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Original Contribution

MODELING SLOW CORRECTING GAZE MOVEMENTS

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☐ **Abstract – Recent experimental results show evidence for the corrective role of postsaccadic drifts. This paper addresses the modeling of these slow correcting gaze movements (SCMs). Classical arguments to explain drifts are presented, both in the head fixed condition (pulse-step mismatch) and in the head free condition (vestibulo-ocular reflex (VOR)). The most significant behavioral and electrophysiological experimental data related to SCMs are then briefly reported, with the conclusion that SCMs have a clear corrective role, incompatible with classical explanations of drifts. Based on these experimental data, existing models of the saccadic system are then compared. A theoretical comparison of the classical Robinson model with an alternative model is proposed. Two possible (*slow* and *fast*) pathways involved in the control of SCMs are examined, and simulation results are presented. Finally, the discussion addresses the observed species differences in SCMs. The link between natural SCMs and electrical SC stimulations, and the interactions between saccades, VOR, and SCMs are also discussed.**

☐ **Keywords – saccades; drifts; gaze; modeling.**

Introduction

Most of the existing models of the saccadic system consider that saccades are always followed by pure fixation, for stationary targets. However, experimental data ([1–3] in the cat; [4,5] in man) clearly show that, both in head fixed and head free conditions, gaze saccades are very often followed (or even preceded) by slow gaze displacements. In the head fixed condition, these peri-saccadic "drifts" are usually thought to be due to a mismatch between the pulse and the step components in the saccadic command. On the other hand, it is classically attributed to an imperfect vestibular compensation in the head free condition. However, these conjugate peri-saccadic drifts might have a corrective role, and the superior colliculus (SC) could be involved in this process, because it is a key site for saccadic control. This hypothesis was recently proposed on theoretical (6) and experimental (2,3) grounds.

In this paper, a modeling approach is used to try to understand the possible pathways involved in slow peri-saccadic gaze movements, which can appear in the absence of target movement. On the basis of existing experimental data, several hypotheses are discussed as possible mechanisms for such slow correcting movements (SCMs). A model is proposed, and simulation results are presented. Possible species differences in the mechanisms generating SCMs are also discussed.

Drifts: Classical Explanations

Head Fixed

In the head fixed condition, postsaccadic drifts are usually attributed to a mismatch between the pulse and the step components of the saccadic command (7). During a saccade, the neural command to the ocular motoneurons can be split into two distinct parts. First, a high-frequency burst of activity overcomes the viscoelastic forces exerted by the oculomo-

tor plant and produces a fast shift of gaze: this is the pulse. Second, when the eye has reached the target, an appropriate constant activity is necessary to maintain the eye in the desired position, in spite of the presence of the elastic forces exerted by extraocular muscles: this is the step.

When the saccadic command is appropriate, the step component is adapted to the pulse, and the eye stays stationary in the orbit at the end of the saccade. But if there is a mismatch between the pulse and the step, the eye must drift exponentially from its position at the end of the saccade (specified by the pulse) to the position specified by the step. This drift is exponential, and its time constant is the time constant of the eye plant (200 ms).

When they are large, postsaccadic drifts are pathological. They can be induced by injections in the extraocular muscles that change the biomechanical properties of the eye plant (8). A pulse-step mismatch is specific to one particular direction: if, say, pulses were too large for steps in abducting saccades, then all abducting saccades should have same backward drift regardless of residual gaze error.

Head Free

In the head free condition, postsaccadic drifts are classically attributed to the vestibulo-ocular reflex (VOR). Indeed, if vestibular compensation is not perfect at the end of gaze shifts (VOR gain $\neq 1$), the residual head displacement will induce a slow drift of gaze. As in the case of the pulse-step mismatch, this mechanism has a priori no corrective role. These imperfections deteriorate vision and, thus, are corrected through adaptive mechanisms. The cerebellum plays a major role in the adaptation of the VOR gain, as in the adaptation of the pulse and the step (8).

SCMs: Functional Corrective Role

Behavioral Data

Guitton and colleagues (1) studied eye-head coordination in the cat. For multiple gaze saccades, they report ramps in gaze position during compensatory movements of the eyes. They tested the value of the VOR gain during these ramps in gaze and found that immobilizing the head had no effect on gaze trajectory during compensatory eye movements. Head immobilization immediately stopped the eye compensation, and no effect was observed on gaze trajectory. This seems to indicate that the VOR was active during these ramps and that the gaze drift was induced by a driving signal added to the vestibular compensatory signal.

