From brainstem to cortex: Computational models of saccade generation circuitry.

B. Girard * A. Berthoz

Laboratoire de Physiologie de la Perception et de l'Action, UMR 7152, CNRS - Collège de France, 11 place Marcelin Berthelot, 75231 Paris Cedex 05, France.

Abstract

The brain circuitry of saccadic eye movements, from brainstem to cortex, has been extensively studied during the last 30 years. The wealth of data gathered allowed the conception of numerous computational models. These models proposed descriptions of the putative mechanisms generating this data, and, in turn, made predictions and helped to plan new experiments.

In this article, we review the computational models of the five main brain regions involved in saccade generation: reticular formation saccadic burst generators, superior colliculus, cerebellum, basal ganglia and premotor cortical areas. We present the various topics these models are concerned with: location of the feedback loop, multimodal saccades, long-term adaptation, on the fly trajectory correction, strategy and metrics selection, short-term spatial memory, transformations between retinocentric and craniocentric reference frames, sequence learning, to name the principle ones.

Our objective is to provide a global view of the whole system. Indeed, narrowing too much the modelled areas while trying to explain too much data is a recurrent problem that should be avoided. Moreover, beyond the multiple research topics remaining to be solved locally, questions regarding the operation of the whole structure can now be addressed by building on the existing models.

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* corresponding author. Tel.: +33 1 44 27 13 91; fax: +33 1 44 27 13 82.
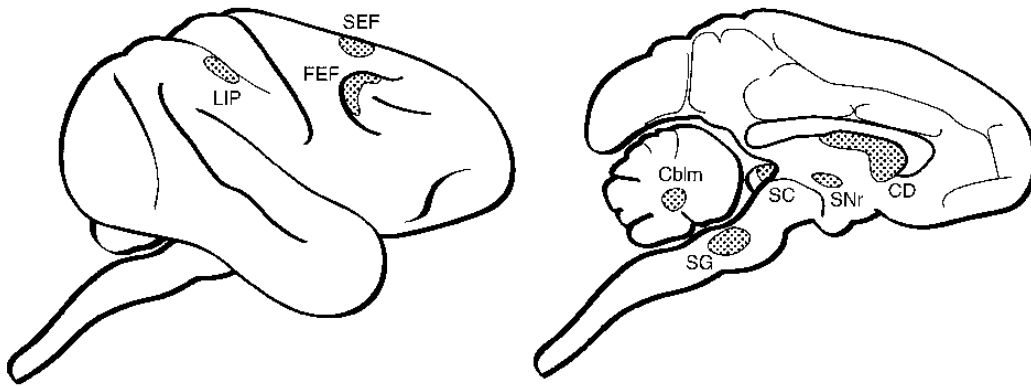 *Email address:* `benoit.girard@college-de-france.fr` (B. Girard).

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Fig. 1. Saccade-related brain areas (macaque monkey). Cblm: cerebellum; CD: caudate nucleus, input of the basal ganglia; FEF: frontal eye fields; LIP: lateral intraparietal area; SC: superior colliculus; SEF: supplementary eye fields; SG: saccadic burst generator; SNr: substantia nigra pars reticulata, output of the basal ganglia. From Hikosaka *et al.* (2000).

1   Introduction

There are three main different types of primate eye movements (slow, fast, and vergence movements), that are controlled by partially separate brain structures (Henn, 1993). The slow movements include the vestibulo-ocular reflex, the slow-phase of the optokinetic reflex and the smooth pursuit. The fast movements are the fast-phase of the optokinetic reflex and the saccades. Saccades are used by species (like humans and primates) whose retina have a central high-resolution region (the fovea) to explore visual scenes by redirecting gaze from one important visual stimulus requiring precise analysis to another. Their speed may reach 1000°/s in some primate species.

The mechanics of saccadic eye movements are relatively simple when compared to limb movements, which use multiple joints and operate with varying loads. Saccadic eye movements have therefore been studied for the intrinsic interest of understanding how they are generated, but also as a simple way to more generally study motor and premotor mechanisms in the brain.

Numerous brain regions are involved in the generation of saccades (Berthoz, 1996; Moschovakis et al., 1996), from the cortex down to the brainstem (Fig. 1). The closest to the movement execution are the vertical and horizontal saccadic burst generators (SBG), two sets of nuclei of the reticular formation which directly drive the ocular motoneurons (Scudder et al., 2002). Their function is to produce, from eye displacement instructions issued from higher level structures, the commands appropriate to generate saccades with the desired

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metrics. They are supposed to ensure accuracy by monitoring their own commands through an efferent copy-based feedback.

The superior colliculus (SC) is, with the frontal eye fields (FEF), the main structure sending saccade orders to the SBG (Moschovakis, 1996). The SC is a place of convergence and integration, often designed as the *final common path* of saccades. It receives projections carrying simple visual, auditive and somatosensory information along with more cognitive signals, where the sensory inputs are affected by attention, motivation and context. The SC drives the orientation of the whole body: it does not control the eye direction with regards to the head, but the gaze direction. Therefore, it activates not only the SBG but also, for example, the neck muscles.

The commands directed from the SC to the SBG are under the influence of adaptive modulations issued from the cerebellum (CBLM). It provides the SBG with additional input during saccades which are interpreted as 1) a calibration of the system induced by long-term adaptation of the saccadic gain, and 2) an on the fly correction of every single saccade, made necessary by the apparent variability of the rest of the saccade generating circuitry (Optican and Robinson, 1980).

The activity of the SC is gated by inhibitory inputs issued from a set of subcortical nuclei called the basal ganglia (BG) (Hikosaka and Wurtz, 1983a,b,c,d; Chevalier and Deniau, 1993; Hikosaka et al., 2000). Whereas the cortical areas generate numerous motor orders wich are directly send to the SC, they also project to the BG which is implicated in choosing which orders to execute, by disinhibiting the corresponding subregion of the SC. The role of the BG might however not be restricted to that metric selection (Handel and Glimcher, 1999).

Atop all these structures, many cortical areas are involved in saccade generation: the posterior parietal cortex (PPC), the dorsolateral prefrontal cortex (DLPFC), the anterior cingulate cortex (aCG), the pre-supplementary, supplementary and frontal eye fields (pre SEF, SEF and FEF respectively) (Platt et al., 2004). They provide rich inputs for the SC which allow the selection of targets by cognitive processes influenced by motivational and attentional states, along with the possible use of working memory or sequence learning capabilities (Pierrot-Deseilligny et al., 2003).

Numerous computational models of all these saccade-related brain regions have been proposed in the last thirty years. They helped to understand their operation, functionality and interconnections by proposing computational mechanisms and predictions that could be tested experimentally. However, most of them were restricted to one or a few subparts of the whole circuitry. This may sometimes cause problems when one attempts to replicate with such re-

stricted models experimental results that are indeed generated by another brain structure or by interactions with other structures. The objective of this paper is to review the computational models of saccade-related brain circuits, from brainstem to cortex, in order to propose an ensemble view of the system, of the numerous problems remaining to be solved at each level and of the relationships between each levels.

The computational models of the five categories of brain areas involved in saccade generation are reviewed in the next sections in the following order: reticular formation saccadic burst generators, superior colliculus, cerebellum, basal ganglia and cortex. Each of these sections have their own specific discussion, while global considerations are provided in a final conclusion.

2 Reticular formation saccadic burst generators

The reticular formation saccadic burst generators (SBG) generate activations transmitted to vertical and horizontal ocular motoneurons. The ocular motoneurons have a "burst-tonic" discharge pattern: the tonic activity is proportional to the eye position along the vertical or horizontal axis and the superimposed bursts, corresponding to saccades, are proportional to the amplitude of the saccade. The tonic activity is provided by the tonic neurons (TN) of two neural integrators (Moschovakis, 1997) located in the interstitial nucleus of Cajal (vertical integrator) and nucleus prepositus hypoglossi (horizontal integrator), in interaction with the vestibular nuclei. The bursts of activity are provided by two distinct burst generators (horizontal and vertical) composed of a set of neuron classes having specific patterns of activity (Scudder et al., 2002) :

- Medium-lead burst neurons (MLB): these neurons emit bursts of discharge beginning before saccade onset, they are composed of excitatory and inhibitory burst neurons.
- Excitatory burst neurons (EBN): the EBNs are active during ipsilaterally directed saccades, their afferents are the superior colliculus tectal long-lead burst neurons (TLLB), the brainstem long-lead burst neurons (LLB) and the IBNs. They project to ipsilateral motoneurons and to the tonic neurons (TN) of the neural integrators.
- Inhibitory burst neurons (IBN): the IBNs are active during ipsilaterally directed saccades, their afferents are the TLLBs, the LLBs and the EBNs. They project to contralateral motoneurons and to the tonic neurons (TN) of the neural integrators.
- Omnipause neurons (OPN): these neurons discharge tonically during fixation and stop during saccades. They project to and inhibit the MLBs and therefore function as a saccade temporal switch.

- Long-lead burst neurons (LLB) : the LLBs emit bursts well before the saccade, often reaching their maximum firing rate at saccade onset. LLBs with various properties (direction selective, direction and amplitude selective, etc.) and putative roles (relay between the SC and the MLBs, latch controlling OPN activity, relay between the SC and the cerebellum) were found in various brainstem nuclei.

Numerous computational models of these neurons interactions have been proposed, most of them exploring the putative feedback mechanisms ensuring saccade accuracy.

2.1 Robinson (1975)

The model proposed by Robinson was the first to incorporate the idea that saccades are not a ballistic movement, but that they are driven by a fast feedback loop allowing trajectory corrections on the fly. As the duration of many saccades is smaller than the delay needed for visual information to reach the reticular formation, this feedback can't be based on visual estimation of error. It was postulated by Robinson that it is in fact based on an efferent copy of the output of the saccadic burst generator itself.

The input of the model (Fig. 2, A) is the position of the target to foveate, in craniocentric coordinates, which generates a discharge of the excitatory burst neurons (EBN). This discharge is sent to the motoneurons (MN) where it generates the phasic part of their activity profile, while the remaining tonic activity, related to the eye position, is provided by the integration of this discharge by the tonic neurons (TN). This integrated eye position information is fed back to the EBN via an inhibitory connection, which decreases the EBN activity until the neural replicas of actual eye position and target position become identical. The omnipause neurons (OPN) have a role of timing trigger: they maintain the EBNs under continuous inhibition until some trigger signal inhibits them, allowing the EBNs activation. This activation, transmitted through the inhibitory burst neurons (IBN) maintains the OPNs silenced until the completion of the saccade. This model is for horizontal saccades only, it does not specify how a combination of two instances of it could interact to generate realistic oblique saccades.

This seminal proposal was the source of inspiration of many models of the brainstem saccadic burst generator, which extended and modified it in order to overcome its main shortcomings. These concerned specifically (1) the generation of straight oblique saccades by bidirectional extensions of the model and (2) the inadequacy of the craniocentric coding of target position, as the afferent structures of the saccadic burst generator (frontal eye fields and superior
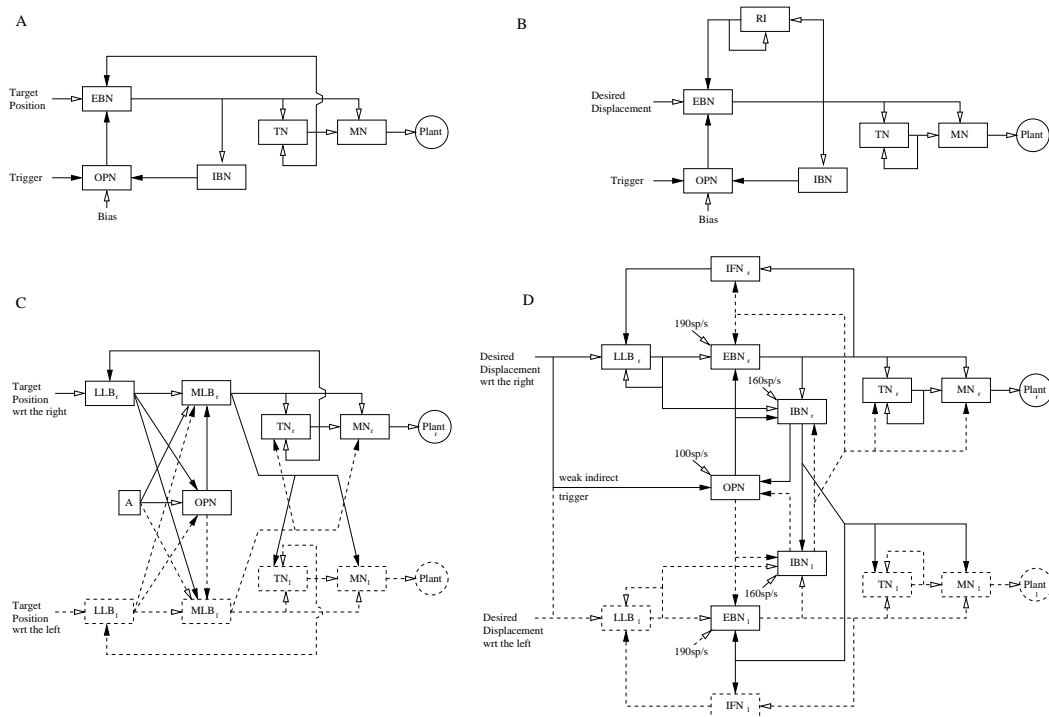
Fig. 2. saccadic burst generator models (1).A: Robinson's model. B: Jürgens et al. model. C: Grossberg and Kuperstein model. D: Scudder's model. EBN: exitatory burst neurons; IBN: inhibitory burst neurons; MLB: medium-lead burst neurons; LLB: long-lead burst neurons; OPN: omnipause neurons; TN: tonic neurons (integrators); MN: motoneurons; RI: hypothetic resettable integrator neurons; A: hypothetic arousal neurons; IFN: hypothetic inhibitory feedback neurons. Filled arrows: inhibitory connections; empty arrows: excitatory connections. Plain and dashed lines distinguish agonist/antagonist circuits, r and l subscripts respectively stand for the right and left circuits.

colliculus) appear to code it in retinocentric coordinates.

2.2 Jürgens et al. (1981)

Jürgens et al. investigated this idea of local feedback control in the saccadic burst generator and showed that (1) despite the duration and speed of saccades of a given amplitude may vary of 60%, their accuracy was almost constant and that (2) administration of diazepam, which increases the variability of duration and speed, does not affect accuracy. This ability to maintain the accuracy of saccades favours the hypothesis of a local feedback.

They however proposed a modification of Robinson's model, based on the observation that the actual amplitude of saccades evoked by a fixed stimulus increase with duration. Indeed, in such a situation, the addition of a realistic
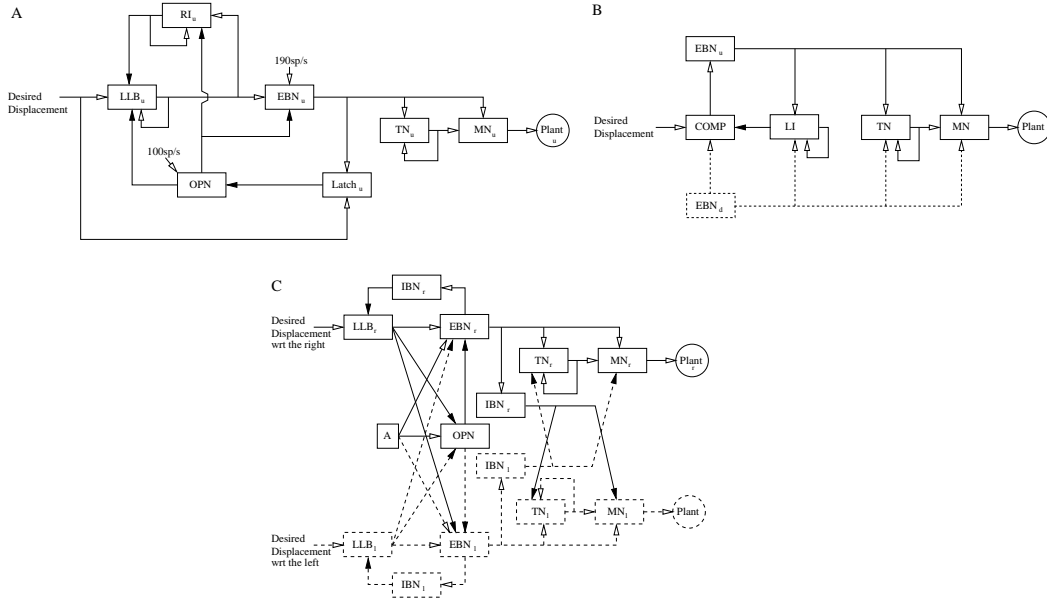
Fig. 3. saccadic burst generator models (2). A: Moschovakis' model. B: Nichols ans Sparks model. C: Gancarz and Grossberg model. EBN: exitatory burst neurons; IBN: inhibitory burst neurons; LLB: long-lead burst neurons; OPN: omnipause neurons; TN: tonic neurons (integrators); MN: motoneurons; RI: hypothetic resettable integrator neurons; LI: hypothetic leaky-integrator; A: hypothetic arousal neurons; COMP: comparator. Filled arrows: inhibitory connections; empty arrows: excitatory connections. Plain and dashed lines distinguish agonist/antagonist circuits, r, l, u and d subscripts respectively stand for the right, left up and down circuits.

delay in the transmission of signal along the feedback circuitry in Robinson's model tends to reduce the amplitude with respect to saccade duration rather than to increase it. In order to cope with these delays, they proposed (Fig. 2, B) that EBNs could rely on position information integrated directly from their output by a resettable leaky integrator (RI), whose biological equivalent remained to be found. Its leaky property would explain the observed variations of amplitude.

Beyond this minor adjustment, the input command in this model is set in retinocentric coordinates, *i.e.* it codes the desired displacement from the current eye position rather than the absolute final eye position. The RI therefore only integrates the current displacement and has to be zeroed at the end of each saccade by an unspecified mechanism. As stated by the authors, regular resets prevent accumulation of errors from saccades to saccades until error dominates the integrated signal, a problem to which continuously integrating mechanism are confronted (the question of error accumulation in the TN integration is however not raised). They did not emphasied the fact that a saccade generator using retinocentric coordinates is much more compatible with afferent structures (SC and FEF) coding the potential targets locations in retinotopic coordinates.

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This model was neither subject to an implementation nor to simulations in the work described by this original paper, but was in following papers (Arai et al., 1994; Dean et al., 1994; Nichols and Sparks, 1995; Das et al., 1995; Dean, 1995; Breznen and Gnadt, 1997; Arai et al., 1999).

2.3   van Gisbergen et al. (1985)

Van Gisbergen and colleagues studied the dynamic properties of oblique saccades, showing that (1) the onset of movement was simultaneous for horizontal and vertical components of the movement, and that (2) the movement along the component needing the shortest displacement is stretched (increased duration and reduced peak velocity) to last as long as the other one, consequently generating straight trajectories. This added new constraints on the coupling of unidirectional saccade generators models to get realistic full-range 2D saccadic burst generator and led them to propose that a vectorial pulse could be generated before being decomposed in vertical and horizontal components.

Their computational model is not as precise as the preceding ones in terms of neuronal circuitry, but assumes that the retinotopically coded signals of the SC and the FEF are transformed in a vectorial eye velocity command. This command is coded in an array of direction-selective long-lead burst cells (LLB), where each neuron is associated with a direction and its firing rate represents the amplitude of the desired saccade. This intermediate stage of computations drives the decomposed coding in the vertical and horizontal saccadic burst generators, therefore ensuring their simultaneous onset and the temporal stretching of the shortest component. Such an architecture necessitates the synaptic weights from the LLBs to the vertical and horizontal EBNs to follow sine and cosine functions respectively.

This model was proved to better fit the obtained data on oblique saccades than a model lacking the intermediate coding stage and therefore generating synchronous but independent movements along the two axis.

2.4   Tweed and Vilis (1985)

Tweed and Vilis were also interested in modelling a 2D saccadic burst generator and developped the van Gisbergen et al. model. Their main proposals were that (1) keeping the direction-sensitive LLB array of van Gisbergen et al. (1985), the local feedback should not occur in the EBNs but in the LLBs, before the component decomposition, and (2) because the 3rd order dynamic of the eye muscles is probably different on the vertical and horizontal axes,

three parameters linked with position, velocity and acceleration are needed for calibration of control on each axis.
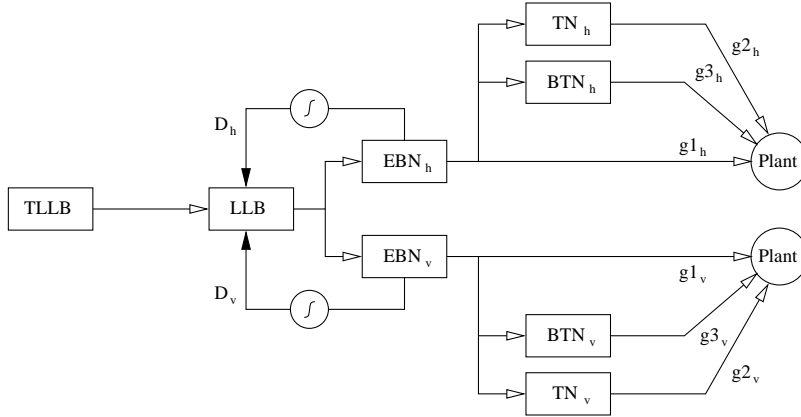

Fig. 4. Tweed and Vilis model of the saccadic burst generator. The h and v subscripts stand for horizontal and vertical respectively. BTN: burst-tonic neurons, considered as motoneurons (MN) in the other models. Filled arrows : inhibitory connections; empty arrows: excitatory connections. The integration of the EBNs output into the displacement signals D_h and D_v is not specified, but the use of a mechanism similar to Scudder's model is evoked. The mechanisms transforming the acceleration and velocity signal of the EBNs into the position and velocity signal of the BTNs and the position-only signal of the TNs the are not specified either.

