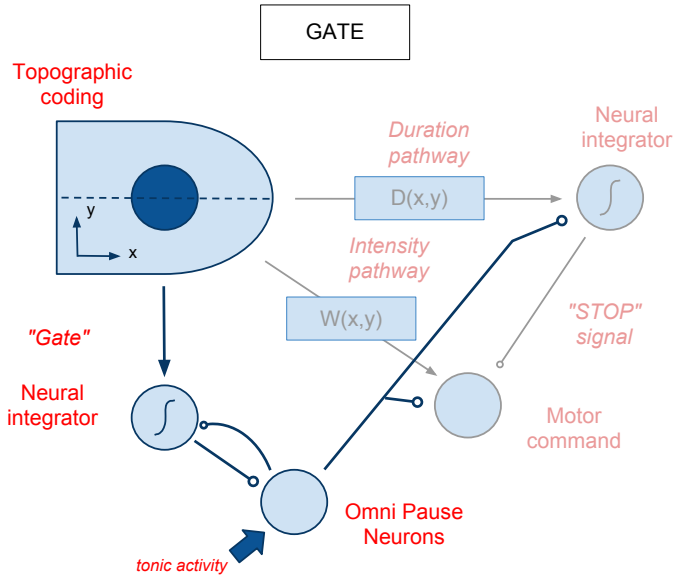

Figure 3: Gate mechanism.

It has been observed that a certain level of activity is needed to initiate a saccade. Under this critical level, no saccade can be triggered out. In classical models, saccade initiation requires a latch, or gate opening mechanism taking place at the level of the inhibitory interneuron, having the role of a switch when the SC activity crosses a certain threshold.

Some observations on SC electrical stimulation however report that the latency between the stimulation of the SC and the discharge of the burst neurons is reversely proportional to the intensity of the stimulation [???REFS???]. This point suggests that not the instantaneous level but rather the accumulation of enough SC activity over time is the critical variable. This conducts to the gate model we propose in fig. 3 where a gate-related neural integrator accumulates the activity coming from the SC and switches off the omni-pause neurons when a certain level is attained.

In modelling terms, this refinement in the gate opening mechanism is at the cost of introducing a new temporal integration unit, whose plausibility is not garanteed and seldom suggested by experimentalists. We put it forward here however for its interesting symmetrical situation regarding saccade initiation and termination. This symmetry is present in the bilateral structure

of the brain and a bilateral population of neuronal integrators could take a symmetrical role regarding saccade initiation and termination depending on the saccade direction with no additional cost. This is one of the modelling hypotheses we will try to develop and challenge in the following sections regarding simulation results and biological data.

A good candidate for this gate-related neural integrator is once again the cMRF for it has a direct monosynaptic (and reciprocal?) connection with the OPNs [34, 35, 3]. The observations of ipsi-lateral cMRF saccade related activity also support this idea [20].

3.5. Controversies

- "Frequency code" models (feedback inside the burst generator) or "Duration code" models (e.g. a feedback signal sent to stop the SC)
- Summation / Average controversy
- Some hidden/little discussed hypotheses :
- Stereotypic collicular activity
- Gradient of weights
- Neural integrator hypothesis and existing models

separation des composantes horizontales et verticales
BUN, LLBN, etc...
Collicular map models. Moving hill hypothesis.
Eye-head coordination

4. Proposal

In the present paper we propose a model that tests a new hypothesis for the organization of the feedback loop in the brainstem from the collicular 2D motor map activity. Our model aims at elicit new hypotheses about the possible role of different groups of cells given the constraints of neurophysiology. We consider here the SC, the cMRF, OPNs and premotor neurons (SLBNs / EBNs).

what the model is :

- the first model of the oculomotor pathway to be build with realistic neurons instead of "boxes".

- we consider the integration properties of the membrane of individual cells regarding the "dual pathway" hypothesis.

what the model is not :
the model is a simplified (or idealized) model of the oculomotor pathway :

- only eye movements and small saccades are consisdered
- not every group of neurons participating on the oculomotor path are considered (bun/bn, ebn/ibn, prelude burst neurons, cerebrellum, motor neurons, bg influence are neglected)

5. Methods

5.1. Neuron Model

The whole model proposed is defined at the neuron level. The neuron models used are based on leaky integrate and fire neurons also called "spiking neurons" [36]. Those widely used models receive presynaptic potentials which impact postsynaptic membrane. A threshold crossing of membrane potential leads to emission of a spike or action potential (figure 4).