On the other hand, Ron and colleagues (5) studied saccade-VOR cooperation in man during eye-head orientation to flashed targets. They also observed ramps in gaze position following saccades, but they conclude that this is due to the action of a corrective VOR. They suppose that VOR gain is controlled by the residual gaze error at the end of the saccade. Interestingly, their conclusion relies on the classical explanation of gaze drifts in head free, but suppose that VOR gain would be controlled by residual gaze error, thereby having a corrective function. This hypothesis seems to be in contradiction with Guitton et al. (1) results showing that VOR compensation is perfect during these ramps in gaze position. Clearly, there are possible differences between cat and man eye-head coordination, but this is not the only possible explanation. Moreover, Guitton et al. did not study these SCMs in details, and Ron et al. did not test the value of the VOR gain during their observations.

In the head fixed condition, Kapoula et al. (7) and Collewijn et al. (9) report in man very small postsaccadic drifts (less than 1° and 5°/s). More recently, Missal et al. (2) reported data from the head restrained cat, with SCM amplitudes and velocities much larger than those observed in man (5° and 45°/s). For both terminal and intersaccadic drifts, they report a good correlation between SCM velocity and SCM amplitude. SCM amplitude is also correlated with residual gaze error at the end of the saccade. The dynamics of SCMs can vary, and their direction is quite variable, despite a clear corrective role. These findings are difficult to reconcile with the classical pulse-step mismatch explanation of SCMs.

Electrophysiological Data

Olivier et al. (3) studied the activity of tectoreticulospinal neurons (TRSN) in alert, head-fixed cats, more specifically during postsaccadic slow eye movements. They found that the probability of occurrence of postsaccadic drifts was higher when TRSN discharge continues after the saccade. These slow movements were associated with the presence of a residual gaze error at the end of the saccade, when a correction is realized by slow eye (and head) movements. Thus, it is quite reasonable to suppose that the SC might be involved in the generation and the control of SCMs. In parallel with this experimental study, Lefèvre et al. (6) proposed on theoretical grounds that the SC might be involved in the control of SCMs, if one supposes that it codes instantaneous gaze error.

Corrective Role

Experimental data briefly reported above are very difficult to reconcile with classical explanations of postsaccadic drifts. In fact, the principal element against these hypotheses is related to the clear experimental evidence that SCMs are corrective. Both pulse-step mismatch and imperfect VOR mechanisms are "passive." Following these explanations, SCMs would have no corrective role. For instance in the head-fixed condition, a pulse-step mismatch is completely predictable in its direction and dynamics; there is no reason for the SCM to be adapted to the characteristics of each saccadic movement, for instance, direction and amplitude of residual gaze error.

Most of the studies having investigated the SCMs in details have a common conclusion, insisting on the corrective role of SCMs, under the control of residual gaze error at the end of the saccadic component of the head free or head fixed movement (1,2,3,5). The next section is based on a modeling study and investigates the possible pathways involved in this corrective postsaccadic function.

Modeling Approach to SCMs

One Unique Saccadic Burster Pathway

Most models of the saccadic system rely on one unique pathway to generate eye saccades, the so-called burst generator. All these models are extensions of the model proposed by Zee and colleagues (10), which is based on a local feedback loop evaluating residual gaze error. The error is defined by comparing desired eye position with an internal copy of instantaneous eye position and is then fed into the saccadic burst generator, to generate desired eye velocity. When the error signal reaches near-zero levels, the saccade ends crisply, and the eye stops its movement, fixating the new target. More recent models of the saccadic system place the SC inside this feedback loop, but they all rely on one unique pathway relating instantaneous gaze error to eye velocity: the saccadic burst generator (Figure 1).

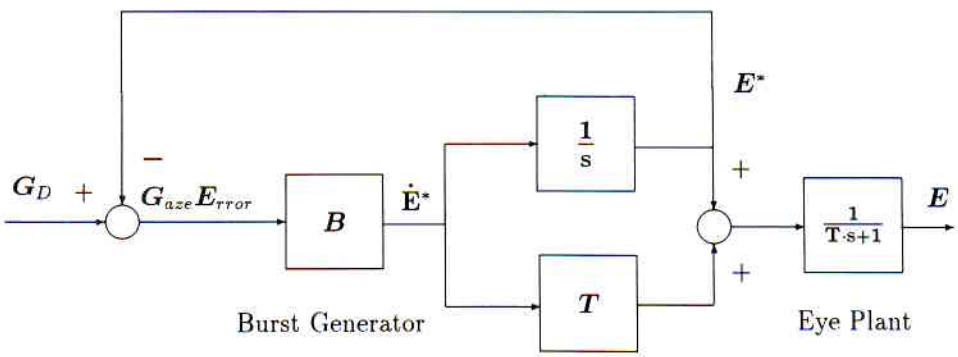


Figure 1. Robinson model of the saccadic system. *T* is the eye plant time constant (200 ms) and *B* is the gain of the Burst generator (18, with output saturation ± 500). G_D is the desired gaze position, *E* is the eye position, E^* is the internal copy of the eye position. *Laplace* notation is used.