The inclusion of the local feedback loop at the level of the LLBs has an interesting side-effect: it generates the experimentally observed saccade averaging, *i.e.* when two different saccade commands from the superior colliculus are simultaneously fed in the model, the resulting saccade is directed to the vector average of the two positions rather than to their sum. However, a consensus has now emerged to consider that this phenomenon is the result of lateral interactions in the collicular maps (see section 3).

Because of their second point, they considered that the eye plant was controlled by a composite signal mixing the output of the EBNs (coding velocity and acceleration), the TNs (coding position) and the burts-tonic neurons - usually considered as the motoneurons (MN)- (coding velocity and position) using three gains (g1, g2 and g3, Fig. 4). As the information concerning position, velocity and acceleration is encoded in these three populations of neurons, a linear combination of their output allows the calibration of the controler of the 3rd order dynamic eye plant.

Like the van Gisbergen *et al.* model, this model is not as close as Robinson's or Jürgens *et al.* models to the anatomy and physiology of the corresponding neural circuitry (OPNs and IBNs not included, integration mechanisms not detailed, etc.).

2.5 Grossberg and Kuperstein (1986)

Expanding the model of Robinson, Grossberg and Kuperstein addressed the problem of controlling antagonist eye muscles in the horizontal plane. They abandon the control theory approach used in the preceding models and use instead an artificial neural network. The architecture of their network is however quite similar to Robinson's scheme (Fig. 2, A and C) and keeps its craniocentric coding of the desired movement.

An intermediate stage is added to Robinson's proposal: the input of the network is LLB neurons projecting to the MLBs. The role of these LLBs is to control the activity of the OPNs, thereby specifying the triggering mechanism that was not specified in (Robinson, 1975). Inhibitory projections from the LLBs to the contralateral MLBs and from the MLBs to the contralateral TNs and MNs implements the coordination of two circuits operating in opposite directions.

Finally, hypothetic arousal neurons (A) control both the level of OPN tonic activity and the strength of the MLBs onset when OPNs inhibition is off. These neurons are the source of three properties of the circuit. First, at the end of a saccade, the OPNs inhibition is off and the LLBs inhibitory influence on the contralateral MLBs decreases as the target position gets closer. Consequently, the excitation of these contralateral MLBs by the A neurons becomes sufficient to generate an antagonistic burst, which helps stopping the eye and was observed experimentally. Second, a low level activation of these arousal neurons generates a saccade undershoot similar to those observed in case of fatigue state. Third, during perpendicular saccades, because of the OPNs deactivation, a coactivation of both circuit is obtained, generating small EBN and IBN bursts, a phenomenon that was also experimentally observed.

The main limitation of this proposal is the use of craniotopic input coordinates, which are not compatible with SC and FEF inputs.

2.6 Scudder (1988)

Building on the preceding models, Scudder kept the retinocentric coding of input orders of Jürgens *et al.*, the artificial neural network approach and the control of antagonist muscles of Grossberg and Kuperstein, and addressed the problem of straight oblique saccades generation raised by van Gisbergen *et al.* and Tweed and Vilis.

Using retinotopic coordinates implies the use of a local integration of the displacement, distinct from the eye position integration performed by the TNs.

Scudder replaces the biologically undefined RI of Jürgens *et al.* by an integration in the LLBs (Fig. 2, D): the burst produced by the EBNs under control of the LLBs is fed back to the LLBs through hypothetic inhibitory feedback neurons (IFN), and thanks to a recurrent positive connection with a gain of one, the LLBs directly integrate the difference between the command issued from the SC and the velocity orders sent to the MNs. In Jürgens *et al.*, integration is carried out before difference computation, here the order of the operations is inverted: the difference is first computed and then integrated. Such an architecture doesn't need any reset mechanism, as the saccade stops when this integrated difference decreases to zero.

The generation of straight oblique saccades is obtained relatively simply: in such saccades, the two active SBG circuits receive exactly the same burst as input (supposedly generated by the SC) and share a single OPN. The amplitude of the saccade component they respectively generate depend on a weight applied to the SC burst, defined by the locus of activity on the SC map (see section 3). The duration of the saccade, defined by the burst duration and the shared OPN gating, is the same for both component. As the SBG circuits operate linearly, their activities are similar but scaled by the weights respectively applied to their common input. The oblique saccade trajectories is thus straight.

The coordination with the contralateral circuit is assumed by the IBNs, which is biologically more realistic than the mixed excitatory-inhibitory neurons used by Grossberg and Kuperstein. The biases imposed on EBNs, IBNs and OPNs firing rate have a similar role as the A neurons excitations in the Grossberg and Kuperstein model, as they are also the source of an antagonistic burst at the end of the saccade and as varying their values accounts for the slowed responses observed in case of fatigue.

Finally, this model reproduces the results obtained by microstimulation of the OPNs at the beginning or in the middle of a saccade: the saccade stops during stimulation and then resumes.

Scudder states a few limits of his model: (1) the EBNs firing rate is too high in saccades larger than 15°, compared to experimental data; (2) the saccade averaging is not supported, but he assumes that this phenomenon is a property of the afferent structures (SC and FEF); (3) the model can produce the staircase saccades observed during prolonged stimulation of a SC site, but these do not match correctly with the observations (amplitude and range of variation are too small).

Moschovakis concentrates on the vertical saccadic burst generator. He observes that neurons having properties similar to the hypothetic IFNs of Scudder exist for the downward part of the system, they are located in the interstitial nucleus of Cajal. However, such neurons were not found in the upward part of the system, while neurons resembling Jürgens *et al.* RI exist in the nucleus of posterior commissure. He therefore proposes a neural network model of the upward saccadic burst generator inspired by the proposal of Jürgens *et al.*, using a RI which is simply reset by the tonic inhibition of the OPNs between saccades (Fig. 3, A).

The results obtained in the simulations are consistent with the known sensibility of the EBNs (1.5 spike per degree) and with the main sequence data in monkeys (saccade duration vs. amplitude and maximal velocity vs. amplitude relationships). Moreover, using a architecture similar to Scudder's proposal, the model generates straight oblique saccades, microstimulation of the OPNs interrupts or truncates saccades, whether they happen near the end of the saccade or not, and staircase saccades are evoked by continuous superior colliculus stimulation over a wide range of amplitudes. Finally, in Scudder's model, as well as in this one, a monotonic relation exists between the curves of the instantaneous motor error plotted against TLLBs intantaneous firing rate. The interpretation that this experimentally observed relation is indicative of a causal relationship between TLLBs discharge and motor error, which gave rise to models including the superior colliculus in the local feedback loop (see section 3.6), is therefore invalid.

A first limitation of the model concerns the fact that the RI is a perfect integrator, however a version of the model using a leaky-integrator RI can be built and was used in recent works of the team (see Bozis and Moschovakis, 1998 and section 3.14). Another one is the use of linear non-saturating EBNs: a nonlinear transfer function can be used instead, the consquences being that the TLLB-to-EBN weight then have a nonlinear relation with desired displacement and that the shape of the saccade duration vs. amplitude function gets steeper. Finally, experimentally observed staircase saccades have their amplitude reduced step after step, which is not the case here. However, this variation might be caused by the effects of direct superior colliculus connections to the motoneurons, bypassing the saccadic burst generator (Moschovakis et al., 1998), thus models of the saccade generator do not have to reproduce them.

2.8 Nichols and Sparks (1995)

The amplitude of a saccade evoked by stimulation of the superior colliculus usualy depends on its location on the retinotopic collicular map. However, when evoked shortly after a visual saccade, this amplitude varies and this variation decays exponentialy with the intersaccadic interval. Nichols and Sparks investigated that point and proposed a modified version of the Jürgens *et al.* model (Fig. 3, B). Being interested in this temporal phenomenon, they only kept the essentials of the model: the decoupled integration of relative displacement (in a leaky-integrator) for the local feedback on the one hand and of the absolute position (in the TNs) on the other hand. They simply extended this basic scheme for the control of upward and downward movements.

Instead of using an unspecified mechanism to reset the local integrator, they relied on its leak only. This precise choice explains the phenomenon of amplitude variation: if a saccade is evoked just after another one, the leaky integrator isn't completely discharged and the remaining charge affects the saccade as if a displacement had already been performed. The exponential decay of the leaky integrator charge after a saccade explains the exponential decay of this amplitude effect.

This Jürgens *et al.* extension was voluntarily kept very simple (OPNs and IBNs removed, use of single integrators for both the upward and downward movements) as its main point was to study the effect of a purely leaky integrator in the local feedback. However, the results concerning saccade amplitude variation are not very well understood, as the decay process seems to start from the begining of the saccade (Schlag et al., 1998) rather than at its end and as it was also proven that normometric saccades can be produced shortly after a previous saccade (Goossens and Van Opstal, 1997).

2.9 Quaia and Optican (1997)

Physiological evidence that the MLBs do not necessarily reach their maximal firing rate for movements aligned with the direction of the motoneurons they project to incited Quaia and Optican to propose a model of population-coding of saccade direction in the MLBs. The *on-direction* of the MLBs (the direction for which they reach their maximal firing rate) may in fact be titled away from the direction of the target motoneurons by as much as 70°, and their tuning curve can be fit by a Gaussian function centered around this on-direction.

This model uses four populations of MLBs (up, down, left and right) having varied on-directions and realistic patterns of activity (Fig 4, left) to code the vectorial pulse $\bar{D}$ corresponding to the order issued from the superior col-
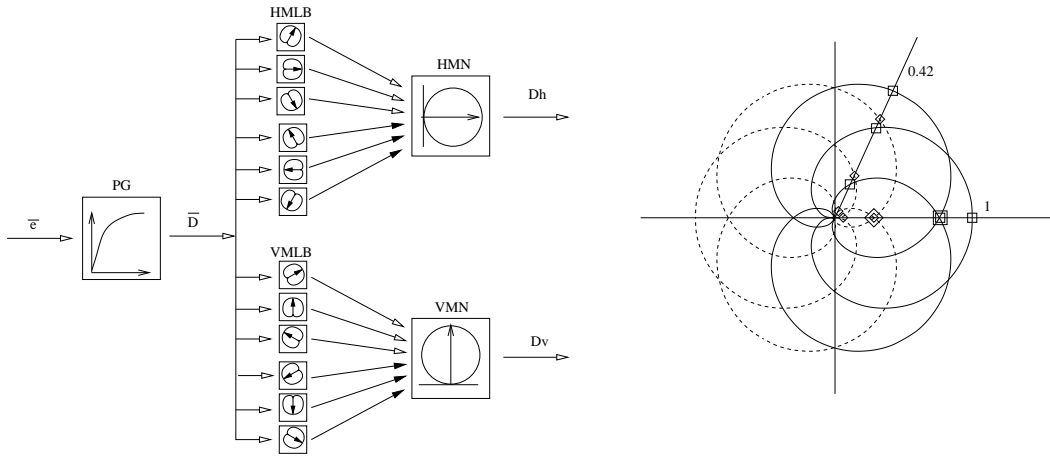
Fig. 5. Left: Quaia and Optican model of MLB population coding of the saccades's direction. $\bar{e}$: vectorial motor error; $\bar{D}$: vectorial drive; D_h: horizontal drive; D_v: vertical drive. Every population is made of 33 neurons (3 represented). Empty arrows: excitatory projections; filled arrows: inhibitory connections. Right: Activity supplied by MLBs to motoneurons of horizontal rightward muscle. Comparison of two rightward movements with the same vectorial amplitude. Each cardioid function represents the tuning curve of a MLB, dashed lines for inhibitory neurons, full lines for excitatory ones. The length of the segments between the origin and the intersection of the movement vector with the tuning curves gives the contribution of each MLB to the motoneuron activity, they are marked with squares for excitatory and rhombuses for inhibitory contributions. The resulting sum for the right-upward movement represents 42% of the sum obtained for the purely rightward movement. Adapted from (Quaia and Optican, 1997).

liculus. Each pair of populations associated to one axis project to the corresponding motoneurons, they exert an opposite effect (excitation or inhibition) depending on their associated direction. For example, the up MLBs excite the up motoneurons and inhibit the down motoneurons. Unlikle van Gisbergen *et al.* or Tweed and Vilis, such a model does not rely on an explicit decomposition of the superior colliculus signal into its horizontal and vertical components and thus avoids the use of sinusoidal connection weights.

The behaviour of this model during oblique saccades is quite interesting, as component stretching occurs even if the superior colliculus signal is not decomposed. This property emerges from the specific firing patterns of the MLBs and the repartition of their on-directions which makes the sum of agonist and antagonist MLBs activities smaller as the direction of movement diverges from the component axis (Fig. 5, right). The generated saccades are almost perfectly straight, because the neural and mechanical components of the model are perfectly symmetric. However, the introduction of asymmetries in the MLBs on-directions repartition or in the plant gains generates slightly curved saccades which are in accordance with experimental results.

This model is not a complete model of the saccadic burst generators: first, it

does not explain how the varied on-directions of MLBs are generated, as its unphysiological to assume that it is a characteristic of each neuron. Second, it does not propose a feedback mechanism in accordance with its distributed coding of the direction of movement. In fact, the estimation of the eye displacement signal is not explicitly available, as it is distributed among MLBs, therefore a local feedback mechanism would have to recompose it for integration, and then to decompose the integrated signal to feed each MLB. Including the SC in the feedback loop could simplify the problem, the authors however signal many flaws in the two main theories (see section 3) and consequently suggest to switch to new models of feedback, and especially to pay more attention to the role of cerebellum in ensuring saccade accuracy (see the cerebellum models of the same team, section 4.4).

2.10   Breznen and Gnadt (1997)

Breznen and Gnadt studied the response of two models (Jürgens et al., 1981; Scudder, 1988) to simulated continous SC stimulations. They confirmed that Scudder's model is unable to reproduce the characteristic staircase and proved that this flaw does not derive from parametrization but from the structure of the model itself. They proposed a first implementation of the theoretical scheme of Jürgens *et al.*, replacing resettable integrator and its unspecified resetting mechanism by a leaky-integrator resetting by spontaneous decay of activity (similarly as in Nichols and Sparks, 1995). This first model does not exactly reproduce experimental SC-stimulation results: frequency and damping of the oscillations generating the staircase saccades should depend on the amplitude of the stimulation currents. A second implementation where the gains and time-constants of the MLB and of the leaky-integrator are a function of their own activity (equivalent to activity-related synaptic adaptation) produces the correct dependence. This additional level of modelling complexity (synaptic adaptation) is reminiscent of the fatigue circuit included in a variation of the Robinson's model (Zee and Robinson, 1979). The model however forgets about eventual influence of the cerebellum on SBG operation, which could be partially responsible for the observed adaptation.

2.11   Gancarz and Grossberg (1998)

In 1998, Gancarz and Grossberg made an update to the Grossberg and Kuperstein SBG model. Their new model uses retinocentric coordinates and consequently eye displacement rather than absolute eye position in craniocentric coordinates.

The model (Fig. 3, C) is similar to the Grossberg and Kuperstein proposal

(Fig. 2, C) and changed on two points:

(1) The negative feedback is provided by the IBNs rather than by the TNs, thus the LLBs can be controlled by *desired displacement* signal and compute the displacement error. The model now operates in retinocentric coordinates.
(2) The inhibition sent by the EBNs to the contralateral SBG is relayed by the same IBNs, making the proposal more anatomically realistic.

This model is able to generate straight oblique saccades using the same solution as (Scudder, 1988; Moschovakis, 1994) (*i.e.* weighted SC burst input and shared OPN). In case of prolonged LLBs stimulation, it generates staircases correctly, and in the case of OPN stimulation, saccades are interrupted and resume after stimulation ceases.

2.12 Discussion

Most of the computational models of the saccadic burst generators have been built to in order to investigate the possibility that the feedback loop monitoring the saccade execution is located in the reticular formation (see Tab. 1). The necessity to operate in retinocentric coordinates, and thus to drive the saccadic burst generators with a command specifying the desired displacement relative to the current eye position in the head, was rapidly established. This led to questions about the nature (resettable or leaky) and the neural substrate of an eye displacement integrator distinct from the neural integrator driving the motoneurons (which codes eye position in craniocentric coordinates). These have not been decided yet.

The studied models evaluated their performance given a number of criteria corresponding to their abilities to reproduce some specific properties of the SBG:

- generation of saccades belonging to the main sequence,
- generation of straight oblique saccades,
- resumed saccades after interruption by OPN stimulation,
- generation of staircase saccades in the case of continuous input simulating SC stimulation,
- generation of a small burst by the antagonist SBG at the end of a saccade,
- coactivation of the vertical (resp. horizontal) SBGs during a purely horizontal (resp. verical) saccade.

However, the choice of evaluating isolated SBG models by their ability to produce main sequence saccades might not be a good one: when the cerebellum is deactivated or lesionned, saccades do not belong to the main sequence

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Table 1
Topics adressed by the reviewed saccadic burst generator models.

Model	LFM	SOS	AMC	SI	SCS	SA	AB	CoA
Robinson, 1975	•							
Jürgens et al., 1981	•							
van Gisbergen et al., 1985		•						
Tweed and Vilis, 1985	•	•				•		
Grossberg and Kuperstein, 1986	•		•				•	•
Scudder, 1988	•	•	•	•			•	•
Moschovakis, 1994	•	•	○	•	•		○	○
Nichols ans Sparks, 1995	•		•					
Quaia and Optican, 1997		•	•					
Breznen and Gnadt, 1997	•				•			
Gancarz and Grossberg, 1998	•	•	•	•	•		•	○

LFM: local feedback mechanism; SOS: straight oblique saccades; AMC: coordination of antagonist muscles control; SI: saccade interruption by OPN stimulation; SCS: staricase saccades; SA: saccades averaging (a property usually associated to superior colliculus processing); AB: antagonistic burst at the end of a saccade; CoA: coactivation of perpendicular SBG in purely horizontal or vertical saccades. • indicate that the model addressed the specific topic; ○ indicate that the experimental result was not explicitly simulated but should be explained by the model.

anymore (Robinson et al., 1993). If the main sequence is the result of SBG-cerebellum interactions, then SBG model parameters should be tuned using data gathered in animals with lesioned or deactivated cerebellum. Concerning the generation of straight oblique saccades, it seems that the mechanism proposed by Scudder (1988) (weighted SC burst input and shared OPN) and used since in many models including the SBGs (Moschovakis, 1994; Gancarz and Grossberg, 1998; Arai et al., 1994; Lefèvre et al., 1998, to name a few) is the simplest one and provides the best explanation of the phenomenon. Thus, we suggest that future models of the SBG should meet the last five criteria before exploring any new exotic property of the SBG.

Table 1 first summarizes which models explored the mechanisms of a feedback loop local to the SBG. It then distinguishes which models were dedicated to the study of a very specific point and the broader ones, which tried to explain as much experimental data as possible. Among these, it appears that (Moschovakis, 1994; Gancarz and Grossberg, 1998) are the most complete.

The preceding remark concerning the role of the cerebellum in the generation of main sequence saccades and the simulation of saccade averaging with the

sole SBG proposed by Tweed and Vilis (1985) advocate for a careful approach of modelling, where one should avoid narrowing to much on a single brain area and consider the role of its interactions with its neighboring circuits, before trying to explain with that single area more experimental data than what it can afford.

The Quaia *et al.* model (1997) is the only one which uses realistic population coding of the desired movement in the MLBs, the other models lump neurons in single computational units. This specific approach led them to questionning the existence of a feedback loop local to the SBG (see section 2.9). They also rejected the numerous proposals to include the superior colliculus in a larger feedback loop (see section 3), mainly because the SC does not appear to be crucial to ensure saccade accuracy. This resulted in models (Lefèvre et al., 1998; Quaia et al., 1999; Optican and Quaia, 2002) where the control of accuracy is controlled by the cerebellum only. Their point of view is a bit extreme, as, in their models, the superior colliculus and SBGs have great difficulties generating saccades –be them inaccurate– without the help of the cerebellum. However, they opened a path that should followed, where the SBG operation is explored in interaction with the SC and the cerebellum and where a feedback loop local to the SBGs might not be necessary.