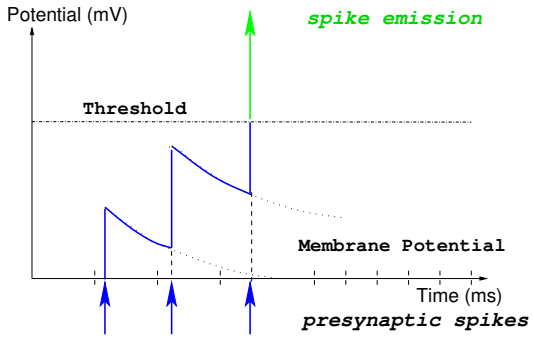

Figure 4: Leaky integrate and fire membrane potential response to synaptic spikes.

The model used in this work is a conductance based model [37]. This model is defined by the following equations :

$$\tau_M \frac{dV_M}{dt} = -(V_M - V_r) - g^+(t)(V_M - E^+) - g^-(t)(V_M - E^-) \qquad (1)$$

Where V_M is the membrane potential, V_r is the resting potential and τ_M is the membrane time constant. Each incoming spike triggers a variation in the membrane potential depending on the synaptic conductances. E^+ (resp. E^-) is the synaptic reversal potential and g^+ (resp. g^-) the excitatory (resp. inhibitory) synaptic conductance defined as follows :

$$\tau_s \frac{dg}{dt} = -g \tag{2}$$

Each incoming spike instantaneously increases synaptic conductance $g \leftarrow g + \omega_i$ where ω_i is the synaptic weight.

When V_M crosses a threshold Θ a spike is emitted, the membrane potential is reseted to it resting potential V_r for an absolute refractory period h.

This model lets synaptic influences to depend on the actual membrane potential thanks to reversal potentials. Details on the parameters used in this study can be found in section 9.

5.2. Collicular map

Physiological background ⇒ Introduction.

5.2.1. Motor map intralaminar connections

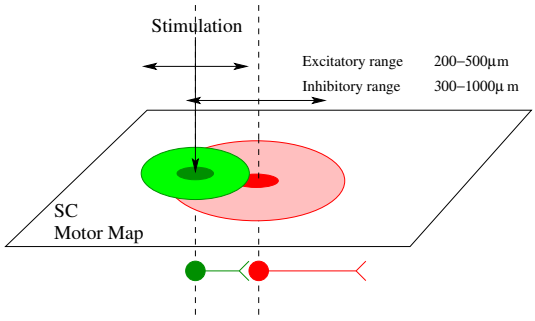

Figure 5: Disynaptic inhibition in the SC motor map.

The collicular motor map saccade related activity is initiated by the input (retina, thalamus, cerebellum and cortex) and further maintained by internal connection patterns. Intralaminar inhibitory and excitatory post-synaptic potentials have been recorded in-vitro through whole-cell patch-clamps via

photostimulation [38]. This study shows a raise of excitatory (inhibitory) post-synaptic potentials in a 200 up to 500 (200 up to 1000) range from the stimulated cell. These results confirm that excitatory and inhibitory neurons exhibits local axons in the intermediate layer of the SC.

Though disynaptic inhibitions (Figure 5) in the motor layer of the SC could shape the activity of map.

5.2.2. SC map model

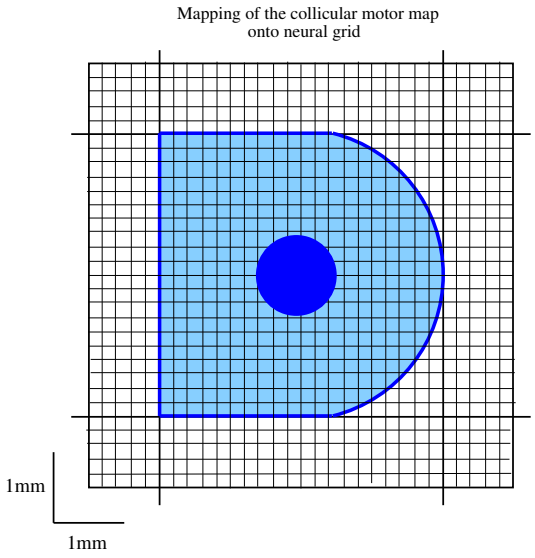

Figure 6: SC motor map mapping onto the 30×30 neural grid. The neural map is larger than the semi colliculus in order to avoid side-effects.