In these models, the saccadic system is usually supposed to produce saccades of appropriate size. If saccades stop too early (undershoot), the only possibility to acquire the target is then to trigger a second, corrective saccade. In other words, the residual gaze error signal can only influence the eye trajectory through the burst pathway (saccade). Within this context, slow peri-saccadic gaze displacements are classically attributed to a peripheral cause, that is, a pulse-step mismatch (or an imperfect VOR in head free condition).

But is was demonstrated in "Corrective Role" that this explanation of slow peri-saccadic gaze displacements cannot apply to most of these movements. In particular, SCMs have a clear corrective role and quite variable dynamics, which is incompatible with a simple pulse-step mismatch interpretation. Therefore, previous models of gaze control seem to lack essential properties to reproduce realistic SCMs.

In fact, theoretically, there is a possible explanation for these SCMs, without modifying the structure of classical models. These slow correcting gaze movements might be due to an intermediate firing of burst cells, which would correspond to a nearly stopped saccade. This is the only way to explain such peri-saccadic slow movements with one unique burster pathway. Indeed, in Figure 1, the transfer function linking desired gaze position (G_D) to eye position (E) is the following:

$$E(s) = \frac{G_D(s)}{\frac{1}{B} \cdot s + 1} \qquad [1]$$

where the parameter B represents the gain of the saccadic burster, and s corresponds to the *Laplace* notation. In equation [1], the static gain between E and G_D is 1, and the time constant is $1/B$. This means that the dynamics of saccadic movements only depend on the B parameter. If we want to generate slow peri-saccadic gaze displacements through this pathway, we must hypothesize that B can take very low values. The nominal value of B is about 18 during saccades, yielding a value of 56 ms for the time constant (11). Experimentally observed SCM time constants are in the range of 100–500 ms (2), requiring a value for the B parameter ranging between 2 and 10. Hence, in the classical saccade models, intermediate levels of firing on burst cells would be associated with an abnormally low value of the B parameter and a larger time constant in the eye movement.

This possible explanation of peri-saccadic slow gaze displacements would be due to a particular state of the saccadic gate, in between the 2 classical stable states of the saccadic toggle: omnipause neurons (OPNs) on, excitatory burst neurons (EBNs) off, or OPNs off, EBNs on. This intermediate state would correspond to a partial inhibition of EBNs by OPNs, both cells populations discharging weakly. This explanation might also account for the appearance of peri-saccadic drifts, when the rostral (fixation) zone of the SC is stimulated at various intensities during gaze shifts. This protocol would certainly affect the activity levels of brainstem omnipausers, known target areas for the SC fixation cells (12).

The likelihood of this explanation for SCMs will be addressed in the discussion. But it is worth noting that this hypothesis is attractive, at least theoretically. Indeed, in the head free condition, this intermediate EBN firing might be coupled with a partially restored VOR, participating in gaze error reduction together with the burst cells. So, in the head fixed condition, bursters alone would control SCMs, while bursters and a corrective VOR would work synergistically, head free.

Two Parallel Pathways, slow *and* fast

In the previous section, we concluded that for most existing models of the saccadic system, there is only one possible explanation for peri-saccadic slow correcting gaze movements (SCMs), that is, the action of a partially inhibited burster pathway. However, we recently proposed a model of gaze control (13) that can provide an alternative hypothesis for the control of slow corrective gaze displacements. This model is based on gaze velocity feedback

to the SC and proposes an original distributed parameter system to model collicular function. Figure 2 shows a simplified version of the original model, for head fixed movements. When compared with Figure 1, 2 important differences are present:

1. The integral action in Figure 1 is replaced by a mathematically equivalent combination of an eye plant internal model with a positive feedback loop.
2. There is one unique pathway from gaze command (G_D) to eye movement (E) in Figure 1, whereas there are two pathways from gaze command (ΔG) to eye movement (E) in Figure 2, a *fast* route (classical, through saccadic bursters), and a *slow* pathway, active during both saccades and slow phases (fixation or SCMs).

