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Modelling Eye-Head Coordination without Pre-Planning – A Reflex-Based Approach*

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Abstract— **The gaze orientation system is a prime example of the CNS using multiple platforms to achieve its goal. To move the gaze in space, the eyes, head, and body cooperate to place the image of the target on the fovea. Understanding the underlying neural circuitry innervating this collaboration could also be a cue to understanding other movement related CNS tasks involving multiple platforms, i.e., posture and locomotion. Basically two major network topologies for modeling the gaze orientation system have been proposed: the independent controller model and the shared gaze feedback controller model. In the independent controller model, each platform (i.e., eyes, head or trunk) receives its own share of the retinal error (distance of the target from the current gaze position) independent from other platform(s) and its goal is to null its individual error, whereas, in the shared gaze feedback controller all platforms collaborate to null the shared global error, which is calculated on the fly using feedback from all platforms or reflexes. Each of the mentioned general topologies has its own supporters and the question is which does the CNS actually use. In this article, based on evidence from neurophysiology and behavior, complemented by simulation data, it will be shown why a shared feedback controller is the better candidate for this task. More specifically, simulations of an updated Prsa-Galiana model (the Shared Sensory-Motor Integration (SMI) model) will be discussed in more detail and, where applicable, compared with other popular models, including independent and shared controller models. It provides plausible explanations for observations on gaze shifts with various interventions.**

I. Introduction

Understanding how the CNS innervates the collaboration of platforms during gaze orientation can be a cue to understanding its role during other complex behaviors. Basically there are two schools of thought for explaining this process:

- Independent controller models: In this scheme, each platform tries to null its own individual share of the overall gaze error, allocated apriori.
- Shared gaze feedback controller models: In this scheme, all platforms collaborate to null the overall gaze error that is calculated on the fly using feedbacks from platforms.

In the next sections, by referring to data from neurophysiology and behavior, it will be explained why the gaze feedback schemes can replicate a broader set of experimental data. Moreover, simulations from the updated Prsa-Galiana model ([1]), which is of this type, will be shown to justify this claim. Where applicable, this model will be compared with other popular models on the basis of efficiency in replicating behavioral and central responses and their predictions in different gaze orientation scenarios. For simplicity, our focus in this study will be on gaze orientations in the horizontal plane in fixed-body head-free scenarios.

Some of the major questions that are intended to be addressed, at least in part, in this article are:

- Is it necessary to have trajectory planning, or gaze error decomposition between platforms to be able to replicate behavior and model gaze shifts?
- How important is the impact of the circuit topology on platform coordination in modeling gaze orientation and the compatibility of the model with neurophysiology?
- Do we need highly independent controllers governing different types and phases of gaze and eye movements, or is a shared controller enough? What does physiology suggest?

II. Brief Physiological Background for Gaze Orientation

Numerous centers are involved in the process of gaze orientation. In brief, some of the key players in gaze orientation in the horizontal plane are:

- The Superior Colliculus (SC): the SC is a visuomotor map with its dorsal layers forming a retinotopic map and projecting to its intermediate and deep layers with motor properties. The superficial layers of the SC seem to reflect the location of targets relative to the fovea, rather than their nature [6].
- The Paramedian Pontine Reticular Formation (PPRF) contains burst neurons that fire during saccades: a subgroup of the PPRF burst neurons project directly to ipsilateral motoneurons. Another subgroup of these neurons are the Omni-Pause Neurons (OPN) that only discharge tonically during the slow phases. These neurons trigger saccades by pausing and releasing their target burst neurons [7].

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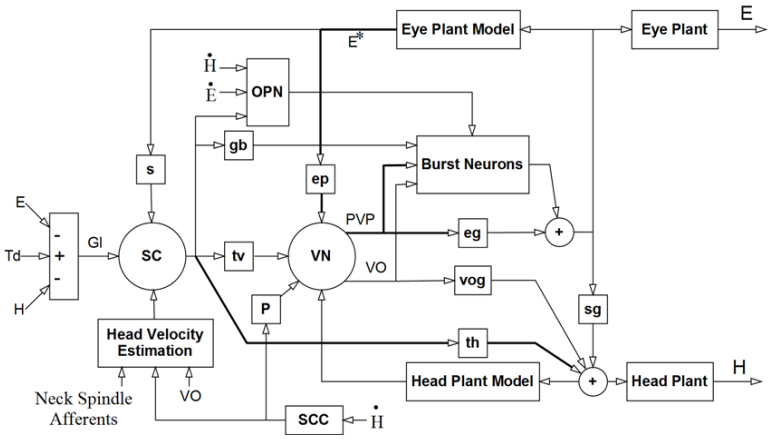

Figure 1. The Updated Prsa-Galiana Model (the SMI model). VO cells and Neck Spindle Afferents have been added to the original model [1].