3 The superior colliculus

The superior colliculus (SC) is a multilayered structure. Its superficial layers are visual, they receive direct retinal inputs and are topographically organized: their rostral parts respond to visual stimuli close to the fovea while peripheral ones activate more caudal sites (see Fig. 6 for a precise diagram of the mapping in monkeys). Neurons of this layer consequently discharge for targets situated in a limited area of the visual field -the visual field of the neuron- and their activity follows a bell-shape tuning function centered around a preferred position. The deeper layers of the SC are presaccadic, they are also called visuomotor as they exhibit small visual bursts before the motor bursts, which are similar to those of the visual cells and usually not sufficient to generate a saccade. They are also topographically organized: the neurons discharge before saccades directed to a specific region of the visual field -the movement field of the neuron- which is in register with the visual fields of the visual neurons situated above them.

The neurons of the intermediate and deep layers are classified in four main categories (Mays and Sparks, 1980; Wurtz and Optican, 1994; Moschovakis, 1996; Moschovakis et al., 1996):

- The *fixation cells* are located in the rostral SC (in the monkey) they exhibit
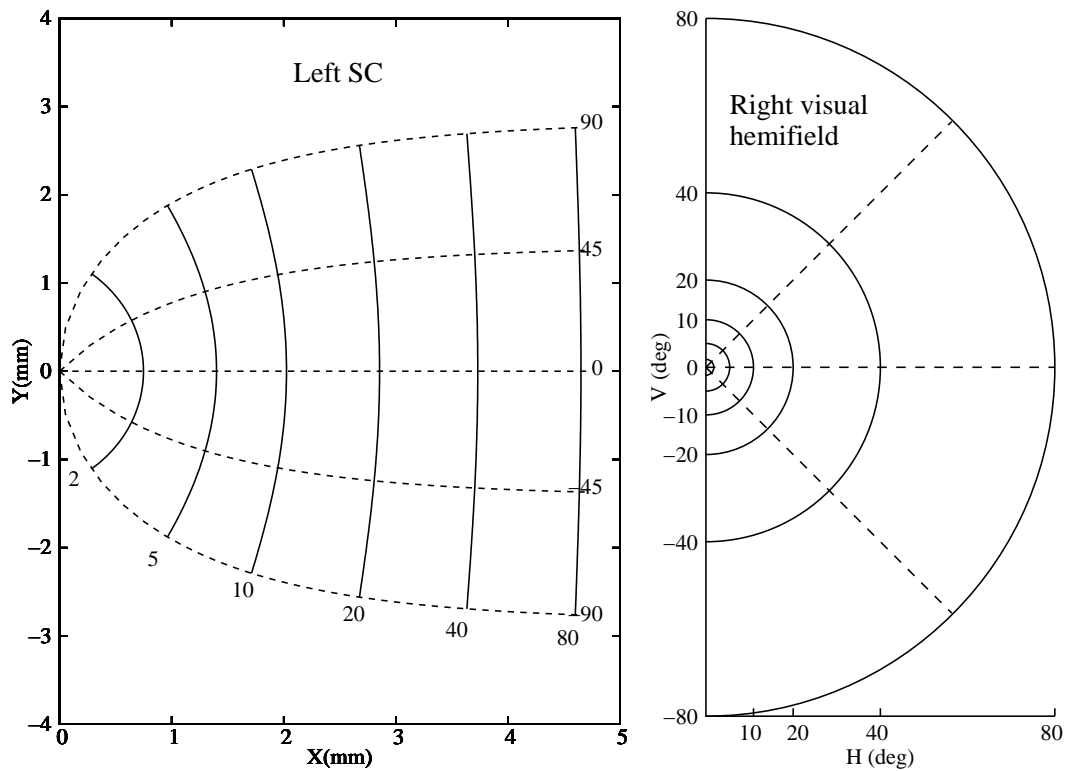
Fig. 6. Mapping from polar coordinates in visual space (right) to cartesian coordinates in the primate SC (left). Adapted from (Optican, 95).

increased activity during fixation (even in the absence of a fixation visual stimulus), stop firing during the saccades and also display continous activity during pursuit. They are considered as inhibitors of saccadic eye movements.

- The *burst neurons* (BN or tectal long-lead burst neurons, TLLB) have a very low firing rate during fixation and generate bursts of spikes immediately before saccade onset, if the saccade target is located in their movement field.
- The *build up neurons* (BUN) are located deeper in the SC than the burst cells, they also have very low firing rate during fixation, but begin to discharge at the target onset and the firing rate slowly increases until saccade onset. The movement fields of the build up cells are different from those of the burst cells: they are not centered around a prefered position, but begin at a given position and extend to any position with a larger amplitude.
- The *quasi-visual cells* (QV) are neither visual nor motor cells. They respond to visual stimulus in their visual field, like visual cells, even if a saccade towards that stimulus will not be performed. They may, unlike visual cells, continue to discharge for a while after stimulus removal, with longer discharges if the stimulus is a saccade target. They can also discharge without any visual stimulus at all if a saccade to the position corresponding the the receptive field of the cell is about to be elicited. However the activity pattern in that case is not tightly linked to the saccade onset, as in motor (BN and BUN) cells. The precise role of these cells is not clearly defined, an interpretation of their activity as a dynamic spatial memory (Droulez and

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Berthoz, 1991) of stimuli locations is proposed by Bozis and Moschovakis (1998, see section 3.14).

Models of the SC were at first interested in the description of the mappings of the visual fields on the superficial SC and of the motor fields on the deeper SC. Neural network models then allowed the study of the internal dynamics of the SC maps (implying lateral excitations and inhibition) and the possible roles of velocity and/or position feedback (simple remapping of targets after saccades or involvement in the SBG control loop by spatial integration of displacement).

3.1 Ottes et al. (1986)

In 1986, Ottes *et al.* proposed a model of the structure of collicular neural maps and of the shape of collicular visuomotor fields, based on the initial mapping experimentally obtained by Robinson (1972) with monkeys. The mapping between the position of a stimulus in the retina and the location of activated cells in the SC is based on logarithmic a function (Fig. 6) and the mapping between this location and the corresponding saccade vector is defined as the inverse function. The activity profile generated in the SC map by a stimulus is defined as a 2D Gaussian function of constant size, mapped in physical coordinates on the collicular surface. The combination of these mappings and activity profiles accurately simulates the increase of the visual and motor fields size with the distance to retina center and the skewness of their sensitivity profile.

This quantification of mapping of the visual space onto the physical surface of the colliculus was essential to further proceed with quantitative modelling of the working superior colliculus. As a consequence, it was extensively used in subsequent studies.

3.2 van Gisbergen et al. (1987)

Van Gisbergen *et al.* modeled the ensemble coding of movement in the collicular map. They used the SC mapping and population activity proposed by Ottes *et al.* (1986) and tested the hypothesis that the resulting saccade vector is the sum of the saccade vectors associated to each SC visuomotor cell, weighted by the cell activity.

The choice of using a vector sum to define the resulting saccade has many implications. The first is that limited lesions (1mm in diameter) of the SC don't eliminate the corresponding saccades from the saccades repertoire, as the stimulation is larger than the lesion and therefore generates some activity in the SC cells. However, saccades whose population activity partially overlap

the lesioned region are systematically hypometric, as the vector sum lacks the contribution of the lesioned cells. It was later observed (Lee et al., 1988) that in such a situation, saccades "diverge" from the lesion site, which does not automatically result in hypometric saccades.

An other effect of summation is that it causes edge effects that limit the maximum amplitude of saccades as stimulation Gaussians close to the limits of the SC are truncated.

Finally, in case of double stimulation, the resulting saccade is the sum of the the two vectors associated to the sites of stimulation instead of the average. This last point was solved in the 1989 model (van Opstal and van Gisbergen, 1989).

3.3 van Opstal and van Gisbergen (1989)

Following the 1987 model, van Gisbergen's team proposed an improved version of it, integrating a nonlinearity in the collicular cells firing rate and some lateral inhibitions whithin the SC.

The collicular cells firing rate is simply set to 0 for negative activations and depends linearly on positive activations until it reaches its maximum (500 spikes/s). This allowed them to reproduce qualitatively the fact that the amplitude of electrically-evoked saccades depends on the strength of the current and does not increase further beyond a certain strength.

As various results suggested the existence of lateral long range inhibitory interactions within the SC, these were simply modelled by the addition of global inhibition constants generated by each active cell on every other cells. This modifies the shape of the activity profile in the SC and reduces its global level of activation in the case of double stimulation. The amplitude of a saccade in the case of double stimulation is therefore reduced, so that it corresponds to an average saccade weighted by the relative current strength rather than a vector sum.

These modification produce a more realistic model, however, the effect of lateral short-range excitations and long-range inhibitions was modelled as purely static. Consequently, the resulting SC activity profile remained bimodal. Following models (Arai et al., 1994, for example) using a neural network approach showed that the simulation of the dynamic of such a connection pattern can results in unimodal activity, depending on the strength of the connections.

In a early work, Tweed and Vilis (1987) studied the effects the noncommutativity of the rotations has on 3D eye movements, and especially the fact that the derivative of orientation is not angular velocity. To solve the problem of computing head orientation from vestibular velocity signals, they proposed a transformation based on the use of quaternions[1]. They also showed that the simple integrators used in 1D feedback models of control of saccades should be replaced by such systems in 3D models.

This led them to propose a model specifically dedicated to quaternion-coded saccades. This has the advantage of solving the problem of rotation noncommutativity, of simply explaining why the amplitude of a saccade is affected by stimulation position in the SC map only, and of replicating weighted averaging resulting from double stimulations.

The model SC is composed of map of neurons without lateral connections. As each SC cell codes for a particular eye rotation, it sends four projections to the SBG, whose relative strengths code the four components of the corresponding quaternion. The angle coded by a quaternion is not affected by multiplication by a nonzero scalar, therefore, variations in the frequency of activation of the SC cells do not change the metrics of the generated saccade. The generation of weighted averaging saccades in the case of double stimulations with different frequencies directly derives from the same property. Finally, Tweed and Vilis proposed that extending their model to take into account the Listing law[2] would result in the addition of a dedicated module situated upstream from the SC.

Solving the problem of the noncommutativity of rotations with quaternions is quite elegant. However, the discovery of orbital pulleys and of their role in changing the axes of action of the extraocular muscles depending on eye position may simplify the problem. Indeed, Quaia and Optican (1998) showed that if the pulleys are correctly placed, the saccadic circuitry can be commutative, because the eye plant itself converts the neural signal into the appropriate rotation of the eye. Moreover, in such a configuration, the implementation of the Listing's Law is simplified. Nevertheless, Tweed and Vilis (1990) rose the problem of integrating rotations in 3D, often neglected in earlier -and also most of the following- studies.

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[1] Quaternions are a four-component extension of complex numbers and are of common use to represent rotations and rotations composition.

[2] The eyes have three degrees of rotational freedom. The Listing's Law however states that, to avoid eye torsion, the static positions that the eye can assume are described by Euler axes that lie in a plane perpendicular to the primary position, called Listing's plane.

Droulez and Berthoz proposed a neural network model of a dynamic spatial memory. It is not explicitly modelling the SC, but is a theoretical proposal of generic premotor mechanisms used to store the position of targets and to update this position according to movements performed. These might occur in the SC but also in cortical areas.

Considering the case of saccade-related brain structures, the dynamic spatial memory is a 2D retinotopic map of interconnected neurons, each of them coding for a specific eye displacement. Lateral short-range bell-shaped excitatory connections ensure that any input, representing the position of a target, is stored as a hill of activation centered around the neuron representing this position (Fig. 7, A). The position of these hills is affected by velocity feedback (efferent copy of motor commands), allowing the remapping of target positions during the execution of saccades (Fig. 7, B).
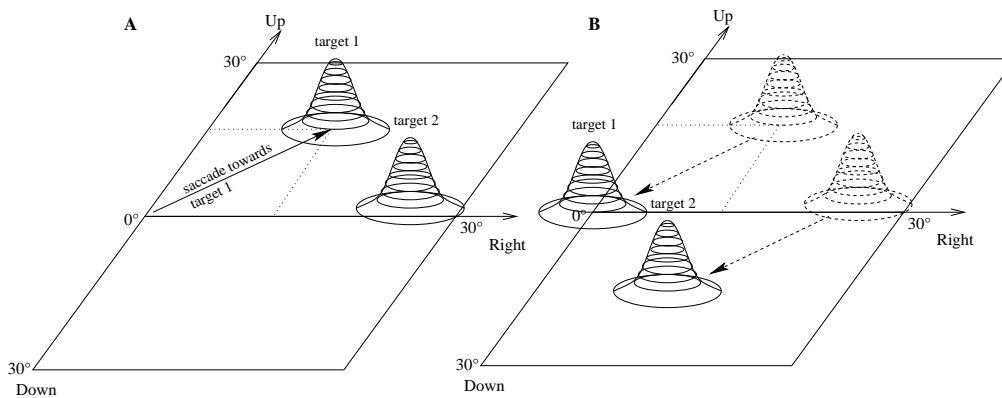
Fig. 7. Principle of *moving hill* models of the SC. A: the position of potential targets is stored as hills of activations in the retinotopic map. B: the positions are continuously updated during the execution of a saccade towards target 1, thanks to velocity feedback, until the corresponding hill of activation reaches the center of the map.

Such a mechanism is not limited to target remapping: the continuous update of the position of the potential targets allows to specifically track the current target position during the execution of the saccade. Therefore, a SC model based on that principle could be integrated in the feedback loop of the saccadic burst generator, and thus provide an implementation of a principle first proposed by Keller (1980), but computationally unspecified at that time. This idea of *moving hills* was included in many following SC (Lefèvre and Galiana, 1992; Optican, 1994; Grossberg et al., 1997) and cortical map (Dominey and Arbib, 1992; Mitchell and Zipser, 2003) models.

Waitzman *et al.* studied the variation of the firing rate of TLLBs during the execution of saccades directed to their movement fields and discovered that there is a quasi-linear relationship between this rate and the radial error. This result led them to propose a model including the SC inside the feedback loop of the saccadic burst generator.
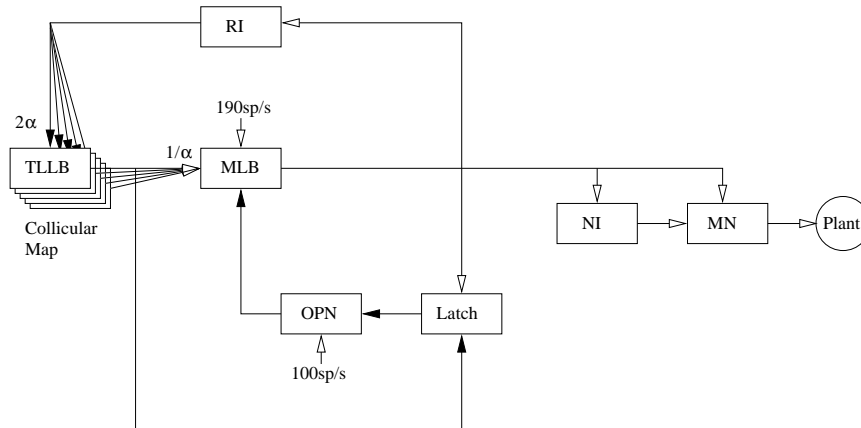
Fig. 8. Waitzman et al. model of the SC. The TLLB are placed inside the feedback loop. Filled arrows : inhibitory connections; empty arrows: excitatory connections. Parameter α is specific to each TLLB, as it varies with the amplitude of the saccade.

Their model is a modification of Jürgens *et al.* model of the saccadic burst generator, where the resettable integrator which provides position error feedback performs an inverse temporal to spatial transform before projecting to the SC (Fig. 8). The dynamic of this model is different from the Droulez and Berthoz *moving hills*, and could instead be described as a *stationary decreasing hill*.

Including the SC in the feedback loop necessitates to proceed to both spatial to temporal (SC output) and temporal to spatial (RI output) transformations. The potential functional advantages derived from such a structure were not assessed. Moreover, Moschovakis showed in his 1994 study of the saccadic burst generator (see section 2.7) that it is possible to reproduce the relationship between TLLB firing rate and radial error with both Scudder's and his own model, where the SC is kept outside the feedback loop. The inclusion of the SC into the feedback loop is thus not necessary to explain the Waitzman *et al.* experimental results.

3.7 Dominey and Arbib (1992)

In 1992, Dominey and Arbib proposed a model including a large part of brain circuitry involved in saccade generation (saccadic burst generator, superior colliculus, basal ganglia, frontal eye fields and parietal cortex), to which was added, in 1995 (Dominey et al., 1995), reinforcement learning and working memory capabilities and, in 1996 (Schweighofer et al., 1996a,b), a cerebellar module. Concerning the SBG, they used Scudder's model (see section 2.6), the rest of the model is made of original proposals. The SC part of their model is reviewed here, as the BG, cerebellum and cortex components will be treated in the next sections (see sections 5.1, 4.3 and 6.2).

The proposed SC model is made of 4 retinotopic layers (Fig. 9). The first one represents the superficial layer of the SC, it receives direct retinal input. The second one represents the quasivisual cells (QV) of the deep SC, these cells are supposed to be the substrate of the dynamic remapping of targets positions after a saccade. This layer operates qualitatively like the dynamic spatial memory of Droulez and Berthoz, however, the remapping itself does not occur in the SC but is under control of QV-like cells in the parietal cortex and it uses a position instead of a velocity feedback. The third layer receives excitatory inputs from the FEF carrying information about visible and memorized targets. The last layer receives excitatory inputs from the three previous ones and is gated by the basal ganglia selective disinhibition. In addition to this selection, a winner-takes-all (WTA) mechanism, whose neural substrate is not specified, selects the actual target of the forthcoming saccade.

This SC model is the first one to include to include the QV cells and the influence of the basal ganglia over the SC (tonic inhibition with selective disinhibition of restricted areas of the SC map). However, as if this BG selection was not sufficient, a final WTA is added. More recent models showed that the BG selection is sufficient (Bozis and Moschovakis, 1998; Brown et al., 2004) and the WTA therefore unnecessary.

3.8 Lefèvre and Galiana (1992)

Lefèvre and Galiana explored further the idea that the SC could be included in the SBG feedback loop and proposed a SC-centered model belonging to the *moving hill* family.

The model SC is one dimensional map of laterally interconnected neurons (short-range excitations) receiving a gaze velocity feedback directed to its most caudal sites only. This map drives a model including eye and head control (modified version of (Galiana et al., 1992) model of eye-head coordina-

Fig. 9. Dominey and Arbib (1992) model of the saccade-related brain circuitry, the extensions from Dominey *et al.* (1995) are included, the brainstem saccadic burst generator is an implementation of Scudder 1988 model. Each layer represents 2D surfaces of topographically organized neurons, except for PFC and IT (see text). CD: caudate nucleus; FEF: frontal eye fields; LGN: lateral geniculate nucleus; PFC: prefrontal cortex; PPC: posterior parietal cortex; SC: superior colliculus; SBG: reticular formation saccadic burst generator; SNr: substantia nigra pars reticulata; TH: thalamus; V4: extrastriate visual cortex area 4; VisCx: visual cortex; fx: fixation cells; mem: memory layer; mot: motor layer; qv: quasivisual layer; vis: visual layer; wta: winner takes all layer. Filled arrows : inhibitory connections; empty arrows: excitatory connections.

tion) through linearly weighted projections (increasing with the distance to the fovea). However the amplitude of produced saccades does not depend linearly on the stimulation position on the SC map. This emerging property is somewhat reminiscent of the non-linear mapping modelled by Ottes et al. (1986), however a bidimensional extension of the model would probably be necessary to compare this non-linearity with the logarithmic mapping of the SC.

In this model, the gaze-velocity feedback is supposed to be provided to the SC by projections from the prepositus hypoglossi. The authors however mention

that (1) the projections from the prepositus hypoglossi reach the whole SC rather than its caudal part only, distributed feedback over the SC should therefore be modelled, and (2) prepositus hypoglossi cells carry a mixture of eye position, eye velocity and gaze velocity, a property that will be fully exploited in the Arai et al. (1999) model. Finally, even if it is beyond the scope of this report, note that this model is the first SC model dealing with gaze control during saccades, thus including both eye and head dynamics.

3.9   Krommenhoek et al. (1993, 1996); Krommenhoek and Wiegerinck (1998)

Krommenhoek *et al.*, also considering that the SC is in the saccadic burst generator feedback loop, studied in 1993 the question of the necessity of a step of craniocentric coding (a proposition from Robinson (1975)) in order to solve the remapping of activity in the SC. To do so, they used a methodology very similar to the one used by Zipser and Andersen (1988) in their model of area 7a of the PPC (see section 6.1). They trained (back-propagation algorithm) two nonlinear feedforward neural network architectures to produce the actual motor error coded in retinocentric coordinates as a hill of activation on a 2D map, using the initial retinal error and the initial and actual positions of the eye in craniocentric coordinates.