In this case, the hypothesis of SCMs controlled by an attenuated burst (*fast*) pathway, possibly coupled with a corrective VOR, can still be valid, as in the previous section. However, in the model described in Figure 2, there is a more natural way to explain peri-saccadic slow gaze displacements: through the second *slow* pathway, linking gaze command (ΔG) with eye displacement (E) when the saccade is stopped (saccadic burst gate open). During slow phase, the transfer function linking desired gaze (ΔG) to eye position (E) is the following:

$$E(s) = \Delta G(s) \cdot \frac{K^*}{T^* \cdot s + 1} \qquad [2]$$

with

$$K^* = \frac{e_g \cdot t_v}{1 + e_g \cdot (t_v - 1)}$$

and

$$T^* = \frac{T}{1 + e_g \cdot (t_v - 1)}$$

In equation 2, T^* depends on three parameters: T, t_v, and e_g. T is the eye plant time constant, and e_g is fixed by the slow phase VOR time constant. Parameter t_v may vary and influence movement dynamics. A larger t_v yields faster saccades and faster peri-

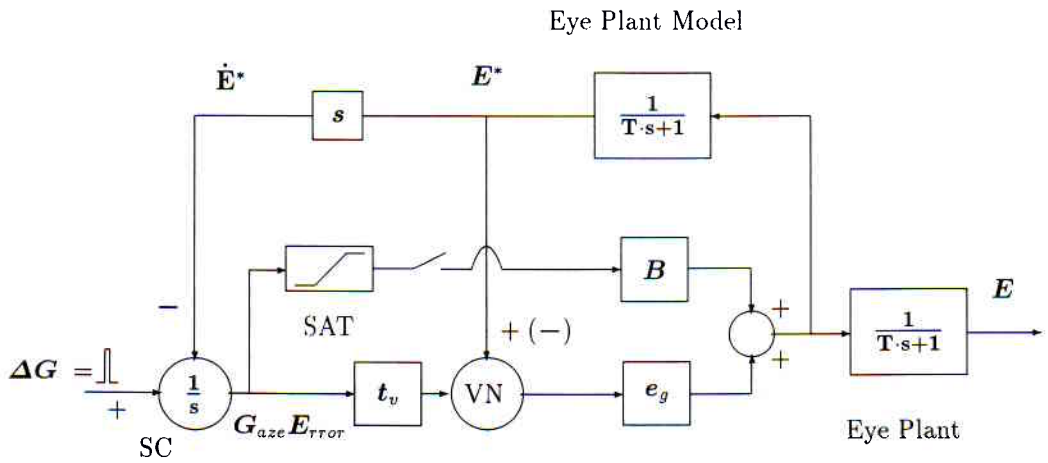


Figure 2. Simplified version of the Lefèvre and Galiana (13) model of the saccadic system. The switch in the model (output of SAT) is closed only during saccadic mode: this is the *fast* pathway; t_v, e_g, and B are static gains; T is the eye plant time constant. ΔG is the desired gaze displacement, E is the eye position, and E^* is an internal copy of E. Parameters were adapted to fit cat responses: $B = 2$, $t_v = 0.5$, T = 200 ms, $e_g = 0.5$ (saccadic mode), $e_g = 0.98$ (slow phase mode), SAT = gain of 4 with output saturation ±20. *Laplace* notation is used.

saccadic slow gaze movements to the same target, corresponding physiologically to higher mean collicular activity.

In particular, if one supposes that perisaccadic SCMs are controlled by this *slow* pathway, dynamics of saccades are expected to be correlated to dynamics of SCMs, that is, faster saccades of equal size should be followed by faster SCM's for equal postsaccadic errors. This is not the case if SCMs are only controlled by the *fast* pathway, because intermediate burst firing is expected to vary from trial to trial. An experimental comparison of saccade and SCM dynamics should be performed.

In the head free condition, gaze drifts might also be controlled by this *slow* pathway, as suggested by Guitton et al. (1) data. This slow pathway would then add an active driving signal to the vestibular compensation, that is, a *corrected* VOR and not a *corrective* VOR. However, it is clear that both signals could be present at the same time, yielding a *corrected corrective* VOR.

Simulation Results

Simulation Parameters

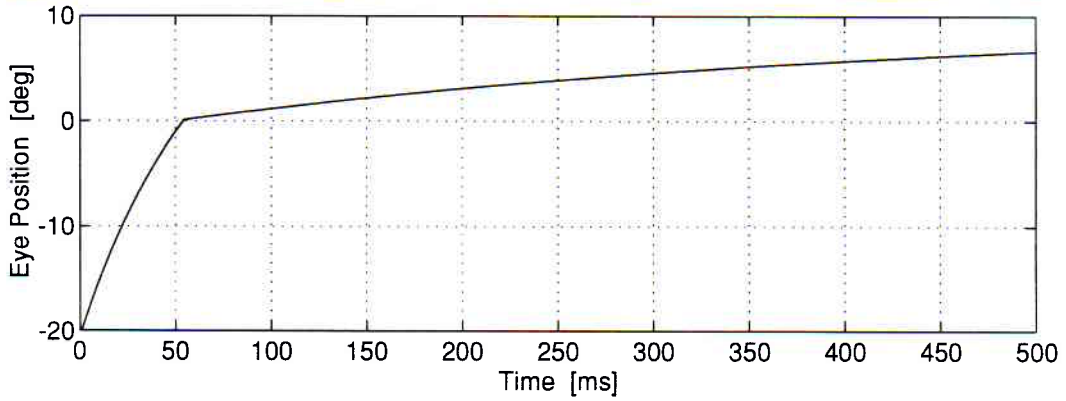
Simulations are based on the model proposed in Figure 2. This model is a simplified version of the model proposed by Lefèvre and Galiana (13), where the collicular network is represented by its resettable integral action. In this paper, only simulation data in the head fixed condition are presented, but the same kind of results can be obtained when the head is unrestrained. Model parameters are chosen in order to fit cat responses. This is due to our intention to show simulation results closely related to experimental data of Missal et al. (2) and Olivier et al. (3), from cat SCMs in head fixed.