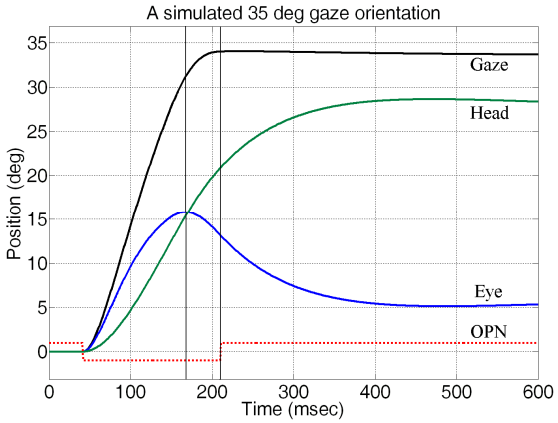

Figure 2. A 35 degree gaze shift simulated by the SMI model. The moments of maximum eye deviation and switching (OPN) have been highlighted with vertical lines.

- The Vestibular Nucleus (VN) and Nucleus Prepositus Hypoglossi (PPH): a group of neurons in the VN called the position-vestibular-pause (PVP) neurons are key components in the Vestibulo-Ocular Reflex (VOR) [8]. Another group of VN neurons are the Vestibular-Only (VO) neurons that encode the passive component of head movements [9]. Neurons in the PPH interconnected with the Medial VN have tonic discharges proportional to eye position and hence, embody an efference copy of eye position [1].
- Sensors and actuators: the sensors include the retinas, the horizontal canals and otolith organs, and the neck muscle spindles. The actuators are the muscles of the eyes and head.

The SC is a common component in all gaze orientation models [1, 10-11]. In head-free scenarios, electrical stimulation of the SC results in both eye and head movements [12]. In independent controller models its role is to define the initial target and decompose gaze error for each plant. In the shared gaze feedback controllers, using the feedbacks provided to the SC, the SC encodes the ongoing gaze error during the gaze shift [13]. There is evidence of eye and head related feedback signals from the Mesencephalic Reticular Formation (MRF) to the SC [14]. Moreover, activities on the SC map are known to be modulated with the ongoing gaze error [6, 14-15]. Thus, neurophysiological evidence seems to be more consistent with the shared gaze feedback controller scheme.

If the eye and head plants were driven using the same motor error signal, one would expect to see a high correlation between the eye and head movements. Indeed, this is the case. The eye position trajectory is highly correlated with the head velocity trajectory [16]. It is also known that the eye and head plants share many premotor centers [5, 16-17]. These cues also favor the shared gaze feedback controller since shared premotor centers for different plants makes independent controller schemes redundant.

Another aspect for modeling gaze orientation is whether we need independent circuits to replicate different phases of movements – saccade vs. fixation for example. From the physiological viewpoint, there is much overlap regarding the neural substrates that govern the saccadic phase and the VOR phase (stabilization) of gaze orientation [1]. Moreover, some brainstem regions, e.g., PH-VN, are known to contribute to, and modulate with, different types of eye movement, which suggests the existence of shared circuitry which should contribute at least partially to any eye-head movements. Thus, depending on the nature and mixture of input stimuli and the desired task, a circuit could generate different responses.

An issue worth mentioning in this section is the integration of velocity and position signals in different neural centers. The firing rate of motoneurons can be divided into a tonic component, which is proportional to position, and a phasic or velocity component [18]. This trend holds for other neural centers, e.g., burst neurons and PVP neurons, meaning that we rarely observe pure position or velocity neurons. Hence, it is not necessary nor physiologically justifiable to keep velocity and position components of a movement drive separate. An implication is the use of plant models instead of perfect neural integrators to estimate eye position [1].

III. The Updated Prsa-Galiana Model: Shared Sensory-Motor Integration (SMI)

Fig. 1 shows the updated Prsa-Galiana model (SMI). The major differences between this model and the original Prsa-Galiana model are the incorporation of VO cells and the integration of neck spindle afferents. We will assess the performance of this model in replicating different observed behavioral and central responses. All simulations were performed in Simulink (Mathworks, MA) with a sampling rate of 1 kHz. The major parameter elements are shown in Table I, but readers wishing a full model definition can contact us by email. The model is used to replicate classical head-free saccades including both the saccadic redirection of gaze and the following gaze stabilization where one would observe a compensatory ocular response (VOR) to head perturbations (Fig. 2).