They showed that the simplest model (all inputs connected to a single hidden layer) does not have to explicitly compute to the position of the target in craniocentric coordinates in order to achieve the task. Indeed, the eye displacement, which does not depend on the reference frame, can be obtained by substracting the two eye positions in craniocentric coordinates. Consequently, the activity in the resulting hidden layer is a distributed coding of the motor error in retinotopic coordinates. The second network first combines the retinal error and the initial eye position in a hidden layer before a second hidden layer combines the output of hidden layer 1 with the actual eye position. In that configuration, the first hidden layer codes the target position in craniocentric coordinates and the second codes the motor error in retinocentric coordinates. The role of these hidden layers is proposed to be similar to the one of, respectively, area 7a and LIP in the posterior parietal cortex.

In these experiments, the hidden layers were not 2D maps but simple populations of neurons witout any topological significance. Consequently, this study proposes an interesting framework for analysing the gain field coding of coordinates by populations of neurons, which was further developed in the 1996 paper (Krommenhoek et al., 1996). However, it might seem a bit unrealistic to consider that parietal cortex is incorporated in the saccadic burst generator feedback during the execution of sacades, because of the long delays implied by such a large loop.

The 1998 paper (Krommenhoek and Wiegerinck, 1998) studies the behaviour of the model in a situation of double stimulation. It is capable of generating an averaging saccade, this averaging takes place in the first hidden layer (representing area 7a of posterior parietal cortex) and not in the SC. It is influenced by the total stimulation intensity and the initial eye position. The model is however unable to account for the bistability (the generated saccade is directed to either one or the other target) obtained in double stimulations when the locations of stimulation are more than 30° away or when the latency is longer than 300ms.

3.10   Arai et al. (1994); Das et al. (1995); Arai et al. (1999)

Arai *et al.* proposed in 1994 a model where the SC is incorporated to the feedback control loop, belonging to the *decreasing hill of activity* family. Its architecture is quite similar to other proposals, its main originality is that the weights of the lateral connections in the motor layer of the SC were obtained by a supervised learning algorithm using real neurons data as training set.

The network is made of two layers: the visual-only layer relays the visual input to the motor layer. The cells of the motor layer are laterally interconnected and project to a SBG model belonging to the Jürgens *et al.* family of models (driven by eye displacement and using a resettable integrator). The weights of the lateral interconnections, which where generated by supervised learning based on electrophysiological recordings, consisted in short-range excitation and long-range inhibitions. Feedback from the SBG was provided as additional input to the motor layer, the learning algorithm converged (*i.e.* satisfactory duplication of real cells activity profiles) for both velocity or position feedback. The excitatory weights of the projections to the SBG were also obtained by supervised learning, but using a criterion of resulting saccade accuracy. A global shunting inhibition over the SC is used to control the onset of the saccade. This inhibition is supposed to be generated by either the FEF or the basal ganglia, and canceled during the generation of the saccade. It however lacked the spatial selectivity one may expect from basal ganglia output.

This model generates saccades in accordance with the main sequence, it can generate express saccades (saccades with a very short latency) if the inhibition is absent and accurately accomplishes saccade averaging. As a result of the learning algorithm used, the activity profile of the motor layer neurons is qualitatively similar to recorded cells.

In the case of saccade interruption by OPN stimulation, the model was not able to resume saccades correctly. However, refinements of this initial model (Das et al., 1995; Arai et al., 1999) resulted in better results, albeit a slight

hypermetria tendency remained and the SC neurons did not completely turn off during the interruption, in contradiction to experimental data. The last version of the model used both velocity and eye displacement feedback. The weights of these feedback connections over the SC are learnt using a back-propagation algorithm which gives better results using both velocity and displacement. This led the authors to suggest that neurons carrying feedback to the SC might not carry pure velocity or displacement signals, but a mixture of both.

3.11   Optican (1994)

In 1994, Optican proposed a model preserving the idea that the SC might be in the saccade trajectory control feedback loop. However, where models with the SC outside the loop produce a desired displacement signal and models with the SC inside the loop produce the displacement error, Optican suggests that the SC produces both a desired displacement signal and an actual displacement signal. The first one is produced by the BNs, which are unaffected by velocity feedback, and the second one by the BUNs, which thus constitute a spatial equivalent of the RI in the Jürgens *et al.* family of SBG models.
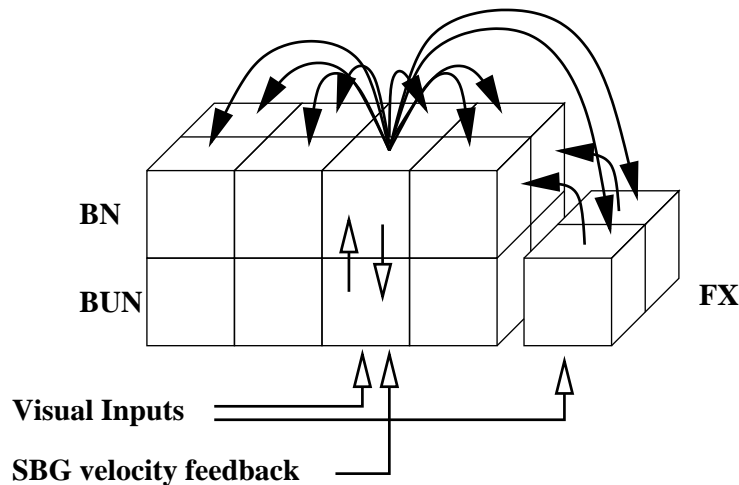

Fig. 10. Optican 1994 model of the superior colliculus. BN: burst neurons; BUN: build-up neurons; FX: fixation cells; SBG: brainstem saccadic burst generator. Filled arrows: inhibitions; empty arrows: excitations. Adpated from (Optican, 1994).

The model is composed of two layers (Fig. 10). The first one contains the BUNs and the fixation cells (fixation cells are located centrally and inhibit the second layer), it receives sensory inputs signaling the presence of potential targets and velocity feedback provided by the vertical and horizontal saccadic burst generators. The second one is the BNs layer, these have lateral inhibitory connections, implementing a spatial winner-takes-all. The two layers are inter-connected by bidirectional excitations which connect BN and BUN neurons

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having the same movement fields. The difference between the BUN layer signal (actual displacement) and the BN layer signal (desired displacement) is computed in the horizontal and vertical saccadic burst generators.

This model is the only one which integrates actual displacement rather than displacement error in the SC, so that the error is computed in the SBG. This specificity, which was never reused in following models, has the advantage of proposing different functional roles for the BNs and BUNs.

3.12   van Optsal and Hepp (1995)

Building on experimental results showing that the activity of the neurons in the deep layers of the SC are modulated by initial eye position, van Opstal and Hepp (1995) proposed a very simple model of the SC motor map which could produce the three-dimensional rotation axis for saccades in Listing's plane.

The model SC is a retinotopic map where every movement field considered is covered by nine cells. Each of these nine cells has the same sensibility to eye displacement in its movement field as its eight neighbors, but this basic pattern is modulated by eye position (what Zipser and Andersen (1988) defined as gain fields, see section 6.1) in a specific way, as each cell has a specific preferred eye position. The gain field modulation is 1% of the cell's peak firing rate per degree change in eye position. This map simultaneously encodes the desired eye displacement (spatially) and the eye position (by gain fields). Van Opstal and Hepp showed that projections to a simple perceptron trained using the Widrow-Hoff learning rule allowed the decoding of both variables and also the computation a a good approximation of the ocular rotation axis in Listing's plane.

Despite its extreme simplicity with regards to known anatomy and physiology of the SC, this model suggests an interesting role for gain fields: allowing the computation of rotations compliant with Listing's law at the level of the SC.

3.13   Grossberg et al. (1997)

As the superior colliculus is an area of multisensory integration, Grossberg et al. proposed the SACCART model, specifically dedicated to that aspect of the SC operation. As depicted in Fig. 11, their model contains both a burst neurons map (*stationary decreasing hill of activity*) and a build-up neurons map (*moving hill of activity*).

The BN map receives an input from the retina via the superficial layers of the
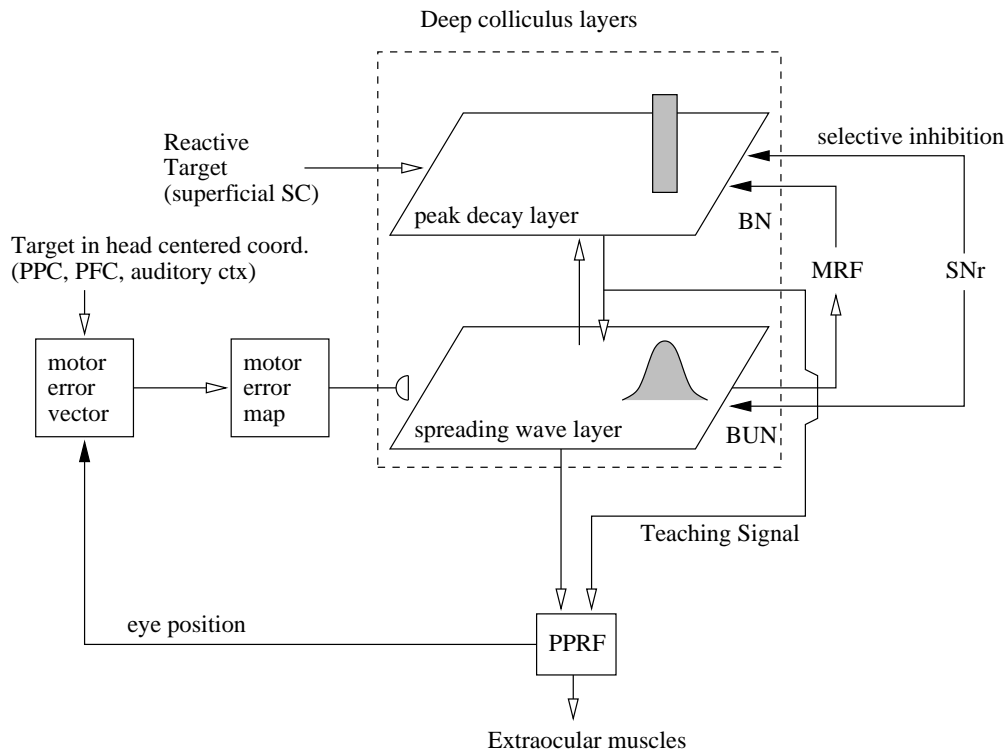
Fig. 11. SACCART model (Grossberg *et al.*, 97) of multisensory integration in the SC. Filled arrows : inhibitory connections; empty arrows: excitatory connections. Adapted from (Grossberg *et al.*, 1997).

SC and is therefore capable of initiating visual saccades on its own. This visual saccade part of the SC is used for the calibration of the connections between the SC and the SBG, as a visual feedback is available at the end of the saccade to evaluate ist accuracy, which is not the case for memory saccades.

The BUN map is in charge of integrating the information for the non-visual saccades, from the PPC (attentional system able to store one target location), the PFC (working memory storing sequences of saccades) and the auditory cortex (auditory saccades). The target locations in these areas are supposed to be coded in craniocentric coordinates and the transformation into retinocentric coordinates substracting eye position is made by specific weighting of their projections to BUN map, obtained by learning. These non-visual saccades do not necessarily have a visual feedback available at the end of the saccade to be calibrated, they therefore take advantage of the accuracy developped by the visual pathway.

Three types of inhibitions affect the activity on both SC maps and limit the spatial extend of the reverberation generated by their excitatory interconnections: a global shunting inhibition from the mesencephalic reticular formation, a time-selective inhibiton from the SC fixation cells and a spatial selective inhibition from the basal ganglia.

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This model was tested in simple visual saccades and also in memory, overlap, gap and multimodal tasks. It is the first one to study the multimodality of the SC and the associated problem of calibration, it also provides an interesting interpretation of the respective roles of BNs and BUNs, different from the Optican (1994) proposal.

3.14   Bozis and Moschovakis (1998)

In his 1994 model of the SBG -called MSH-, Moschovakis showed that the correlation between the variation of the firing rate of the TLLB and the displacement error does not necessarily imply that the SC is in the control feedback loop. The Bozis and Moschovakis model of the SC is therefore built on the MSH and isn't included in this feedback loop.

The model is composed of two main layers. The first one is a motor TLLB layer very similar to earlier models (van Opstal and van Gisbergen, 1989; Arai et al., 1994; Das et al., 1995; Grossberg et al., 1997), where localised hills of activity are generated by local lateral excitations and global or long-range inhibition. The weight distribution of lateral excitations is derived from synaptic button density measures. The global inhibition depends on the total TLLB activity and is provided by speculative inhibitory neurons. The dynamics of this layer is however different from previous proposals: the TLLB-inhibitory neurons loop is supposed to give rise to an oscillating activity, where the hill of activity on the map repetitively grows and vanishes as long as there is an excitatory input on a single TLLB. An additional inhibition provided by basal ganglia gates the saccade onset and offset, allowing a single oscillation to occur. The detailed mechanisms of this oscillating system are however unclear, and thus difficult to replicate.

The second layer of the model is composed of the visual cells of the superficial SC, which contact a complex network of predictive visual and quasivisual cells in the deep SC. This network works as a spatial memory which projects to the TLLB layer and where the position of disappearing visual target is stored. An eye displacement feedback from the SBG is capable of shifting the hills of activity of this memory in order to update the position of targets after a saccade occured. This circuit operates in a qualitatively similar manner as the dynamic spatial memory proposed by Droulez and Berthoz (1991), however this feedback is not used for the control of the occuring saccade metrics as in (Lefèvre and Galiana, 1992), but only for the memory update as in (Dominey and Arbib, 1992). The choice of the targets to be stored in this memory is supposed to be the result of the gating of a second group of basal ganglia inhibitory cells which where shown to decrease their activity at the presentation of a target and to recover their tonic rate only after a saccade

towards that specific target. This selective inhibitory mechanism is however unspecified by the model. Bozis and Moschovakis acknowledge the fact that such a dynamic memory also probably exists in cortical areas, but they propose that they are parallel and complementary rather than hierarchically organized (Dominey and Arbib, 1992).

This model exhibits saccade averaging in the case of double stimulation, amplitude modifications in the case of local lesions which are compatible with the results from (Lee et al., 1988), and finally, correct vector substraction in double saccade task. It does not include BUN, however the authors note that slightly modifying the paramaters of the model allows the generation of activities in the TLLB layer similar to BUN activity.

3.15   Anastasio et al. (2000); Patton et al. (2002)

Anastasio and colleagues are interested in multisensory integration in the SC, and more specifically in the so-called multisensory enhancement. A subset of neurons from the deep layers (about a quarter of them in primates) are multisensory, as they are affected by inputs from various sensory modalities (visual and auditory, for example). Stimuli occuring at the same time and place can increase the response of a multisensory neuron with regards to its response to only one of these stimuli (multisensory enhancement). This enhancement, defined as the ratio of the discharge increase in a combination of multisensory inputs over the maximal discharge obtained with only one of the sensory component, is larger for small inputs than for large ones (inverse effectiveness). The model they propose focuses on these multisensory neurons only, trying to grasp these two properties.

The model is built in a Bayesian framework, it is hypothesised that the deep SC neurons compute the probability that a target is present in their receptive fields given the sensory inputs and prior knowledge about environment (target appearance frequency) and sensor (reliability) properties. Three likelihood functions of the input firing rates (no target present, a target in one modality or in another one) are modelled by three Poisson distributions with different means. The larger the difference between the mean of the "no target" distribution and the two others, the easier the discrimination between the two possibilities (target present or not). When this difference is small, the distributions overlap on a wide firing rate range, and on that range, discrimination is poor. However, when two input modalities are available, even if the overlap is large for both of them, an enhancement of the discrimination is obtained, which is subject to inverse effectiveness. Finally, an information theoretic analysis (Patton et al., 2002) suggests that the numerous unimodal neurons might be those for which the means of the distributions are separated

enough, so that a good discrimination between target absence or presence can be obtained, and for which the addition of a second modality does therefore not add much information.

This work is an interesting first step in modelling the SC using a Bayesian framework. It does however not currently explore the mechanism that would allow neurons to carry out the Bayesian computation, it is restricted to the computation of the deep SC neurons (BN or BUN) and should be extended to other neurons and structures. Finally, the hypothesis that the deep SC neurons compute target presence probability is quite simplifying, knowing that the activity of these neurons is rather sensorimotor and reflects a target selection process, probably mediated by the projections from the basal ganglia.

3.16   Trappenberg et al. (2001)

Trappenberg *et al.* studied in 2001 the influence of the lateral connections in the SC (short-range excitation and long-range inhibitions) on the saccade response time (SRT) in various experimental setting.

Their model is made of two layers: the first one simulates the laterally inter-connected SC fixation and build-up cells, it integrates both exogenous and endogenous stimulations; the second one simulates the burst cells and is under control of the previous layer (BN discharge when the BUN with the same movement field reaches 80% of its maximum firing rate). Exogenous stimulation are purely visual stimuli, while endogenous stimulations are the results of cortical processes taking into account visual stimuli, task context and *a priori* knowledge of target position. The lateral connections in the first layer have a short-range excitation/long-range inhibition pattern similar to those of earlier models (van Opstal and van Gisbergen, 1989; Arai et al., 1994; Das et al., 1995; Bozis and Moschovakis, 1998). The connection weights are here tuned using indirect experimental data: a distractor is presented after the fixation point offset and before the target onset, the activity profile of the SC cell corresponding to the target is then amplified or inhibited, depending on distance between target and distractor; if one supposes that this modification is caused by the activity of the cells corresponding to the distractor through lateral interconnections in the SC, an approximation of their weighting can be deduced.

The dynamic generated by these lateral interconnections can explain various effects. Concerning the gap effect, the reduced reaction time is caused by the removal of the fixation point which implies a cancelation of the fixation cells long-range inhibition over the build-up cells, which therefore reach the threshold of BN activation faster. The model also explains the fact that the

inhibitory or excitatory effects of a distractor is stronger when it is displayed in close temporal proximity with the target. Antisaccades were simulated by first sending the exogenous visual stimuli to the SC, followed by an endogenous signal exciting the cells corresponding to the opposite saccade: the build-up activity generated by the visual signal is not strong enough to generate a saccade on its own, it however inhibits the endogenous signal and thus justifies the increased SRT. *A priori* knowledge of the target position was simulated by an endogenous input preceding the visual input and exciting the cells of the area with the highest probability of target onset. It increased or decreased the SRT depending on the actual position of the target.

This model, which is structurally close to earlier ones, proposes a detailed study of the effects of the lateral interactions in the SC on SRT, providing a link between saccade behavioural studies and the corresponding neural substrate.

3.17   Discussion

The modelling of logarithmic visuomotor mapping of the monkey SC did not generates much controversy, but wasn't much used in models (see Tab. 2, MAP column). Concerning the transformation of this SC spatial code into the SBG temporal one, the so-called *spatio-temporal transformation* or STT, most of the models built on the idea of van Gisbergen et al. (1987). It is based on a spatially (and often also temporally) stereotyped hill of activity on the SC, which can easily be decoded using an appropriate weighted sum of this activity. However assuming such a perfect stereotypy is a bit unrealistic, given the experimental results concerning SC population activity (Anderson et al., 1998). Without stereotypy, an additional normalization step is needed. While Optican and Quaia (2002, see Section 4.4) thus prefer to negate the existence of a STT and to delegate the whole task of controlling the amplitude and direction of saccades to the cerebellum, Groh (2001) proposes an architecture that explains microstimulation results and should be further explored in future SC models. The question of how precisely vertical saccades and saccades close to the vertical, probably involving activity on both colliculi, are coded in the SC and decoded in the STT was rapidly addressed in (van Gisbergen et al., 1987) and is not solved yet.

Noncommutativity of 3D rotations and implementation of the Listing's Law have been important preoccupations of early modelers. However, the use of quaternion coding, proposed by (Tweed and Vilis, 1990) in order to cope with the noncommutativity, is probably not necessary. Quaia and Optican (1998) showed that orbital pulleys may convert the command of a commutative saccadic circuitry into the correct eye rotation. Moreover, the pulleys also

Table 2
Topics addressed by the reviewed superior colliculus models.

Model	Model features			Evaluation criteria			
	MAP	LAT	FBL	LES	DSA	MTR	MMS
Ottes et al., 1986	•						
van Gisbergen et al., 1987	•			•			
van Opstal and van Gisbergen, 1989	•	•		•	•		
Tweed and Vilis, 1990					•		
Droulez and Berthoz, 1991		•	•			•	
Waitzman et al., 1991			•				
Dominey and Arbib, 1992						•	
Lefèvre and Galiana, 1992	•	•	•				
Krommenhoek et al., 1993			•		•		
Arai et al., 1994	•	•	•		•		
Optican, 1994			•				
Grossberg et al., 1997		•	•				•
Bozis and Moschovakis, 1998		•		•	•	•	
Anastasio et al., 2000							•
Trappenberg et al., 2001		•					

MAP: modelling of the topological mapping of the SC; LAT: simulated lateral inter-connections in collicular maps; FBL: SC included in the SBG feedback loop; LES: effects of localised SC lesions; DSA: double stimulation averaging; MTR: multiple targets remapping; MMS: multimodal saccades.

simplify the implementation of the Listing's Law: as long as the pulse vector, defined by the horizontal and vertical EBN activity, lies in Listing's plane, the Listing's Law is respected. This adjustment of the pulse might be generated by a simple gain-field coding in the SC, similar to the proposal of van Optsal and Hepp (1995).