Matlab and Simulink softwares were used on a Sparc station 10 (integration step 1 ms). The eye plant was modeled by its first order approximation (time constant 200 ms). Responses with a second order eye plant (second time constant 16 ms) yield results very close to those presented in this paper, with smoother velocity profiles closer to actual experimental data. We preferred to show results with a first order eye plant in order to emphasize transition from fast to slow phases and to unmask central mechanisms controlling saccades with SCMs. Figure 2 legend provides numerical values for model parameters.

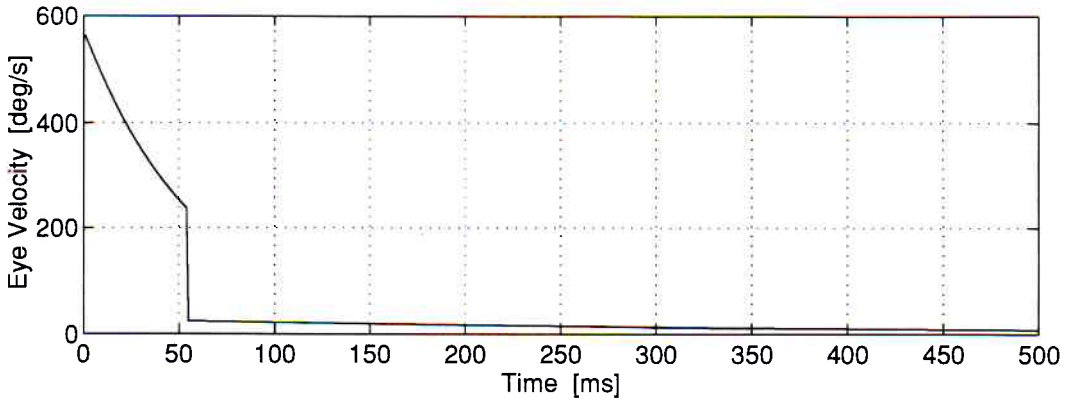
Figures 3 and 4 present two simulation examples, in the case of a rather slow final SCM (Figure 3) and of a rather fast intermediate SCM combined with a corrective saccade and with a small terminal SCM (Figure 4).

Figure 3 presents an eye saccade of 20°, followed by a large SCM, for an initial 30° gaze error (a: position trace; b: velocity trace). The saccadic mode was switched off 10° away from the target. Afterwards, the residual error drives the eye exponentially toward the target (time constant of 400 ms, following equation [2], with $t_v = 0.5$). At the time of phase transition, eye velocity drops from 240°/s to 25°/s instantaneously (first order eye plant), even though the residual error (the driving signal) does not change instantaneously. Figure 3c shows the value of the following ratio: $\dot{E}/G_{aze}E_{rror}$. This variable is related to the inverse of the time constant relating eye movement to driving signal. In slow phase, the value of this gain is 2.5, which is the inverse of T^* for $t_v = 0.5$.