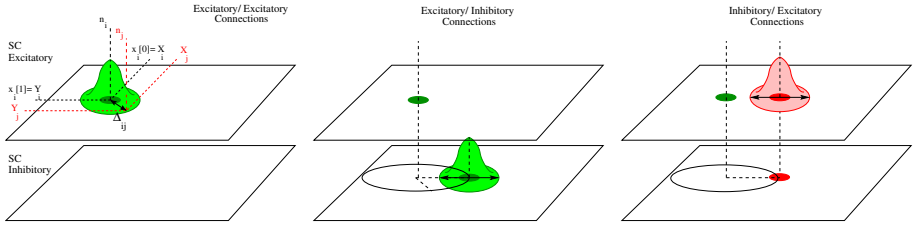

Figure 7: Intralaminar connections in the SC map. The projections are gaussian depending on neurons positions inside the map.

In this paper, the collicular map is made of a 30×30 grid of excitatory neurons and a 20×20 grid of inhibitory neurons. This map stands for a $4mm \times 4mm$ half colliculus (figure 6). The connection weights between excitatory neurons depend on neurons positions on the map. The shape of projection weights distribution is gaussian. The gaussian is centered around the presynaptic neuron for excitatory to excitatory connections and inhibitory to excitatory connections. The gaussian is centered distal to the presynaptic neuron for excitatory to inhibitory connections (figure 7).

The *Gaussian shaped projections* are defined by equation 3.

$$\omega_{ij} = \frac{1}{\sigma\sqrt{2\pi}} \times \exp\left(-\frac{(\Delta_{ij} - \mu)^2}{2\sigma^2}\right) \tag{3}$$

Where ω_{ij} is the connection weight between neurons i and j and Δ_{ij} the distance between neurons i and j defined by equation 4. μ is the center (average) of the gaussian and σ indicates the spread range (deviation).

$$\Delta_{ij} = \sqrt{\sum_{d=0}^{D} (x_d^i - x_d^j)^2} \tag{4}$$

Where $X_i = \{x^i[d] \in \mathbb{R}, 0 < d < D\}$ contains the neuron i coordinates in the $D = 2$ dimensions of the spatial area defined by the motor map where $x^i[0] = \mathbf{x} \in [0, 4], x^i[1] = \mathbf{y} \in [-2, 2]$ (figure 7).

The SC motor map specific parameters are given in section 9.2.1.

The SC map receives inputs from an electrode which stimulates a region around its spatial position. We consider an exponentially decreasing impact of the electric current around the electrode defined by equation 5.

$$\omega_{ij} = I \times \exp\left(\frac{-\Delta_{ij}}{\sigma}\right) \tag{5}$$

Where I is the current intensity and σ the electrode impact radius.

In this paper neuron models were defined with the following parameters.

- Threshold $\Theta = -50 \times 10^{-3} V$
- Membrane resting potential $\mathrm{V}_r = -60 \times 10^{-3} V$
- Excitatory neurons membrane time constant $\tau_{Me} = 10 \times 10^{-3} s$
- Inhibitory neurons membrane time constant $\tau_{Mi} = 200 \times 10^{-3} s$
- Absolute refractory period $h = 1 \times 10^{-3} s$
- Excitatory synaptic time constant $\tau_e = 5 \times 10^{-3} s$
- Inhibitory synaptic time constant $\tau_i = 10 \times 10^{-3} s$
- Excitatory synaptic conductance $g^+ = 6 \times 10^{-3} S$
- Inhibitory synaptic conductance $g^- = 67 \times 10^{-3} S$
- Excitatory synaptic reversal potential $E^+ = 0V$
- Inhibitory synaptic reversal potential $E^- = -80 \times 10^{-3} V$
- Inhibitory synaptic reversal potential for integrative neurons $E^{-I} = -60 \times 10^{-3} V$

9.2. Projections

Five different kinds of projections are used in the proposed model.

9.2.1. Gaussian shaped projections

The connection weights ω_{ij} depend on the distance Δ_{ij} between neurons and is given by equation **??**. Specific parameters are given for each projection between two neuron populations is the proposed model.

The SC motor map intralaminar projections are applied with the following parameters :

- Excitatory to excitatory connections : $\mu_{SC}^{ee} = 0mm$ and $\sigma_{SC}^{ee} = 0.5mm$.
- Excitatory to inhibitory connections : $\mu_{SC}^{ei} = 1.15mm$ and $\sigma_{SC}^{ei} = 0.2mm$.
- Inhibitory to excitatory connections : $\mu_{SC}^{ie} = 0mm$ and $\sigma_{SC}^{ie} = 0.2mm$.

Acknowledgements

This work is supported by the french ANR grant : MAPS (ref).

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