IV. Results & Behavioral Implications

Defining saccade intervals: The transition between the saccadic part and the VOR part of gaze saccades is triggered by OPNs. The moment when the eye reaches its maximum deviation in its orbit is considered by many to be the moment

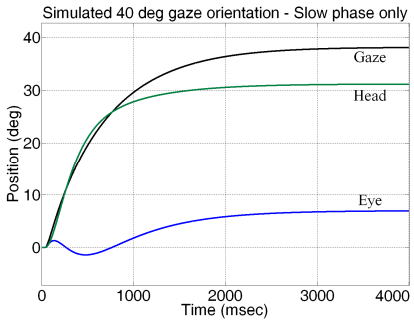

Figure 3. Drifting in the slow phase: simulation of a 40 deg gaze re-orientation in slow phase with Burst cell loss.

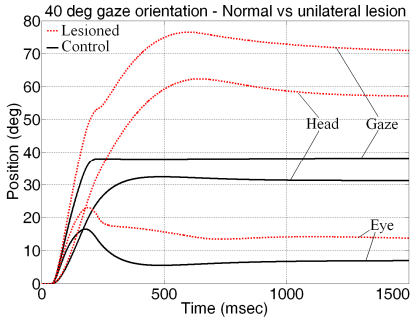

Figure 4. Prediction of the SMI model for acute unilateral vestibular lesion; Compare with [4].

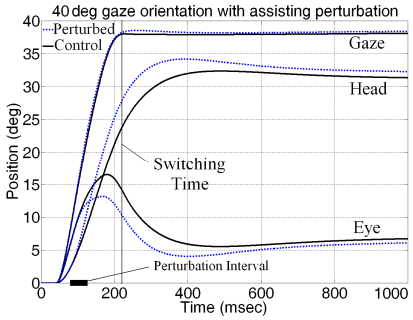

Figure 5. Simulated 40 degree gaze orientation with assisting head perturbation during the saccadic interval [2].

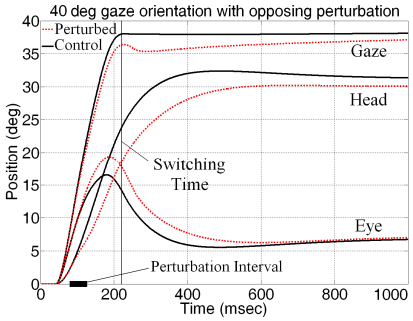

Figure 6. Simulated 40 degree gaze orientation with opposing head perturbation during the saccadic interval [2]. Compare to Fig. 5.

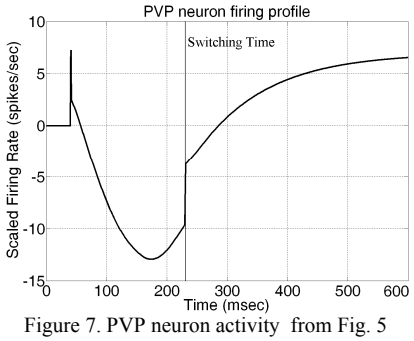

Figure 7. PVP neuron activity from Fig. 5 control simulation; Compare with [3].

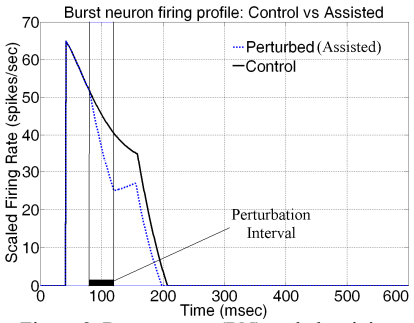

Figure 8. Burst neuron (BN) scaled activity from Fig. 5 simulation: contains a soft saturation followed by exponential decay (Compare with [5]). An assisting perturbation (blue) inhibits the BN firing profile.

of switching [10-11]. However, experimental data show that, quite often the switching occurs after this moment meaning that before the switching the eye can have a 'VOR-like' turn around and the eye velocity at the moment of switching can be negative (considering the gaze shift direction positive) [10-11, 16]. The Prsa-Galiana model is the only model capable of predicting this phenomenon. Fig. 2 shows a simulation by the SMI model that highlights this prediction. It is interesting to see that even during the counter-rotation of the eyes during the saccadic part of gaze orientation, the gaze is still approaching the target. This is not surprising considering the shared gaze feedback structure since in this scheme the goal is to null the overall gaze error and not the individual errors and hence, the moment of maximum ocular deviation is just like any other moment in this transition. With this perspective, the SMI model uniquely defines the saccadic part of gaze orientation for both eyes and head as the interval in which the OPNs pause their discharge. In this viewpoint, there is no individual eye goal or head goal as in other models [10-11].