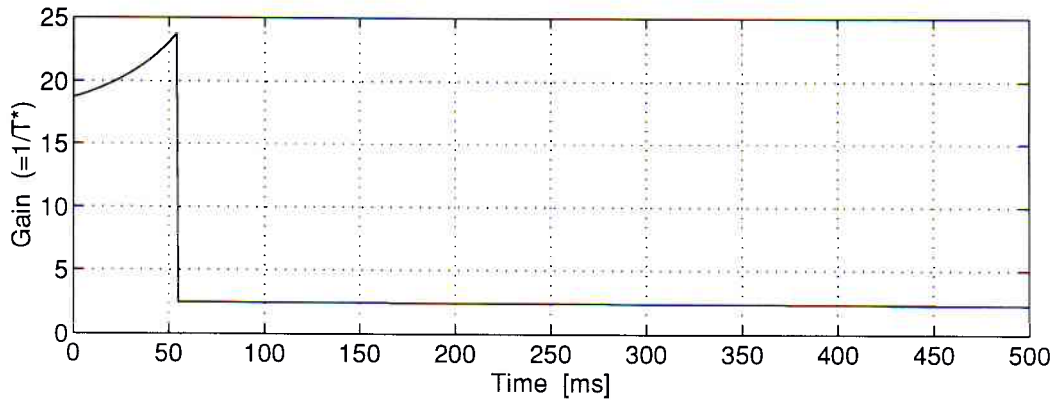
Figure 4 shows simulation results related to a faster SCM, which is followed by a corrective saccade. In this simulation, the saccadic mode was arbitrarily interrupted during a period starting 30 ms after saccade onset and lasting 100 ms. Parameter t_v was set to 2 in order to observe faster SCMs. At the time of saccade interruption, eye velocity drops from 330°/s to 45°/s instantaneously. The same abrupt transition in eye velocity occurs when the corrective saccade starts and finishes. The exponential profile of SCMs is easier to grasp on Figure 4b, during the intermediate SCM, because this SCM was associated with a higher t_v gain than in Figure 3. Figure 4c yields the same kind of information as Figure 3c, but this



(a)



(b)



(c)

Figure 3. Simulation result obtained with the model presented in Figure 2. The initial eye position is −20°, and the desired gaze shift is 30°. The saccade is artificially stopped when residual gaze error is still large (10°). The system enters then in slow phase mode (see text), and the remaining gaze error drives the SCM. (a) Eye position versus time. (b) Eye velocity versus time. (c) Value of $\dot{E}/G_{aze}E_{rror}$ versus time, which represents the inverse of the time constant of the SCM ($T^* = 0.4$ s) during slow phase mode.