An important progress in the understanding of the intrinsic computations of the SC came from the evolution of the modelling methodology. In the first relatively abstract models (van Gisbergen et al., 1987; van Opstal and van Gisbergen, 1989), the temporal dimension was neglected, while the explicit simulation of the neural activity opened a new pathway and allowed the study of the evolution of SC activity during the execution of a saccade. This was of primary importance as evidence of lateral short-range excitatory and long-range inhibitory interconnections in the collicular maps was accu-

mulating. Numerous models explicitly include such connections (see Tab. 2, LAT column), and attribute to them various roles : spatial memory (Droulez and Berthoz, 1991; Arai et al., 1994), shift of activity locus during saccades (Droulez and Berthoz, 1991; Lefèvre and Galiana, 1992), generation of oscillatory activity (Bozis and Moschovakis, 1998) or influence over the selection of saccade target (Trappenberg et al., 2001). However, beyond these various –and sometimes contradictory– simulation results, a theoretical study of the intra-map dynamics is now needed.

After the relative failure of a purely SBG-based feedback loop, the possible inclusion of the SC in this loop was studied and is still debated. Models addressing it also question the relative roles of the two populations of motor cells (BN and BUN).

The correlation between the BNs firing rate and the remaining displacement error inspired the *stationary decreasing hill* family of models including the SC in the SBG control loop (Waitzman et al., 1991; Arai et al., 1994; Das et al., 1995; Arai et al., 1999). These models were however contradicted by the results obtained in paradigms, where saccades are interrupted by either OPN stimulations or eye blinks (Keller and Edelman, 1994; Goossens and van Opstal, 2000), as the monotonic relations between instantaneous firing rate and dynamic motor error is lost in the resumed activity. It was moreover shown (Moschovakis, 1994) that this correlation can be obtained in models lacking causal relationship, *i.e.* where the SC is outside the feedback loop.

The "open" motor fields of BUNs (with a lower but not an upper bound in amplitude) along with their slowly increasing firing rate led to the *moving hill* family of models, where the remaining displacement error is monitored by the continuously updated position of the hill of activity with regards to the foveal area of the map (Droulez and Berthoz, 1991; Lefèvre and Galiana, 1992). These models also suffer from flaws: the center of gravity of BUN activity does not significantly move on the SC map and this spreading activity starts before saccade onset (Anderson et al., 1998), and in interrupted saccades, the locus of the resumed activity does not move rostrally as predicted, but stays at the initial position (Keller and Edelman, 1994; Goossens and van Opstal, 2000).

Finally, the latest developments of these models led to hybrid solution assigning complementary roles to BNs and BUNs in order to ensure saccade accuracy (Optican, 1994; Grossberg et al., 1997). Electrophysiological studies (van Opstal and Frens, 1996) however revealed that collicular activity undergoes large changes depending on the modality of the saccade (activity decreased down to 70% or increased up to 350%) which do not change the saccade properties. The existence of a feedback from the SBG does not necessarily imply that it is used to control saccade dynamics: it can also be useful for simply remapping

the position of multiple potential targets on collicular maps. This was included in some models (see Tab. 2), and in particular in the Bozis and Moschovakis (1998) model, which was the only one to include the QV cells as the substrate of this dynamic spatial memory.

The different models reflect different views of the respective functions of the BNs and BUNs. In (Optican, 1994), they contribute to two different SC outputs (BNs produce a desired displacement and BUNs actual displacement). In (Grossberg et al., 1997), the BNs are used by visual saccades and subserve the calibration of the saccadic system, while the BUNs are the interface with other modalities. Finally, Quaia *et al.*(1999), in their cerebellum-centered model (reviewed in section 4.4), suggested that they are in fact the two extremes of a continuum of neurons targetted by different cortical areas, some providing saccadic command (burst activity) and the others anticipatory saccadic plans (build-up activity). In that case, they would be functionally indistinguishable as long as saccade execution is concerned.

In addition to the various ways they implement the STT, exploit the lateral interconnections and consider the respective roles of BNs and BUNs, the reviewed models can be distinguished by the various SC properties they simulate (see Tab. 2):

- the effect of local lesions: the metrics of saccades evoked by stimulation of cells close to the lesion are shifted away with regards to the metrics of the saccades associated to the lesioned area.
- the double target averaging: short-latency saccades evoked by two targets close from one another tend to end in a position between the two targets, in the case of double stimulation of the SC, the ending position is the barycentre of the two positions, weighted by stimulation current strength.
- multisensory enhancement and inverse efficiency: multisensory neurons' activity is increased by inputs from various sensory modalities compared to the single modality activity; this enhancement is relatively larger for weaker inputs.
- gap effect and other SRT effects: saccade reaction time is affected by the suppression of the fixation point before the presentation of the target (gap effect), the presence of distractors and preliminary knowledge of the task.

Future models should be able to simulate these properties properly, and could also explore a bit more the role of the QV cells. Finally, the influence of the basal ganglia over the SC is often considered as a purely external inhibitory input (Dominey and Arbib, 1992; Arai et al., 1994; Grossberg et al., 1997; Bozis and Moschovakis, 1998). However, the SC and the BG form various loops involving either the superficial or the intermediate layers of the SC (McHaffie et al., 2005). The hierarchical point of view, where the BG control the SC, is probably a too simple story, modelling effort should therefore study

the interconnection of SC and BG models to fully explain the corresponding selection processes.

4 Cerebellum

The models of the saccadic burst generators (see section 2) were often evaluated by their ability to accurately reproduce motoneuron firing patterns and, using a model of the eye plant, to produce saccades in accordance with the main sequence data recorded in primates. These models however neglected the fact that the motoneurons activity during saccades is not solely driven by the SBG, but is also significantly influenced by projections from the cerebellum. Indeed, damage or inactivation of the cerebellar afferent areas induces hypermetria, alteration of the main sequence and an increased variability of the amplitude (Optican and Robinson, 1980).
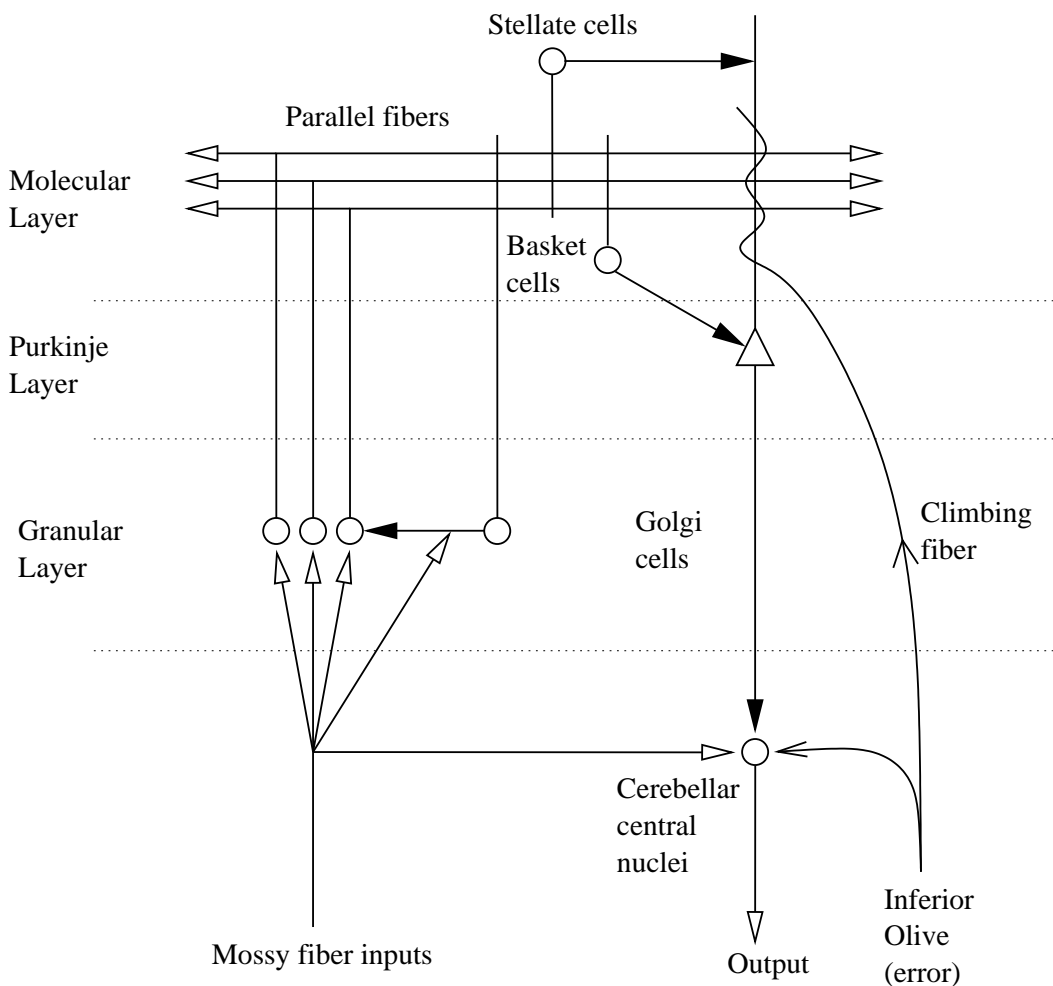

Fig. 12. Schematic diagram of the structural organization of the cerebellum. Filled arrows : inhibitory connections; empty arrows: excitatory connections.

The architecture of the cerebellum is extremely regular and very well known (Fig. 12). Inputs to the cerebellum are carried by mossy fibers which project to a huge population of granule cells, which is thought to recode these inputs; the granule cells send T-shaped parallel fibers to the superficial layer of the cerebellum; these parallel fibers have synaptic contacts with Purkinje cells dendrites; Purkinje cells project to the cerebellar central nuclei which are the ouput of the cerebellum; additional intermediate inhibitory cells play a role of activity regulation. A classical point of view considers that this structure performs supervised learning. Purkinje cells would behave like perceptrons: they have access to a large input space, and the plasticity mechanisms (long-term depression - LTD) taking place at the granulo-Purkinje synpases would be driven by teaching or error signals emitted by the climbing fibers coming from the inferior olive. This interpretation of the cerebellum's operation is however challenged by recent results concerning the plasticity of other cerebellar synapses and by the existence of long-term potentiation (LTP) processes (Hansel et al., 2001), and alternate models are accordingly being proposed (see (Medina and Mauk, 2000) and section 4.4 for example).

Numerous experimental results agree on the fact that the lobules VIc and VII of the vermis are the areas of the cerebellar cortex involved in saccade generation. These lobules draw ipsilateral and topographically organized projections towards a subpart of the caudal fastigial nuclei (a cerebellar central nuclei) called FOR (Fastigial Oculomotor Area). FOR affects the saccade generation circuitry at the level of the saccade generator, by projections to MLBs.

Models of the saccade related circuitry including the cerebellum are broadly subdivided in two classes: those derived from supervised learning modelling of the cerebellum, which are concerned with long-term calibration and adaptation of saccade gain, and those interested in the ability of the cerebellum to affect individual saccades on the fly in order to compensate for the variability of the rest of the saccadic system.

4.1 Dean et al. (1994)

The model proposed by Dean *et al.* studies the influence of the cerebellum on long-term adaptation in the brainstem cicuitry. It is inspired by Kawato's ideas on supervised learning in biological systems (Kawato, 1990) , concerning the use of the commands generated for corrective movements (here, corrective saccades) as an error signal for the supervised learning system.

Its architecure is relatively simple (fig. 13): the saccade order, issued from the superior colliculus, is sent to a SBG model of the Jürgens *et al.* family (see section 2.2) with a high feedback gain and also to a cerebellum module which
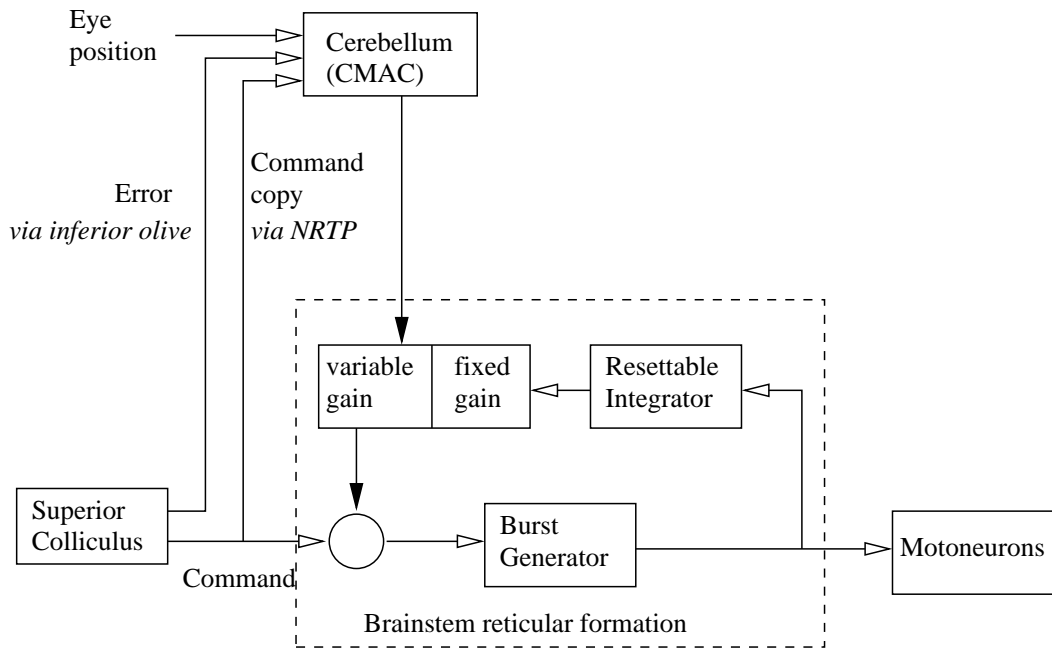
Fig. 13. Dean *et al.* (1994) model of long term adaptation of saccadic gain by cerebellar inputs. CMAC: cerebellar model arithmetic computer; NRTP: nucleus reticularis tegmenti pontis. Empty arrows: excitatory projections; filled arrows: inhibitory connections.

can modify this gain. The cerebellum is an implementation of the Cerebellar Model Arithmetic Computer (CMAC) of Albus (1971, 1981), which has to learn to produce the correct gain, given the collicular command, the initial eye position and an estimation of the error. The principle of the CMAC is to learn to produce the correct output given a specific input by simply building a lookup table. However, as a pure lookup table approach would not be tractable because of huge memory requirements when coding numerous dimensions with a high resolution, inputs are sent to parallel tables with low input resolution, and the CMAC output is the average of the output of the tables.

The amplitude of a saccade generated by a given motoneuron output varies depending on the initial stretching state of the muscle, *i.e.* the initial eye position. As the saccadic burst generator model does not adapt its command depending on eye position, the cerebellum module is successfully trained to learn to correct this effect. Satisfactory training could be achieved with a perfect error signal providing the exact gain the cerebellum should have chosen (a signal that can be generated in simulation but unavailable to animals), but it was also achieved with simpler and more biomimetic signals: the distance between actual and desired gaze position and even a +1/-1 signalling under- or over-shoot without quantitative evaluation of error.

The adaptation to the weakening of one eye muscle and to target displacement during saccade were also successfully tested. However, the experimental finding

that adaptation speed is faster in case of overshoot is not reproduced by the model, suggesting that error signals concerning under- and over-shoot might be handled differently.

This model first proposed a long-term adaptation mechanism for the saccade generation circuitry involving a model of the cerebellum. The CMAC architecture is however quite abstract and does not produce artificial neuron activations that could be compared to electrophysiological results. Finally, the mechanism that could allow the cerebellum to modify the gain of the feedback loop is not specified; experimental results tend to show that the cerebellum affects the SBG by adding excitatory or inhibitory influences on the burst rather than by gain modification (see next section).

4.2 Dean (1995)

In its 1995 work, Dean studies how the electrophysiological findings concerning FOR neurons firing patterns during saccades could be integrated in a saccadic burst generator model including the cerebellar influence.

For that purpose, he uses a bidirectional SBG model derived from the Jürgens *et al.* proposal (see section 2.2) to which a realistic 5ms delay is integrated in the feedback loop, as initially suggested by Jürgens *et al.*. A first observation deriving from this SBG model, without cerebellum influence, is that the inclusion of this delay generates a systematic hypermetria, which was overlooked in earlier SBG modelling studies and which cannot be corrected by a simple modification of the feedback gain. This SBG model is then modified in order to produce the degradations concerning hypermetria and main sequence alteration observed in cerebellum inactivation studies. This is done by decreasing the feedback gain and increasing the slope parameter of the EBN transfer function.

To correct the hypermetria, the simulated activity of the FOR neurons during a saccade is projected on the EBNs of the SBG model. Actual FOR neurons have a tonic baseline activity, produce bursts during contra- and ipsilateral saccades and sometimes pause either before or after the burst elicited for a large saccade. The variability of firing pattern between cells and for the same cell during different saccades is considerable. As only the bursts of activity seem to be related to saccades parameters, the tonic and pause activities were ignored. Contralateral bursts seem to begin approximately 12ms before saccades, mainly affect the beginning of the saccade and have an excitatory effect. Their amplitude and duration depends on the saccade amplitude. Ipsilateral burst onset varies with the amplitude of saccades, affect the end of saccades, have an inhibitory effect and their amplitude and duration also depend on the

saccade amplitudes. The circuitry generating these bursts is not explicitly modelled, rather, bursts mimicking the observed ones are fed into the SBG model as additional inputs. The actual target for the FOR neurons is not clearly established but probably lies in the SBG, Dean chose to affect the input of the EBN, where the comparison between SC order and feedback is made.

The resulting model corrects the systematic errors induced by the delay in the feedback loop (hypermetria and main sequence alteration). Qualitatively, the command issued from the cerebellum is giving an additional boost in acceleration in the beginning of the saccade, followed by a stronger brake, allowing faster saccades without loss of accuracy. Finally, noting the variability of the FOR neurons activity and the loss of accuracy induced by cerebellar inactivation, Dean suggests that this activity could also reflect the on the fly correction of saccade trajectory induced by the variability of the brainstem saccadic circuitry, but without specifying the mechanism.

4.3   Schweighofer et al. (1996a,b)

The cerebellar module added by Schweighofer *et al.* to the Dominey and Arbib model (1992) also deals with the long-term adaptation of saccadic gain. It proposes a neural network model comprising granule, Purkinje and nuclear cells of the cerebellum and also of inferior olive neurons. It therefore allows comparison with electrophysiological results.

The granule cells are the input of the cerebellar network, they receive visual input, proprioceptive input (providing eye position) and feedback from the EBNs. The visual input is supposed to come from the cortex rather than from the SC *via* the nucleus reticularis tegmenti pontis (NRTP), because experimental results show that saccades generated by SC stimulation can not be subject to adaptation. The granule cells recode their inputs and project to two Purkinje cells (right and left) with a set of weights, which are randomly initialized and subject to adaptation. Each Purkinje cell inhibits one FOR neuron. The FOR neurons receive excitation from the EBNs, this activity is shaped by the inhibition of the Purkinje cells and then transmitted back to EBNs and IBNs. However, modifying the EBN activity does not change the metrics of the saccade, as they are involved in a control feedback loop. Thus, to allow cerebellar adaptation of saccade metrics, Schweighofer *et al.* hypothesise the EBNs are divided in two populations, a first one embedded in the SBG feedback loop and projecting to the FOR, and a second one, outside the loop, under control of the FOR and projecting to the motoneurons.

The adaptation of the granule-Purkinje synapses is controlled by an error sig-

nal generated in the inferior olive (IO) using proprioceptive rather than visual error estimation. The model IO neuron may fire a spike when it simultaneously receives memory input, signalling the occurence of a saccade in the past, and the signal that the target was not foveated, provided by frontal eye fields fixation neurons. In that case, the probability that a spike is emitted increases with the strength of the proprioceptive input signalling the occurence of a corrective saccade, *i.e.* with the amplitude of this corrective saccade. As the IO error input at the level of the granule-Purkinje synapses is delayed until the occurence of the corrective saccade, the synapse has an time window of eligibility during which IO input induce long term depression. In order to avoid all weights to tend to zero, a biologically unspecified weight normalization mechanism is also added.