In the shared feedback scheme, the collaboration of the plants involved in the gaze orientation results in increased robustness in a *gaze* trajectory. However the individual contributions of the eyes and head can vary with model parameters in different loops – allowing for variable eye and head trajectories despite robust gaze profiles. These variations are observed among and within individuals. To obtain the same phenomenon in independent controllers, a new gaze decomposition method needs to be introduced to create each new profile (e.g., all the 'noise' in SC mapping).

Integrators and gaze saccades: An interesting observation from experimental results is the drifting of gaze during the slow phase (fixation) after gaze reorientation (VOR part) [3, 11]. Other models rely on a mathematical integration of head perturbations so that the eye cancels the effect of head movement on gaze by counter-rotating with the same magnitude as the head (the VOR) [10]. This keeps the gaze constant on target. However, experimental results show the possibility of gaze drifting during the slow phase [11] – integration is not perfect! In addition, separate processes imply that saccades and gaze re-orientations are impossible without burst cells. However, patients with burst neuron lesions can still realign gaze, albeit much more slowly - gaze orientation movements are carried out in the slow phase of necessity. Our model replicates these findings: gaze re-orientations during the slow phase are possible because the gaze error drive can overcome any oppositely-directed vestibular compensation (Fig. 3). This also implies that the instant of saccade end-fixation start need not be very accurate – both modes can carry gaze to the same location in smooth steps, but with different speeds.

Vestibular lesions: With acute vestibular lesions, attempts to perform gaze orientations to flashed targets towards the side of the lesion result in gaze overshoots [4]. In these cases the eye trajectory also changes. It should be mentioned that independent controllers cannot predict any change in the eye trajectory since the eye and head trajectories are assumed to be independent and pre-determined in that scheme. As a result, any vestibular lesions should leave the eye trajectory untouched. In those independent controller models that add

TABLE I. PARAMETER VALUES

Eye plant time constant	200 msec
Head plant time constant	300 msec
Canal time constant including velocity storage	15 sec
SP VOR gain	85%
Effective integrator SP time constant	1.53 sec (15.38 sec without a visual goal)
Effective FP time constant in the dark	75-100 msec (depending on saccade size)

a. SP – slow phase fixation mode, FP - fast phase or saccade

an inhibitory projection from their 'head velocity command' to 'saccade ocular burst neurons' ([10]) the prediction is decreased eye movements due to increased head velocity after the lesion. This is opposite to what is actually observed [4]. Again, our model predicts very well the gaze overshoot and increased contribution from the eye (Fig. 4). An inherent result of a shared gaze feedback scheme is the dependence of eye and head trajectories *on each other.*

Vestibular compensation during saccades: After saccades, it is accepted that passive head perturbations are compensated using the VOR. However, passive head perturbations during gaze saccades can also be compensated especially if they are in the direction of the gaze shift 'assisting perturbations' [2, 19]. Since the activity of PVP neurons during the saccadic part of gaze orientation is much reduced, this compensation cannot be the result of the VOR in its classical sense [3]. In our model the VO neurons are the key players for this compensation since they only encode the passive component of head movements [20]. Fig. 5-6 show simulated instances for this phenomenon. It should be mentioned that even if the perturbation is not perfectly compensated, the correct target is still acquired due to the shared gaze feedback structure [5]. Independent eye-head controllers can't replicate such compensation during saccades, because the vestibular signal is presumed deactivated.

In brief, some of the other unique features of the SMI model are:

- It is consistent with known physiology, e.g., plant models are used instead of the ideal integrators in other models, and neural centers are defined to validate it [10-11]. Fig. 7-8 show the ability of this model in replicating central responses seen on Bursters and PVP cells.
- Unlike independent controllers, it does not require different trajectory plans and decompositions for different sets of plant initial conditions [10]. Initial conditions are inherently taken care of by the loops and internal models without introducing more complexity.
- This model is successful in replicating numerous experiments, without changing central parameters [1].

V. CONCLUSION

This work suggests that models with accurate neural network topologies will have the capacity to imbed motor control signals for several platforms (one controller) and provide coordination in several levels, i.e., eyes, head, and body. It is also suggested that trajectory planning and gaze decomposition are not necessary and might result in additional computational complexities, e.g., the effects of initial conditions. We have shown that several tasks and responses can be integrated into the same model, here visual target acquisition and resistance to perturbations. Simple parametric noise in such interconnected controllers can provide for robust task execution despite variable plant contributions and explain gaze orientations with different dynamics in other species, e.g., cat and monkey.

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