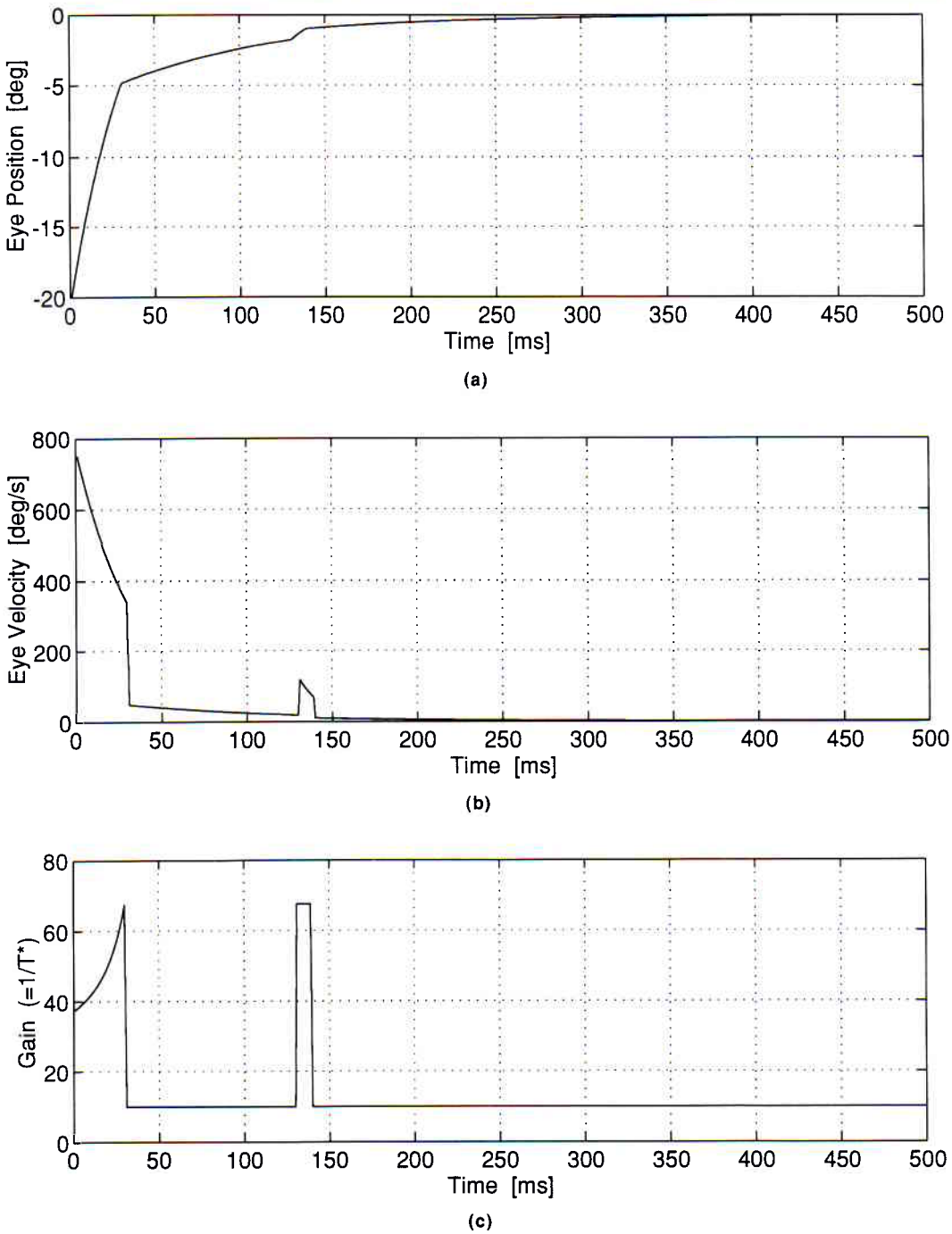


Figure 4. Simulation result obtained with the model presented in Figure 2. The initial eye position is −20°, and the desired gaze shift is 20°. The saccade is artificially interrupted during a period starting 30 ms after saccade onset and lasting 100 ms. During this 100-ms period, the system enters in slow phase mode, and the remaining gaze error drives the SCM, which is then followed by a corrective saccade. (a) Eye position versus time. (b) Eye velocity versus time. (c) Value of $\dot{E}/G_{aze}E_{rror}$ versus time, which represents the inverse of the time constant of the SCM ($T^* = 0.1$ s) during slow-phase mode. In this simulation, parameter t_v is set to 2, in order to reproduce an intermediate SCM with faster dynamics (2).

time the value of the gain during both intermediate and terminal SCMs is larger ($1/T^*$ for $t_v = 2$). Thus, our model is capable to reproduce SCMs of variable dynamics, relying on physiologically plausible mechanisms (population cells recruitment at the collicular output). These results are compatible with SCM cat data (2,3).

In these simulations, we showed how the modulation of t_v parameter could modify SCM dynamics (see equation [2]). Physiologically, this action would correspond to a more intense recruitment of SC output cells, yielding faster saccades and faster SCMs, without modifying saccade amplitude. In the case of extremely fast SCMs, it is worth noting that it would probably be necessary to introduce a saturation at the output of t_v operator in order to maintain saccade velocities in a reasonable domain. Indeed, the *slow* pathway is also active during fast saccadic mode in the model; this saturation is already present at the Burst level in the model and is biologically relevant. Another way to allow the model to generate reasonable saccades with very fast SCMs would be to impose two different values for t_v in the model (for fast and slow phase modes).

Discussion

Species Differences

Several authors demonstrated the corrective role of SCMs. Therefore, the frequency and the relative importance of these SCMs is necessarily related to the precision of the saccadic system. In other words, the high precision of the saccadic system could mask the presence of such corrective mechanism. This could explain the relative absence of SCMs in species having a very precise saccadic system (man, monkey). In man, Collewijn et al. (9) report very small postsaccadic drifts, but their experimental paradigm, based on repetitive saccades, produced very precise saccades (undershoot of less than 0.5° over ± 80° range). If present, a corrective mechanism would certainly be masked in this situation. In man, Kapoula et al. (7) also report weak postsaccadic drifts, but they point out their possible underestimation. Indeed, drift velocity is usually averaged over a period of 100 ms starting 20 ms after the end of the saccade, leading to a drastic underestimation of their importance, because their profile is exponential. Larger correcting movements were recently observed in humans in the head free condition (5). Ron and colleagues flashed two visual targets consecutively with variable latencies. With their protocol, gaze saccades were often followed by a slow gaze movement.

This shows that the occurrence of SCMs, in man, is very sensitive to the experimental conditions. If the experimental protocol produces small residual gaze errors at the end of gaze shifts, the corrective mechanisms will be masked. Because of the good precision of the monkey and human saccadic systems, it is necessary to choose appropriate experimental conditions and to estimate drift velocity very precisely in these two species.

The saccadic system of the cat is much less precise. This could be related to the larger size of the area centralis of its retina. A large proportion of cat gaze saccades, both in the head fixed and in the head free conditions, are followed by slow correcting gaze movements (1–3). These drifts are corrective, and their velocity is proportional to residual gaze error at the end of the saccade (2). They are often associated with high-frequency discharges of TRSNs (3), indicating that the SC could be involved in their control. Moreover, Cullen and Guitton (14) recently recorded from cat inhibitory burst neurons (IBNs) during SCMs, reporting that cat IBNs discharge weakly during these peri-saccadic slow gaze movements. This observation leaves open the 2 possible routes for the control of SCMs in the cat: the *slow* direct pathway and the *fast* route through attenuated Burster discharge.