The mossy fiber inputs and the FOR neurons activity of the model are similar to electrophysiological results. Specifically, the occasional pauses in FOR activity are reproduced, they are caused by too large Purkinje cells inhibitions and tend to disappear with learning. However, the simplification in the Purkinje cells modelling (only two such cells modelled) does not allow comparison with experimental results. The model is able to learn the nonlinearities of the saccadic burst generator and eye plant system, despite the coarse (binary) error signal. In target displacement adaptation, the finding that re-learning is faster is reproduced by the model, and is caused by the weight normalization mechanism. A major drawback of this model is the lack of input from the SC *via* NRTP, as this pathway is generally regarded as essential to the relations of cerebellum with the saccadic circuitry.

This study adds to the previous proposals of Dean a quantitative modelling of the cerebellar neural circuitry involved in the shaping of FOR neurons activity. This makes comparisons with electrophysiological results possible, and thus helps evaluate its verisimilitude.

4.4 Lefèvre et al. (1998); Quaia et al. (1999); Optican and Quaia (2002)

SC models do not explain why lesions of the SC do not generate permanent accuracy deficits, while it is well established that cerebellum lesions result in such deficits (increased variability of both amplitude and direction of saccades). Quaia, Lefèvre and Optican therefore investigated the hypothesis that the cerebellar contribution to saccades is adapted on the fly to compensate for the variability of the rest of the saccadic system.

They propose that two parallel circuits control the saccade:

(1) The SC provides a "go" signal through the deactivation of the OPN by the fixation cells and a "directional drive" depending on the location of

a hill of activity on the SC map.

(2) The cerebellum provides some additional drive, controls the progress of the saccade (via a spatial integration of eye velocity) allowing on the fly adujstements of trajectory, and finally stops the saccade using contralateral IBN activation.

The main hypothesis of the model, derived from this proposal, is that the cerebellum has a topographical organization similar to FEF or SC maps. The spatial integration is implemented by projections from the FEF and the SC (*via* the NRTP) specifying the position of the target and velocity feedback projections from the MLBNs (Fig. 14), interacting in a manner reminiscent of the SC *moving hill* models. As a consequence of this cerebellar spatial integration, the SBG part of the model does not include any resettable or leaky integrator.
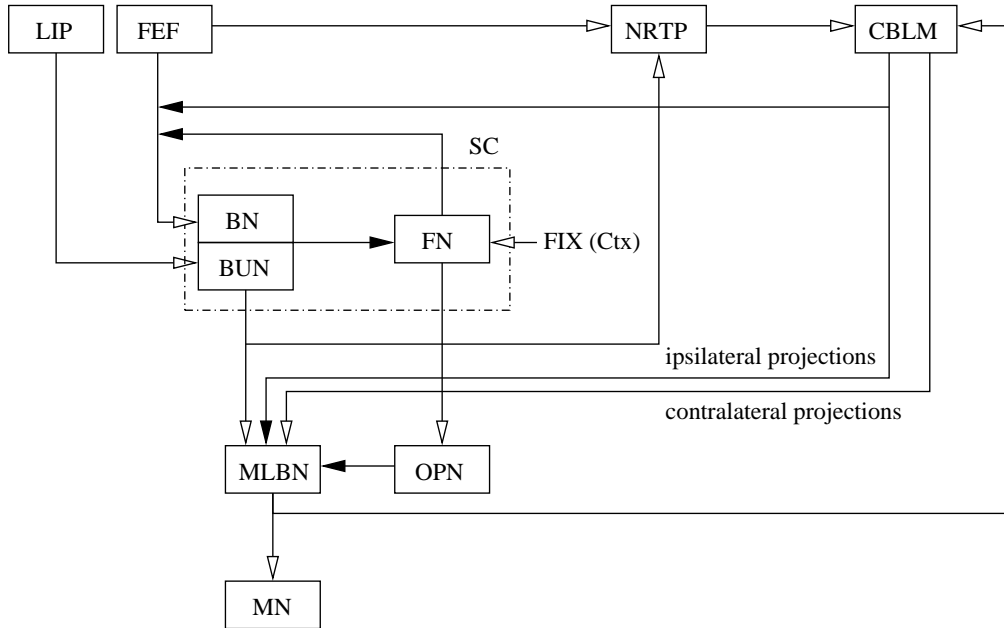

Fig. 14. Lefèvre *et al.* (1998) model. BN: burst neurons; BUN: build-up neurons; CBLM: Cerebellum; FEF: frontal eye fields; FIX: fixation signal from the FEF; FN: fixation neurons of the SC; LIP: lateral intraparietal cortex; MLBN: medium-lead burst neurons; NRTP: nucleus reticularis tegmenti pontis; OPN: omni-pause neurons; SC: superior colliculus. Filled arrows: inhibitions; empty arrows: excitations. Adapted from (Lefèvre *et al.,* 1998).

Another interesting stipulation of the model is that the BN and BUN neurons do not constitute two separate populations, but instead represent the two extremes of a continuum. The burst part of their activity is supposed to be produced by the FEF and specifies the target position, while the build-up

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comes from the lateral intraparietal cortex (LIP) and could represent an anticipatory remapping of the position of the target to be foveated. SC neurons receive weighted connections from both areas, BN putting more weight on FEF input and BUN on LIP input. They are, as a consequence, functionally identical, at least as far as saccade control is concerned. The potential role of BUN in target selection, calibration of maps in other modalities (as in Grossberg et al. (1997)), or determination of reaction time is evoked. The resulting SC model does neither belong to the *decreasing hill of activity* family nor to the *moving hill of activity* family as both of them were judged inadequate, its role is only to designate the goal of the ongoing saccade.

The model is not only capable of correcting saccade trajectories but also reproduces some lesion effects: FOR lesions induce longer and slower saccades while SC lesions induce curvature and speed changes. However experimentally observed latency increase is not obtained.

The topological organization of the FOR which is the basis of cerebellar spatial integration in this model hasn't been much studied and quite unclear given available data (Ron and Robinson, 1973), an alternative is therefore to be proposed.

Some aspects of this model are similar to the Dean (1995) model (additional initial acceleration and final deceleration), however, the main difference between the two is that here the integration is completely removed from the SBG and assumed by the cerebellum only. As a conclusion, this model does not only study the role of the cerebellum in saccade generation, but proposes a completely new view of the respective roles of the SBG, the SC and cerebellum.

4.5   Gancarz and Grossberg (1999)

Learning in the saccadic system comprises at least two types of learning: *gain learning* which allows to calibrate the saccadic burst generator using visual feedback at the end of saccades; and *map learning* which allows the accurate transformations from one coordinate system to another. Gancarz and Grossberg proposed a model including both learning capabilities, the first one depending on cerebellum operation and the second one on cortical interactions.

The cerebellum part of the model is described here while the cortex part is described in 6.3.

Saccades can be elicited by various pathways: reactive saccades deriving from retinal projections on the SC; auditory or visual attentive saccades, resulting from cortical processing of sensory inputs in parietal cortex; memory saccades

or sequences of saccades stored in the prefrontal cortex, etc. Experiments testing adaptation in various tasks by slightly displacing the target position after the beginning of the saccade, showed that:

(1) Adaptation is specific to a movement field (adaptation to the perturbation of a saccade of specific amplitude and direction does not affect saccades with different metrics).
(2) Adaptation does not necessarily transfer from one kind of saccade to the other (for example adapting the metrics of saccades in a memory task will not result in modified metrics in visual saccades).
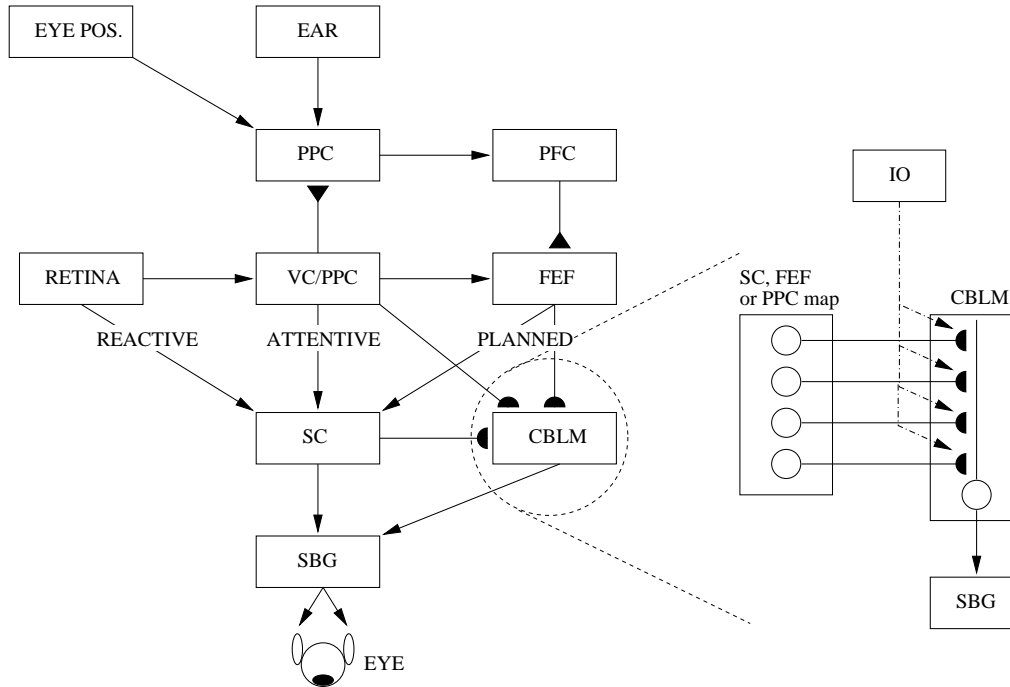

Fig. 15. Gancarz and Grossberg (1999) model. CBLM: cerebellum; FEF: frontal eye fields; IO: inferior olive; PFC: prefrontal cortex; PPC: posterior parietal cortex; SC: superior colliculus; SBG: saccadic burst generator; VC: visual cortex. Half-circle connections: gain learning site; triangular connections: map learning site; dash-dot arrows: learning signal. Adapted from (Gancarz and Grossberg, 1999)

The model is built around the Grossberg et al. (1997) SC model, it includes the three aforementionned saccade generating pahways. To each of them corresponds a specific retinotopic map which projects to the cerebellum (Fig. 15). The weights of individual elements of the map, having their specific movement fields, can be modified depending on an error signal issued from the inferior olive. This signal is generated by visual comparison of the distance between fovea center and target at the end of the saccade. The contribution of the cerebellum to the SBG activity is therefore adapted in a specific manner for each pathway separately, and even more, for each target location in each pathway. The results obtained concerning transfer of learning are in accordance with

experimental observations.

This model of cerebellar role in saccade adaptation is less detailed than Schweighofer et al. (1996a,b) model concerning neural modelling. It however has the specificity of studying the interactions of adaptation in saccade tasks involving different modalities.

4.6   Ebadzadeh and Darlot (2003)

Ebadzadeh and Darlot recently proposed another cerebellar model of long-term adaptation. They use an original approach to design their model. They first physically and mathematically formalize the command circuit necessary to achieve the task. Then, they compare this circuit with known physiology and anatomy, looking for its potential neural substrates. Finally, they simulate the resulting model, check its validity with regards to task achievement and biological significance.
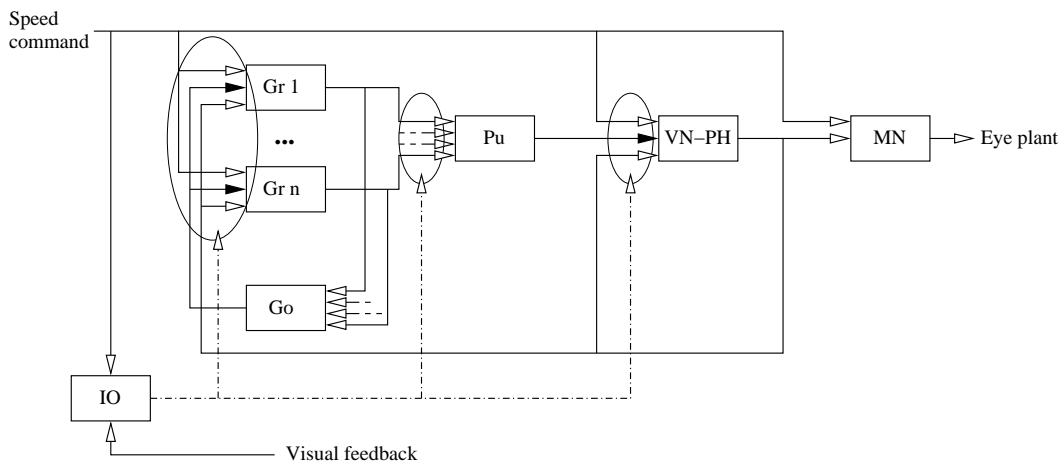

Fig. 16. Ebadzadeh and Darlot (2003) model. Go: Golgi cell; Gr : Granular cells; IO: inferior olive; MN: motoneuron; Pu: Purkinje cell; VN-PH: vestibular and prepositus hypoglossi nuclei. Empty arrows: excitatory connections; full arrows: inhibitory connections; dash-dot arrows: learning signal; ellipses: synapses subject to supervised learning.

The resulting model (Fig. 16) provides the cerebellum with a desired speed command issued from the superior colliculus and generates the phasic and tonic signals which are fed into motoneurons. Granular, Golgi and Purkinje cells are assembled in a multiple-loop circuit which is supposed to provide a direct model of the eye plant used to evaluate the current eye position. The calibration of this direct model is obtained by supervised learning, whose error signal is obtained by comparing the final and desired positions. It is computed in the inferior olive and it is provided to three learning sites. The formal analysis of the circuit allows the determination of the learning rule, which

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fortuitously reproduce the long term depression observed in the Purkinje cells, and predicts that learning should also occur at the level of mossy-fiber-granular cell synapses.

The model was trained to reproduce a finite set of saccades fitting the main sequence and tests revealed it was then able to produce accurate saccades of arbitrary amplitude.

This model has the particularity that its connectivity was deduced from functional considerations, in a top-down manner, and is however compatible with known anatomy and physiology.

4.7 Discussion

The litterature dealing with the modelling of cerebellum supervised learning capabilities is quite abundant (refer to Houk et al. (1996) for a review). Even though it is beyond the scope of this review (models of saccade-related circuits), it provides numerous insights concerning long-term adaptation processes in the cerebellum. Consequently, most of the reviewed models concern long-term adaptation and derive from previous cerebellum models dealing with limb movements.

It is now clear that these long-term adaptation model have to explain the effect of the cerebellum on the SBGs in terms of additional contralateral or ipsilateral bursts (Dean, 1995; Schweighofer et al., 1996a; Gancarz and Grossberg, 1999; Ebadzadeh and Darlot, 2003), rather than in terms of direct gain modification (Dean et al., 1994). Future models should also explore beyond the simplistic perceptron-like Purkinje cell scheme and integrate the latest results concerning the various cerebellar plasticity sites.

Differences in the learning rate are observed in some specific experimental setting:

- adaptation is faster when re-learning to produce correct saccades after an artificial adaptation than during this initial artificial adaptation,
- adaptation is faster when adapting to correct overshoot saccades than undershoot.

Moreover, adaptation is partially dependent on the modality of the saccades (visual, auditory, memorized), as adaptation for one type of saccades is not necessarily inherited by another type of saccades. These specific properties of the cerebellum, partially explained by the reviewed models, should be further explored in future models.

Compared to the numerous long-term adaptation cerebellum models, only the models from Lefèvre, Quaia and Optican studied the role of the cerebellum in instantaneous correction of trajectory. These two potential roles of the cerebellum are, in any event, not necessarily exclusive. For instance, the Optican team's models, which corrects saccades on the fly, have hand-tuned projections to the SBG which could be subject to calibration by supervised learning. The question of whether the cerebellum assumes all trajectory integration computations or shares them with a SBG integrator remains to be decided.

5 Basal Ganglia

The basal ganglia (BG) are a set of interconnected subcortical nuclei involved in large cortico-basal ganglia-thalamo-cortical loops (Fig. 17). Five different parallell loops exist in primates: motor, oculomotor, prefrontal (dorsolateral prefrontal and lateral orbitofrontal) and limbic loops. They have similar internal connectivity but interact with different cortical areas and brainstem nuclei. The oculomotor loop is of primary interest concerning saccade generation, as it interacts with the frontal eye fields (FEF) and the parietal posterior cortex (PPC) and projects to the SC, gating the loci of activition on the collicular map. The prefrontal loop involving the dorsolateral prefrontal cortex (DLPFC) is incidentally involved in saccade generation as it is involved in working memory processes enabling the learning and restitution of sequences of saccades.
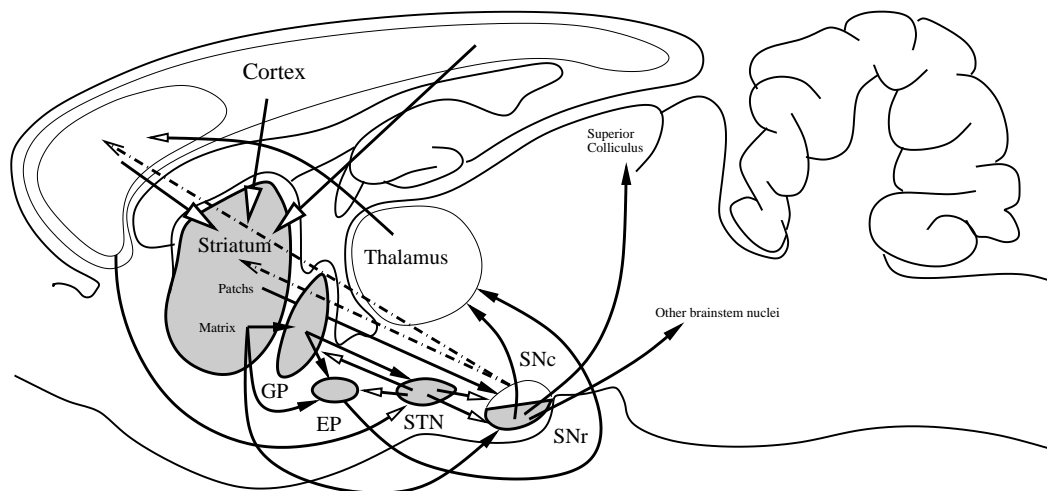

Fig. 17. Overview of basal ganglia nuclei and connectivity in a rat brain. EP: entopeduncular nucleus; GP: globus pallidus; SNc: substantia nigra pars compacta; SNr: substantia nigra pars reticulata; STN: subthalamic nucleus. Striatum's patch and matrix: see text. Empty arrows: excitatory projections; full arrows: inhibitory projections; dash-dot arrows: dopaminergic projections.

The input nuclei of the basal ganglia circuit are the striatum (including the

putamen, caudate nucleus and nucleus accumbens) and the subthalamic nucleus (STN). The striatum can be divided in cytoarchitectonically and neurochemically distinct subpopulations of neurons, a set of isolated areas called patches, surrounded by the so-called matrix. The matrix of the striatum has inhibitory projections to the globus pallidus (GP, or external part of globus pallidus, GPe, in primates), entopeduncular nucleus (EP, or internal part of globus pallidus, GPi, in primates) and substantia nigra pars reticulata (SNr). EP and SNr contitute the output (inhibitory) of the BG, while the GP is part of an internal circuit involving the STN: the GP inhibits the EP and the STN, while the STN excites the GP, the EP and the SNr.

The patches the striatum project to the dopaminergic system (substantia nigra pars compacta, SNc, and ventral tegmental area, VTA) which projects back to the whole striatum, to the STN and to the frontal cortex. This circuitry seems to be central in the neural implementation of reinforcement learning capabilities.

The basal ganglia circuitry of a given loop is constituted of parallel channels: each nucleus can be subdivided in distinct groups of neurons, each group of a given nucleus projecting to a specific group in the target nucleus. The STN is an exception as its excitation seems to be diffuse.

The role of the BG is interpreted as a generic role of selection: it selects which channel inside a loop has to be activated at a given moment. This selection occurs via disinhibition: at rest, the output nuclei of the BG are tonically active and keep their thalamic and brainstem targets under constant inhibition, when multiple channels are activated, the BG operate a winner-takes-all computation and, as a result, the output inhibition corresponding to the winning channel is interrupted, allowing the activation of the targeted circuit.

The complex internal circuitry of the basal ganglia has long been interpreted as constituted of so-called *direct* and *indirect* pathways (Fig. 18, left). The direct is inhibitory to the EP/SNr, the indirect is excitatory (as a result of a cascade of two inhibitions and one excitation), and the balance between the two opposite effects is a key feature of basal ganglia operation. This scheme is however outdated as new experimental data showed that it neglects important connections, it has now been replaced by a direct, indirect and hyperdirect pathways interpretation (Fig. 18, right).