On the other hand, the same authors (14) report, in the monkey, that burst (IBNs) discharge stops crisply at the end of saccades even if followed by drifts. This implies a necessary nonburster SC projection to control SCMs,

because Munoz and Wurtz (12) also report that electrical SC stimulations can produce staircases with intersaccadic ramps in eye position in the monkey. Cullen and Guitton (14) suggest that the upstream switching between fast and slow modes of tracking occurred at different thresholds in these two species (cat versus monkey). These experimental results are of crucial interest in order to understand possible species differences in SCM generation. Their confirmation over a large data set would be fundamental within the context of SCM study.

Electrical SC Stimulation

Electrical stimulation of the SC in the head fixed alert cat evokes gaze shifts with perisaccadic slow gaze displacements (15,16). A more recent study (17) investigated in more details electrically evoked SCMs in the cat. These movements occur for stimulation periods longer than 150 ms, and their direction is roughly the same as the preceding saccade. Electrically evoked SCMs have amplitudes and velocities in the same range as visually triggered SCMs. These preliminary results indicate that the SC is probably involved in the control of SCMs.

From a theoretical point of view, several important differences are expected, between electrically evoked (EE) and visually triggered (VT) SCMs. Indeed, if SCMs are supposed to be controlled by residual gaze error through the SC, then EE and VT SCMs should be different. In Figure 2, instantaneous gaze error is coded inside the SC; in particular, during SCMs, collicular output (gaze error) is continuously reduced as the eye moves toward the target. For EE movements, the control of saccades and SCMs is certainly heavily affected by the artificial electrical stimulus inside the SC. We addressed this problem of collicular control of EE saccades (13), with the conclusion that the collicular feedback loop is probably partially, if not totally, open by such electrical stimulation. The end of EE saccades would be triggered by the lateral spread of SC activity onto the fixation zone of the SC (open-loop mechanism depending only on the properties of the collicular network).

Within this context, it is possible to predict that for EE SCMs, the SC output is probably constant during the complete duration of the movement. Eye velocity is then expected to be rather constant during the evolution of SCM. The only cause of eye velocity decrease would be the well-known effect of orbital eye position on eye movement dynamics (property of the eye plant) (18). On the other hand, VT SCMs are expected to have a more exponential profile, because SC output (coding residual gaze error) continuously decreases. However, it is worth noting that this effect would come to light only when the relative change in residual gaze error is sufficient. For instance, this is not the case of intermediate SCMs because the change in residual error is usually small with respect to the absolute value of residual error. But this is the case for terminal SCMs that start with a significant residual error. This should be investigated in more detail by appropriate experiments.

Saccade-VOR-SCM Interactions

The VOR is known to play a major role in eye head coordination. For large shifts of gaze in the head free condition, the VOR is cancelled during the first part of the movement; vestibular compensation is then restored near the end of the gaze saccade and stabilizes gaze on the target (11,19). In head free, gaze drifts are classically attributed to an imperfect vestibular compensation (VOR gain $\neq 1$). Ron and colleagues (5) studied eye-head orientation toward flashed targets and concluded that postsaccadic gaze drifts were corrective. They propose that this mechanism could be due to a corrective VOR. This supposes that VOR gain would be modulated, after the saccade, in order to correct gaze position and to reach the target. This corrective function implies that VOR gain would be modulated by residual gaze error.

This hypothesis, relying on VOR gain modulation, may be related to the experimental re-

sults recently reported by Lefèvre et al. (19). On the basis of a systematic comparison of head fixed eye saccades with same amplitude head free eye saccades, this study investigated the role played by the VOR in eye-head orientation in man. As a major result, VOR gain modulation during large shifts of gaze was related to saccade parameters. VOR gain was gradually restored at the end of the gaze shift, from a zero value (no vestibular compensation) to one (perfect gaze stabilization at the end of the gaze shift). The period of VOR gain restoration was constant, and it varied linearly with residual gaze error. These data are in agreement with the Ron et al. (5) results. Indeed, if a gaze saccade stops before a perfect reduction of gaze error, the model proposed by Lefèvre et al. (19) predicts a VOR gain less than unity and a correction of gaze position via a partial vestibular compensation (gaze drift due to a corrective VOR). Thus, the experimental data of Ron et al. (5) and Lefèvre et al. (19) are complementary; both studies indicate that VOR gain might be modulated by residual gaze error during gaze shifts in man.

Nevertheless, even though the notion of a corrective function for VOR in head free gaze drifts is very attractive, it is worth noting that it is still very speculative. This phenomenon requires further careful investigation, given the apparently contradictory results of Guitton et al. (1), who report an intact VOR compensation during gaze drifts in the cat.

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