The two models of saccade generation including the basal ganglia explored their role in selecting the saccade target on the SC and in storing in working memory the location of potential targets.
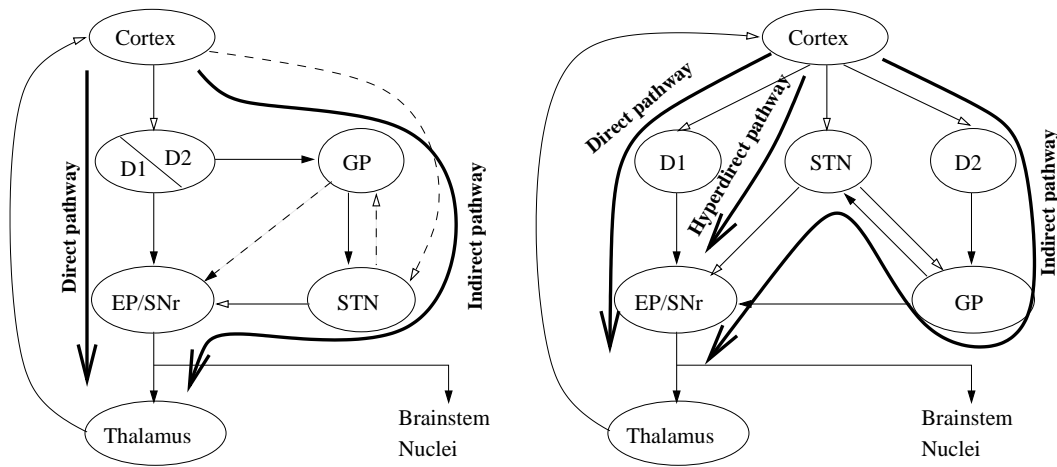
Fig. 18. Interpretations of the BG connectivity. Left: direct/indirect pathways; right: direct, indirect and hyperdirect pathways. D1: striatal neurons with D1-type dopamine receptors; D2: striatal neurons with D2-type dopamine receptors; EP: entopeduncular nucleus; GP: globus pallidus; SNr: substantia nigra pars reticulata; STN: subthalamic nucleus. Empty arrows: excitatory projections; full arrows: inhibitory projections; dashed arrows: neglected connections in the direct/indirect pathways interpetation; bold arrows: pathways.

5.1 Dominey and Arbib (1992); Dominey et al. (1995)

The BG subpart of the Dominey and Arbib model is based on the old direct/indirect pathways interpretation and moreover only includes the direct pathway. Two parallel subloops are distinguished in the BG: a first one projecting to the SC, enabling the selection of the target, and a second one projecting back to the FEF via the thalamus, in charge of storing potential targets in working memory.

The striatum and the SNr only are modelled (Fig. 9). The channels of both circuits are elements of a retinotopic map which allows the specification of the saccade metrics. The striatum of the selection circuit receives inputs from both visual and memory layers of the FEF, the learned weighting of these inputs induces the selective inhibition of the SNr which in turn disinhibits specific areas of the SC, enabling the execution of the chosen saccade. The memory circuit receives inputs from the memory layer of the FEF only, and its SNr projects to the thalamus. The disinhibiton of the thalamus allows the onset of a reverberation between the the thalamus and the memory layer of the FEF, which is the base of the memory.

An extension of the model (Dominey et al., 1995) includes plasticity of the cortico-striatal synapses and dopamine reward signals. Additional cortico-striatal projections from the inferior temporal cortex (IT) and from the prefrontal cortex (PFC) provide additional information concerning the context

and the execution of sequences. The IT provides contextual information relative to the nature of cues (color, shape, etc.) that can be presented before the selection of the target and the execution of the saccade. The PFC provides an input vector with unique patterns of activity for each step during the execution of a given sequence. The model is then able to learn to choose among two similar potential targets depending on precueing and to reproduce sequences of saccades.

The whole Dominey and Arbib model was tested in single, memory and double saccades and some of its simulated cell activity patterns were qualitatively compared with electrophysiological recordings.

This model of the saccade generation circuitry is the first to include the basal ganglia, and thus to propose mechanisms generating the SNr responses measured by Hikosaka and Wurtz in 1983 (Hikosaka and Wurtz, 1983a,b,c,d). It is also the most complete to date in terms of included brain regions, and thus the only one to produce a global understanding of the operation of the whole system, proposing explanations of how individually studied areas interact together during the execution of saccades.

5.2   Brown et al. (2004)

As new experimental data concerning the BG have been accumulated in the last ten year, Brown et al. proposed a new model of the role of the BG in selecting both the relevant strategy for saccade generation and the metrics of the saccade, once the strategy is chosen. The cortical part of this model, and especially the description of the laminar structure of the FEF, is presented in section 6.4.

The oculomotor BG loop is, in this model also, divided in two parallel subloops (Fig. 19). The first one is an IT/FEF-BG-thalamus-FEF loop whose role is to select which strategy will drive the forthcoming saccade decision. These strategies can be based on (1) the state and position of the fixation point, (2) the state and position of the target or (3) the nature of some object in the visual field, independently from its position (cue). This circuit therefore has three channels associated to these three strategies. The second subloop is a PPC/FEF-BG-SC-FEF loop, it has to select the metrics of the saccade by disinhibiting a location on the SC map. The channels of this circuit correspond to the movement fields of a retinotopic map. The decision is taken using a salience map provided by the FEF and depending on the strategy decision taken by the first circuit.

The internal circuitry of the BG is modelled using recent knowledge on nuclei interconnections:
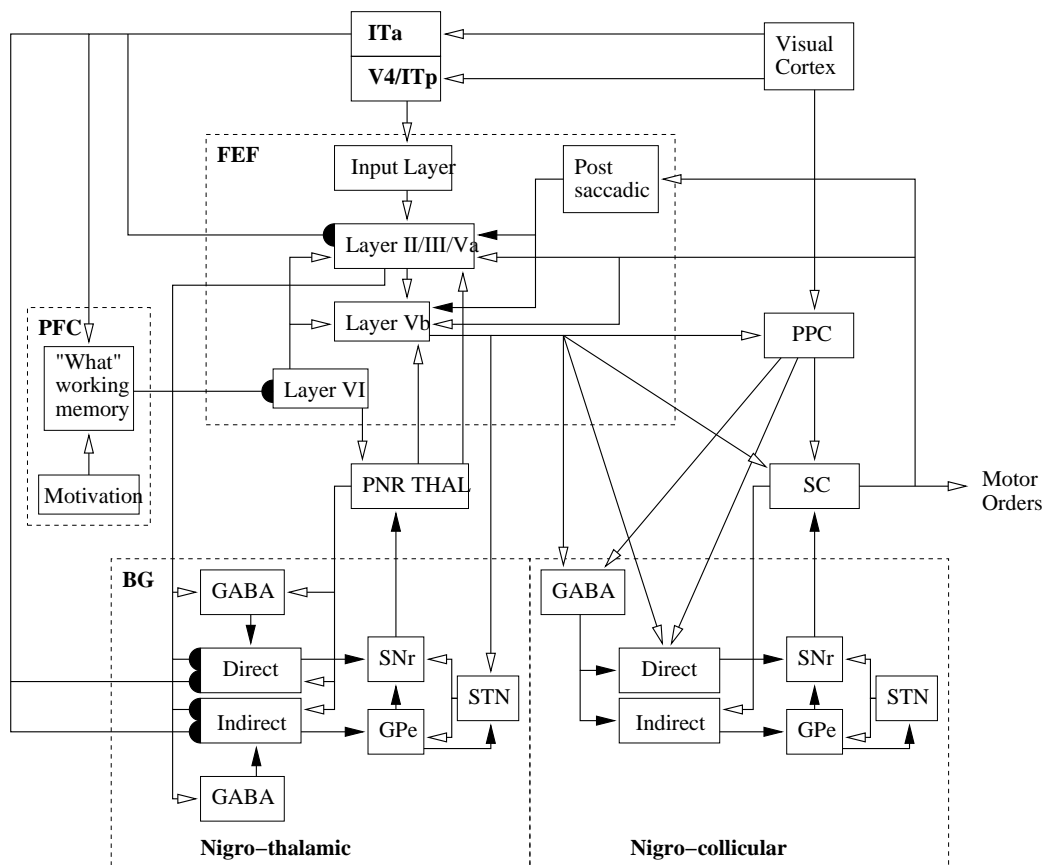
Fig. 19. Brown *et al.* (2004 model). BG: basal ganglia; FEF: frontal eye fields; GABA: GABAergic inhibitory striatal interneurons; GPe: external segment of the globus pallidus; IT(a/p): anterior/posterior inferotemporal cortex; PFC: prefrontal cortex; PPC: posterior parietal cortex; SC: superior colliculus; SNr: substantia nigra pars reticulata; STN: subthalamic nucleus; V4: extrastriate visual cortex area 4. Empty arrowheads: excitatory projections; full arrowheads: inhibitory projections; filled semi-circles: plastic synapses. Adapted from (Brown *et al.*, 2004).

- The *direct* pathway is the place where selection occurs, thanks to an on-center off-surround architecture. Each channel in the direct part of the striatum receives specific excitatory input related to its role only in conjunction with diffuse inhibitory input common to all channels and generated by a combination of all excitatory inputs in the striatal GABA interneurons. For example, in the first BG subloop, the channel corresponding to the strategy based on target state and position is excited by subpart of the FEF corresponding to this strategy, while it is inhibited by a signal generated by the whole FEF.
- The *indirect* pathway is used to generate a trainable stop signal used in tasks involving a delay before the response. It operates by increasing the STN activity: this provides additional activation of the SNr/GPi thus increasing their inhibitory output and blocking the selection process.
- The *hyperdirect* pathway is only present in the first BG subloop, it is under

control of the FEF output layer and is used by the winning strategy to lock the BG in order to prevent the selection of another strategy before the execution of orders. The selection of a strategy by the direct patway involves increased activity in FEF output layer, transmitted to the STN which blocks the BG subloop selection process in a similar manner as the indirect pathway.

The cortico-striatal synapses of the first subloop are trainable using dopamine input signaling the occurence of a reward or a punishment. A burst of dopamine above baseline level (reward) generates a weight increase of the recently activated channels in the direct pathway, favoring the last decision taken in future similar contexts. Conversely, dips in the dopamine baseline level (punishment) generate weight increases in the indirect pathway, which will tend to suspend the last decision taken in future similar contexts.

The model was tested in five standard saccade tasks (fixation, single saccade, overlap, gap and memory saccade) and was able to learn to generate adequate saccades in each situation. The activity profile of some model cells was shown to be qualitatively similar to their experimentally observed counterparts.

Various aspects of the BG part of the model are subject to discussion:

- Considering that the indirect pathway is dedicated to delayed saccade responses seems to be a bit too much task-specific and not necessarily useful in real-life situations. The hypothesis that such a complex cascade of inhibitions and excitation evolved for this sole purpose is debatable.
- In the first BG subloop, two GABAergic striatal interneurons families are distinguished: some are projecting specifically to the neurons belonging to the direct part of the striatum and have an additional thalamic input, as the others are dedicated to the indirect pathway. Knowing that these interneurons are not numerous in the striatum and seem to have broad and extensive projections over the whole striatum, such a discriminative tragetting is unprobable.
- The plasticity of the cortico-striatal synapses in the first BG subloop depends on secondary messenger traces. These are however computed in a different manner for direct and indirect pathways (in the indirect pathway, the trace depends on the thalamic input) even if their inputs are quite similar. No justification for this difference is proposed.
- The plasticity of the cortico-striatal synapses of the second BG subloop is not assigned any role.
- Concerning the connectivity of the second BG subloop, the selectivity of the SC feedback over the indirect pathway is not justified, nor the lack of any cortical modulation of the STN activity.
- Finally, the experimentally observed memory-related SNr cells (Hikosaka and Wurtz, 1983c), which can be necessary for efficient target remapping in

the SC (Bozis and Moschovakis, 1998), are not included in the SC-projecting BG subloop.

Despite these small flaws, this BG model is much more up-to-date than the Dominey and Arbib proposal, it is also the first BG model (even including BG models not related to saccade generation) taking into consideration the laminar structure of the cortex, and specifically the fact that striatal and STN inputs originate from different layers.

5.3 Discussion

The role of the oculomotor loop of the basal ganglia in the saccade-related brain circuitry has not been subject to many modelling studies. The BG clearly have a selective influence over the SC, which was often included in SC models by SNr external inhibitory input (Arai et al., 1994; Das et al., 1995; Grossberg et al., 1997; Bozis and Moschovakis, 1998; Arai et al., 1999). As mentioned in section 3.17, the BG do not form loops with cortical areas only, but also with SC superficial and intermediate layers. The dynamics of these loops can only be studied by modelling the BG-SC interactions with up-to-date models of both regions.

As many models of the other basal ganglia loops performing selection have been proposed (see Gillies and Arbruthnott, 2000; Joel et al., 2002, for recent reviews), some of them could easily be adapted to encompass the oculomotor one (Gurney et al., 2001, for example).

Finally, the involvement of a prefrontal BG loop in working memory has been studied and modelled (Frank et al., 2000) and could conveniently be included in future models, providing them with additional memory and sequences learning capabilities.

6 Cortex

As briefly stated in introduction, many cortex areas are more or less implicated in the saccadic premotor activity. The posterior parietal cortex (PPC), and more specifically lateral intraparietal area (LIP), modulates the "Where" stream of the cortical visual processing by attentional processes. The dorso-lateral prefrontal cortex (DLPFC) stores target positions, acting as a spatial working-memory, which allows temporal organization of saccades (predictions, delays) and even saccade inhibition. The pre-supplementary eye fields (pre SEF) is involved in learning motor programs containing saccades and projects

to the supplementary eye fields (SEF) which can execute them if the context is appropriate by sending saccade orders to the frontal eye fields (FEF). The FEF receives convergent inputs from PPC, SEF and DLPFC, operates the final target selection stage by interacting with the basal ganglia and sends the corresponding motor command to the SC and SBG. Finally, FEF, SEF and DLPFC receive projections from the anterior cingulate cortex which provides them with motivational modulation.

Concerning saccadic system modelling studies, the PPC and the FEF have been the most frequently and extensively modelled cortical areas because of their positions in the flow information: visual "Where" stream input to ocular premotor circuitry and latest ocular premotor area before subcortical stages, respectively.

6.1 Zipser and Andersen (1988)

Zipser and Andersen (1988) studied the transformation of visual input coded in oculocentric coordinates into craniocentric coordinates using eye position. This transformation is thought to occur in the area 7a of the PPC, which receives both eye-position and retinal signals and where neurons exhibit interesting patterns of activity, corresponding to retinocentric receptive fields modulated by so-called gain fields coding eye position in the head. Their model is a perceptron trained to solve the coordinate transformation problem and happens to simulate the area 7a gain-field activity.

The input of the perceptron is a 8×8 array, composed of visual neurons with 15°-wide receptive field, and two 16 neuron vectors coding the azimuth and elevation coordinates of the eye in the head. The hidden layer is composed of 9 to 36 units. The craniocentric output is either a 8×8 map or two 16 neuron vectors coding the coordinates of the target. This perceptron is trained to compute the coordinate transformation using the classical back-propagation algorithm.

After 1000 trials, the networks is satisfactorily trained. The activity of the hidden layer neurons reflects gain fields superimposed on large receptive fields and is qualitatively comparable to 7a neurons. These fields are however not as complex as some of those of real neurons, especially when the output is coded by a vector rather than by a map. These complex receptive fields are more comparable to the hidden layer activity before training.

The main interest of this paper is to show that the gain-field encoding used in area 7a can efficiently perform a coordinate transformation.

In their 1992 model, Dominey and Arbib simulate the activity of neurons in the FEF and PPC, while the 1995 extension adds simulated extrastriate visual cortex area 4 (V4), inferior temporal cortex (IT) and prefrontal cortex (PFC). On the one hand, FEF and PPC are made of multiple layers, each of them organized as retinotopic maps where potential targets are represented by hills of activity (Fig. 9). On the second hand, V4, IT and PFC are modelled by groups of neurons that do not have a topological organization.

The PPC is made of two layers. The first one (visual) receives visual input from the "Where" pathway of the visual cortex, signalling the position but not the nature of potential targets present in the visual field. The second one, a quasi-visual layer (QV), receives direct projections from the visual layer and is able to memorize targets positions thanks to reverberating excitations. Beyond this memory capability, this second layer also receives feedback from the brainstem tonic neurons measuring the eye position and is capable of remapping the positions of targets during eye movements, in a *moving hill* of activity manner, similar to many SC models proposals. This layer drives the activity of the visual and quasi-visual layers in FEF and SC respectively.

The FEF is made of four layers. The first (visual) layer only relays the activity of the QV PPC layer to the memory and presaccadic layers of the FEF. The second (memory) layer of the FEF receives the memorized targets positions from the PPC but these are stored only if the resonant activity with the thalamus is made possible by the disinhibition of the latter by the memory basal ganglia loop (see section 5.1). The PPC memory stores all perceived targets positions, whereas the FEF memory selects those it stores depending on reinforcement learning occuring at the level of cortico-striatal synapses. The third layer is a fixation layer, it is active if the foveal part of the PPC visual layer is activated, in which case it inhibits the memory and presaccadic layers of FEF along with the superficial and winner-takes-all layers of the superior colliculus. Finally, the presaccadic layer gathers the activations of the visual and memory layers of the FEF along with the fixation inhibition signalling the presence of a target in the fovea and projects the resulting landscape of activity to the BG selection circuit and to the SC saccadic layer to enable saccade generation.

The role of the PPC is to relay present and past visual inputs, and to produce the remapping function necessary to keep this information up-to-date after saccades. The FEF interacts with the BG to select 1) the targets to be stored in working-memory and 2) the targets to foveate among suggestions issued from both vision and memory.

V4 is modelled as a group of neurons coding the features (shape, color) of the object lying on the fovea, these project to 25 IT neurons with random connections, therefore encoding feature conjunctions and disjunctions. The projections from IT to BG allow the saccade selection to learn to take into account the nature of cues, as evoked in section 5.1.

The PFC is a group of 25 neurons, it receives randomly weighted inputs from the PPC, providing target positions, and from the SC, providing saccade execution signals. It also receives damped self inputs with five different time constants which provide short-term memory. The resulting activity encodes past sequences of targets and saccades, unique patterns being generated for each step of the execution of a sequence. The projections from the PFC to the BG are subject to reinforcement learning, which enables the learning of sequences by associating specific PFC patterns with the corresponding saccades.

The resulting architecture accounts for the visual, quasi-visual and memory activity patterns observed in FEF and PPC. As previously mentionned, it succeeded in tasks of simple, memory, double and precued saccades, and also in sequence learning tasks. This proves the efficiency of the memory, remapping and learning properties of the simulated cortical areas.

This model was the first one propose an interpretation of the cortical patterns of activity observed in saccade experiments. Concerning the remapping of targets ability, the choice is to have it performed in the PPC QV layer, this layer then controlling its equivalents in the FEF and the SC. This could also be performed in a parallel and redundent manner, as suggested in the Bozis and Moschovakis (1998) model, because in a phylogenetic point of view, this function might have originated in the SC of animals lacking a real cortical structure and kept since then.

6.3   Gancarz and Grossberg (1999)

The cerebellum part of the Gancarz and Grossberg (1999) model is described in section 6.3. Its cortical part deals with the problem of coordinate transformations in the cortex, what they call *map learning*. It is supposed to occur at the level of parietal cortex, where information from visual (retinocentric) and auditory (craniocentric) sources are mixed, and between the PFC and the FEF, as the planning of saccade sequences in PFC is thought to be in craniocentric coordinates.

The model generates saccades in one direction only and considers one visible target at a time. The coding of positions and coordinate changes where therefore highly simplified. The retina is a vector of zeros with a single one

at the target position. As only one visual target has to be coded in the PPC and FEF, a single value representing its abscissa on the saccade axis is coded. Transformations from craniotopic to retinotopic coordinates simply imply the substraction of the eye position signal provided by the tonic neurons of the brainstem saccade generator. An *ad hoc* transformation involving fixed weights and threshold allows the transformation of this value into an activation on a 1D map in the PFC, allowing the storage of multiple targets. Finally, the reverse transformation used to compute a single value (in PPC and FEF) from a 1D map (retina or PFC) involves the calibration of weights, simply using the eye position at the end of the saccade as a teaching signal.

The disturbance of these weights by the perturbation protocol previously evoked (see section 4.5) is the base of the recalibration of coordinates changes. The specific order in which these coordinates changes occur explains why adaptation transfers from scanning task to memory task but not the other way.

This model is the only one exploring the issue of unidirectional adaptation transfers, however its implementation is too coarse to allow comparison of its internal cortical state and recorded cells.

6.4   Brown et al. (2004)

The Brown *et al.* model included various cortical areas (PFC, FEF and PPC) but a special interest was given to the FEF, as its internal laminar structure was modelled and simulated.

The model distinguishes 4 layers and a pool of post saccadic cells in the FEF (Fig. 19). These layers are a sensory input layer, a plan layer (representing layers II, III and VIa), an output layer (layer Vb) and an arousal layer (layer VI). Input, plan and output layers are made of 3 distinct retinotopic maps, representing the three saccade strategies implemented in the model: strategy guided by the position of target, or the position of the fixation object or guided by the features of the foveated object (Fig. 20). The resulting distinct translayer structures associated to the strategies are called gateable cortical zones (GCZ). The plan layer of the FEF, at the GCZ level, interacts with the first basal ganglia circuit (see section 5.2) in order to select the saccade strategy, the retinotopic maps of the output layer then interact with the second basal ganglia circuit in order to select the precise metrics of the saccade.

The input layer receives information about the location of salient stimuli in the visual field, the nature of the stimulus dictating which GCZ is concerned. Additional feedforward inhibitions act to enhance the constrast.
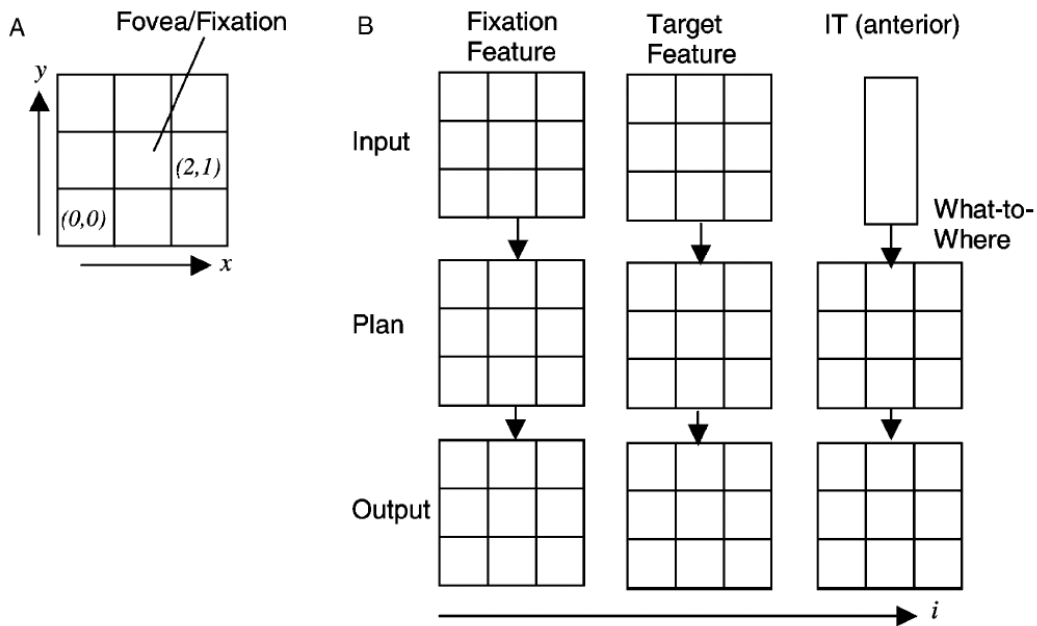
Fig. 20. FEF structure of the Brown *et al.* (2004) model. A: Retinotopic maps at the level of the cortex are 2D-grids with the central unit representing the fovea; B: Rows correspond to different layers, columns correspond to different strategies forming isolated Gateable Cortical Zones (GCZ) across the layers. The IT (inferotemporal cortex) GCZ represents the startegy consisting in the association of a specific cue (the nature of an object provided by IT) with a given saccade, whatever might its spatial position be, the corresponding input layer is therefore not a spatial map. From (Brown *et al.*, 2004).

The arousal layer is driven by inputs from the PFC, which stores the nature of the presented objects, and can provide an additional drive to one of the GCZs if the cue was associated by learning to a specific strategy.

The plan layer receives the sorted and contrast-enhanced visual input of the input layer, local selection of saccade metrics is made in each GCZ by lateral inhibitions and autoexcitation, GCZ selection is made by reciprocal inhibitions of neurons belonging to the different GCZ but associated to the same metrics. The GCZ selection is reinforced by positive feedback provided by the basal ganglia-thalamic circuit and by the possible object-associated drive from the arousal layer. SC feedback reinforces the activity of the units corresponding to the selected metrics during the execution of the saccade, while post-saccadic inhibition resets the activity after saccade completion.

The output layer is very similar to the plan layer, the differences being the absence of between-GCZ inhibitions and the projections to the SC providing the saccade "go" signal.

The PPC is a single-layer retinotopic map. It has a phasic activity because of self-inhibitions, is activated by the FEF output layer and by biased visual input

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(peripheral targets and left-right rather than up-down saccades are favoured), and projects to the SC. Its functional role in the model is to elicit reactive visual saccades, no attentional process was modelled.

The model was tested in a number of tasks listed in section 5.2.

The choice of building two GCZs distinguishing the fixation and the target stimuli seems a bit *ad hoc* and surprising as in monkey experiments, these are often objects of the same nature. This does not however discard the interesting idea of hierarchical selection (first strategy and then metrics) which is present in some basal ganglia models (Monchi et al., 2000). This recent saccade-circuitry model is however the first one to go into such detailled modelling of the cortical laminar structure.

6.5   Deneve et al. (1999, 2001)

Deneve *et al.* address the generic problems of population coding with noisy inputs and of computations based on population-coded variables. Their first model (Deneve et al., 1999) shows how variables can be extracted from noisy sensory input and stored in recurrent networks. Building on this, the second one (Deneve et al., 2001) shows how such networks can be interconnected in order to perform function approximation (computation of a variable from inputs) and cue integration (all variables are known and interact to remove noise). This work is not limited to cortical areas involved in saccades, it is representative of the current state of the art (Pouget et al., 2003) in cortex population coding and computing. However, the example used in the second paper is of peculiar relevance, as it addresses the integration of variables linked with saccades generation and coordinates transformation: the retinotopic position of a target, the craniotopic position of the eye and the craniotopic position of the target.

The maps encoding a single variable are recurrent networks with excitatory lateral connections (Gaussian filters) and global divisive shunting inhibition. The cells of these maps have bell-shaped tuning curves with regard to sensory input with a superimposed noise. Function approximation and cue integration is based on a basis function architecture (Fig. 21). However, the purpose of this study is not to execute simple function approximation (computing one variable depending on the two inputs), but to allow any of the three variables to be computed from the two others along with cue integration. Consequently, the connections of the three sensory layers to the basis function layer are not feedforward but bidirectional. The reliability of an input is represented by its level of activity.

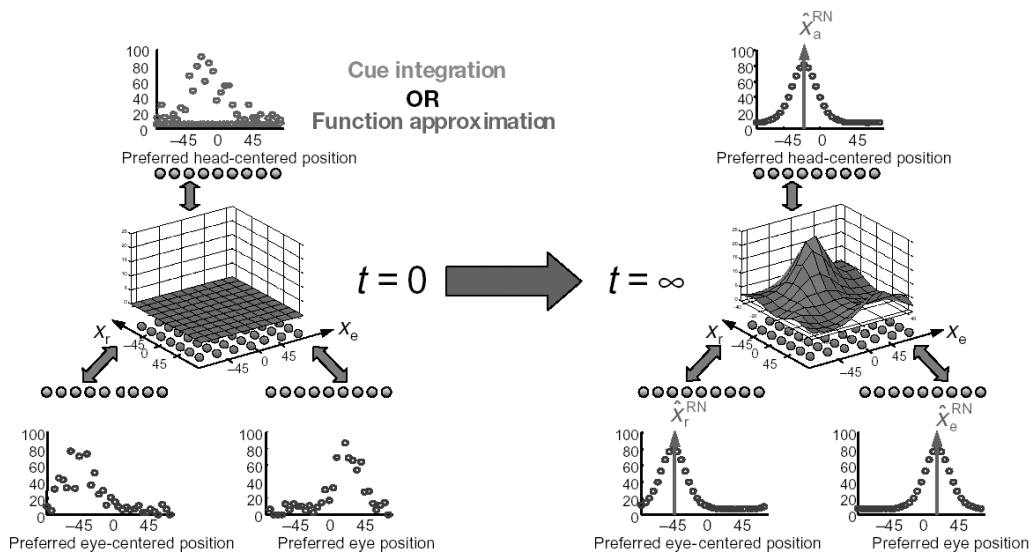The population coding efficiency is evaluated in the maximum likelihood es-

Fig. 21. Deneve *et al.* (2001) model. Left: intial state of the model, right: stable state stabilization. Inputs are one-dimensional, the intermediate layer is a two-dimensional map of basis function units, the four layers interconnections are bidirectional. In the case of function approximation, the head-centered position layer (upper-left) has no initial input, while in cue integration, this layer also has a noisy input. From (Deneve *et al.*, 2001).

timation framework (ML). It can recover the best possible measure of the encoded variable. The operations of function approximation and cue integration are also performed with an efficiency very close to the ML estimates, even in the case of deteriorated inputs (reduced reliability or increased standard deviation).

The activity of the basis function layer mimics the gain fields observed in many cortical areas. It is predicted that multimodal neurons should exhibit partially shifting receptive fields in all modalities, similar to those observed in SC and in ventral intra parietal and premotor cortices.

These models extend function approximation with population codes using basis functions, as it performs optimally with both function approximation and cue integration in the case of noisy input.

6.6 Mitchell and Zipser (2001)

The Mitchell and Zipser (2001) model deals with spatial short-term memory in parietal areas. It is a neural network obtained by optimization, rather than by design, in order to memorize the position of a target in eye- and head-centered coordinates and to update it in case of saccades.

The network is made of a set of recurrently connected neurons, its inputs are two simulated retinas together with eye conjugate and vergence angles and velocities, its output are two maps, respectively eye- and head-centered. The connections of the network are obtained by learning (back-propagation through time algorithm), the evaluation criterion being the ability to store the position of a flashed target and to correctly update its position during saccades.

The resulting network contains neurons that are comparable to parietal neurons from area 7a or LIP: their receptive field responses are modulated by gain fields which are planar functions of eye position. They can be divided in three classes, depending on the nature of their receptive fields: eye-based, head-based or intermediate. The head-based cells obtained in the model have not been found yet in area 7a or LIP, but could have been overlooked or could lie in another parietal area.

Instead of using a purely eye- or head-centered strategy, the network solves the problem with a mixed strategy which is reminiscent of the basis function layer of the Deneve *et al.* model. The possibility to extend this model to the storage of multiple targets should however be carefully examined. The optimization approach to modelling used here generates a solution that would not be easily designed and yet accurately models firing pattern observed in the parietal cortex.

6.7 Mitchell and Zipser (2003)

The 2003 proposal of Mitchell and Zipser models the frontal eye fields, which are supposedly involved in memory guided sequences of saccades. As in their 2001 paper, they make minimal assumptions about the structure of the network they build and use an optimization algorithm to tune the parameters of the connections.

The model is made of 4 interconnected retinotopic layers, the weights of the projections from one layer to the others and itself are based on difference of gaussian functions (mexican hat). Layers one to three receive the visual input and a fixed bias input, and layer one is constrained, during training, to reproduce the firing patterns of the visuo-motor FEF cells. Layer four doesn't have access to the visual input, receives a fixation input determining when saccades should be executed and is trained to mimick the firing patterns of the quasi-visual FEF cells. During training, a sequence of three targets is presented, then the fixation signal is removed three times and the fourth layer has to activate in the correct order the three cells corresponding to the target positions. In this training phase, the saccades are not really executed, as no

remapping of the targets is demanded. The resulting network uses its first three layers to respectively store the location of the first, the first and the second, and the three targets (select one, select two and select all layers). This information is then used in the fouth layer (burst output layer) to select them in the correct order.

The remapping functionality is added afterwards, by adding to the self-connections of the three memory layers an asymetric weight pattern (the derivative of the mexican hat function) gated by eye velocity. The resulting self-connections of a layer are consequently identical to those of the dynamic memory model (Droulez and Berthoz, 1991) -representative of the *moving hill* family of SC models- and the whole architecture is thus a 3-layer dynamic memory, where each layer is specialized to store a specific number of targets. The fixation mechanism is also changed: the external fixation signal is replaced with an internal signal generated by a fixation cell, which is in competition with the "select one" layer via reciprocal inhibitions.

The consequences of these changes are that the model is capable to solve the triple saccade task for which it was built, and also accurately reproduces the patterns of activity of real FEF fixation, burst, build-up and quasi-visual cells. The model is unexpectedly able to produce visual search by exploring simultaneously presented targets in decreasing order of salience, in a manner similar to humans. Inhibition of return, which prevents from coming back to a previously explored target, is a consequence of the fact that the model always makes plans concerning the three forthcoming saccades. It also exhibits saccade averaging, increased reaction time in presence of distractors and the ability to make accuracy vs. latency trade-offs.

The fact that this model, designed for execution of memorized sequential saccades, works also very well for visual search is quite an interesting result. It shows that the two functions can share the same substrate. Moreover, it proposes that the inhibition of return can be the consequence of planning rather than the results of an *ad hoc* feature. The limitation of the model to short-term memory sequences of three items, and the direct relationship between the number of layers and this limitation has to be further explored and compared to human behavioural data. Finally, as noted by the authors, the type of cell activity modelled here is not specific to the FEF, as very similar pattern can be foud in the SC and the LIP area of the parietal cortex (Wurtz et al., 2001), as a consequence, this model could also be the basis of models of these two areas.

The reviewed cortical models address three main types of problems:

(1) the population coding of target positions in oculocentric and craniocentric coordinates and the corresponding transformations from one coordinates system to another (Zipser and Andersen, 1988; Gancarz and Grossberg, 1999; Deneve et al., 2001; Mitchell and Zipser, 2001),

(2) the operation of a cortical spatial dynamic memory, and its extension to sequence encoding (Dominey and Arbib, 1992; Mitchell and Zipser, 2001, 2003),

(3) multi-level selection processes in interaction with the basal ganglia (Brown et al., 2004).

The Deneve et al. model is representative of the current research concerning the generic problems of population codes and computations with population codes, taking into account noise and uncertainty in measurement. These topics are of course not specific to saccade generation, the model is however applied to a problem of change of coordinate frame which is of peculiar importance in saccade generation, where craniocentric and retinocentric representations coexist. This problem was addressed in an oversimplified manner in the Gancarz and Grossberg work, while the proposal of Mitchell and Zipser (2001) of a mixed coding strategy was not evaluated in terms of uncertainty.

Spatial short-term memory with remapping abilities is critical for saccade generation, this question has been explored in some SC *moving hill* models but also concerns cortical computations, at least in FEF and PPC. The question whether a master short-term memory site drives the other spatial representations, as proposed by Dominey and Arbib (1992), or whether various memories operate in parallel, as suggested by Bozis and Moschovakis (1998), is not decided yet. One should however note that SC feedback to the FEF (Sommer and Wurtz, 1998), which seem to preferentially transmit burst activity (high-pass filtering) signalling saccade occurence, provides the FEF with the necessary information to process such computations by itself.

The selection aspects of the cortical part of the Brown et al. (2004) model are intermingled with the operation of the basal ganglia circuits and only make sense when both systems are considered together. This once again highlights the relevance of modelling the interactions of reciprocally connected brain regions. The specificity of the FEF part of this model is the interpretation of the cortical layers interactions and roles it proposes. It is a quite promising proposal that has to be refined.

Finally, the problems of attentive modulation in the PPC, of working-memory operation in the DLPFC, of programms learning and restitution in the pre-

SEF and SEF and of motivation modulation of all these function by aCG were superficially or not addressed at all in saccade generation models, and should be in future works.

7 Conclusion

This review aimed at giving a glimpse of the evolution, during the last 30 years, of the conception of the operation of the various brain regions implied in the saccadic system, through the presentation of the succession of computational models built to explore this subject. More importantly, it also tried to help identify the current questions concerning the roles of the various regions involved and the precise mechanisms implementing these roles, and thus the *hot topics* to be explored by future models. These were exhaustively listed in the five previous discussions, we will thus lay stress here on two points that we think emerge from this review: the importance of thinking models not only in terms of local brain regions operation, but complementarily considering interacting brain regions, and, as a consequence, the necessity to theoretically study the dynamics of the resulting recurrent networks.

7.1 *Beyond the parts: modelling the whole*

We have seen in various reviewed studies examples of models of a given brain region designed to explain experimental results that happened to result from the operation of another brain region, or from the interaction of the modelled region with some other regions. The most representative example of this concerns SBG-only models: they all seek to produce accurate movements, usually fitted to the main sequence, while it is know clear that without their associated cerebellar circuits, the SBGs produce inaccurate movements that do not respect the main sequence. Understanding the accuracy of the saccadic system requires the study of, at least, the interactions of the superior colliculus, the cerebellum and the saccade burst generators.

Similarly, the remapping of targets in dynamic memories implies cortical or collicular interactions with the SBGs, and the target selection processes implicate cortico-basal-thalamo-cortical and collicular-thalamo-basal-collicular loops.

This advocates for systemic studies of the saccadic system (and of other brain systems also), in complement of those focused on a specific region only. Dominey and Arbib (1992) is an example of such an approach, however knowledge has accumulated since and many of their modelling choices are now out-

dated. First, the location of the control feedback loop is subject to much debate (Scudder et al., 2002). Its presence in the brainstem saccadic burst generators is of doubtful validity as no entirely satisfactory neural substrate for the resettable/leaky-integrator has been found. The two theories including the SC in the the loop suffer from various flaws. The cerebellum could be the missing piece of the feedback loop puzzle, even if current models of integration in the cerebellum are not completely satisfactory. Second, the old direct/indirect pathways interpretation of the basal ganglia circuitry has now been replaced by a much more complex pattern of interconnections. Modern knowledge concerning the basal ganglia did not give rise to numerous models of the oculomotor loop, however interesting proposals modelling other BG loops could be the basis of future work. Finally, beyond PPC and FEF, the operation of the other cortical areas involved in high level aspects of saccade generation (short, medium and long-term memory, learning of sequences, of response schedules, inhibition of response, motivational modulation) could be integrated.

A revised Dominey and Arbib model, summarizing the current knowledge of the brain circuitry involved in saccade generation an addressing the various *hot topics* highlighted in the five previous discussions, would provide a better understanding of the operation of the system as a whole and of the importance of the interactions between regions which have often be modelled independently.

7.2   Dynamics considerations

Recurrent neural network models (including internal loops) may exhibit various dynamical behavior, like convergence to some equilibrium point, chaotic trajectories, etc. Some of the reviewed studies considered the internal dynamics of their models, especially the SC models integrating the lateral short-range excitation and long-range inhibitions (see section 3.17), this was however not done in a systematic manner. Moreover, these considerations were almost exclusively drawn from simulations in a limited number of specific cases rather than from generic theoretical analyses.

Nevertheless, when considering large systems composed of multiple loops of interconnected subsystems, controlling the dynamics is crucial to ensure correct operation of the model, given any initial condition and sensory input. Thus, conducting an analysis of the system's dynamics becomes mandatory. The models to be analyzed are intrinsically nonlinear, but theoretical tools are available to study the dynamics of nonlinear systems, and some of them, like the contraction analysis developed by Lohmiller and Slotine (1998), are relatively simple. The contraction analysis has an additional advantage: contracting systems connected together, using a number of combinations (hier-

archy, feedback, etc.) with minimal constraints, produce contracting systems (Slotine and Lohmiller, 2001). As an illustration of this, contraction analysis was recently used to design a contracting basal ganglia model composed of contracting modules (Girard et al., 2005), which could in turn be embedded in a larger system composed of other contracting systems.

Beyond understanding the operation of a given function or behavior in the brain, the modelling of the large systems, like the saccadic circuitry, has an intrinsic scientific interest: exploring the solutions found by evolutionary processes to progressively interconnect new modules with older ones while keeping the whole system operational.

Acknowledgments

Abbreviations

aCG: anterior cingulate cortex

BG: basal ganglia

BN: superior colliculus burst neurons

BUN: superior colliculus build-up neurons

CBLM: cerebellum

CMAC: cereballar model arithmetic computer

DLPFC: dorsolateral prefrontal cortex

EBN: reticular formation excitatory burst neurons

EP: entopeduncular nucleus

FEF: frontal eye fields

FOR: fastigial oculomotor area

GABA: γ-aminobutyric acid

GCZ: gateable cortical zone

GP: globus pallidus

GPe: external part of globus pallidus

GPi: internal part of globus pallidus

IBN: reticular formation inhibitory burst neurons

IFN: inhibitory feedback neurons

IO: inferior olive

IT: inferotemporal cortex

LIP: lateral intraparietal cortex

LLB: reticular formation long-lead burst neurons

MLB: reticular formation medium-lead burst neurons

MN: ocular motoneurons

NRTP: nucleus reticularis tegmenti pontis

OPN: reticular formation omnipause neurons

PFC: prefrontal cortex

PPC: posterior parietal cortex

pre SEF: pre-supplementary eye fields

QV: quasi-visual neurons

RI: resettable integrator

SC: superior colliculus

SEF: supplementary eye fields

SBG: reticular formation saccadic burst generators

SNc: substancia nigra pars compacta

SNr: substantia nigra pars reticulata

SRT: saccade reaction time

STN: subthalamic nucleus

STT: spatio-temporal transform

TLLB: tectal long-lead burst neurons

TN: reticular formation tonic neurons

V4: extrastriate visual cortex area 4

VTA: ventral tegmental area

WTA: winner-takes-all mechanism

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