Evidence suggesting individual ocular motor control of each eye (muscle)

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Introduction

Current models of the ocular motor system are usually presented in their most reduced form. Common functions are lumped into single elements or subsystems. Most models are unilateral in architecture and precise yoking is presumed. Although this simplifies the models, it does not accurately simulate the actual neuroanatomy and limits the models to simple, stereotyped responses. Unilateral, yoked control (UYC) models produce both positive and negative signals despite both the bilateral nature of brain stem organization and the positive-only nature of neuronal signals; neurons cannot fire negatively. UYC models have only one eye, since perfect yoking is assumed; they are essentially monocular models. Such models may fail to duplicate those properties of the actual system that are a function of the neuroanatomical interconnections. One of the basic tenets of control system theory is that the ultimate behavior of a system is more a function of its interconnections (feedback loops) than of the gains of particular elements.

In cases where the bilateral nature of ocular motor control was under consideration, bilateral, yoked control (BYC) models were used. BYC models also have only one eye for output since perfect yoking is assumed. All signals are positive on either side of the brain stem. BYC models utilized "push-pull" interconnections of the common neural integrators to allow integration of differential signals and preservation of common, tonic signals (Doslak et al., 1979).

Observations from both normal and abnormal eye movement data suggest a more complex ocular motor control architecture - *independent control of each eye*. Because of the inherrent bilateral nature of brain stem organization, the latter directly implies independent control of each eye *muscle*. Taken individually, some of the observations are only mildly suggestive of independent control; others are strongly suggestive. None may, by itself, provide conclusive proof of independent control, although some appear to. However, taken together, they support a strong case for the hypothesis that the neuroanatomy of our ocular motor systems is basically configured for independent control of each eye (muscle) and it is binocularity that imposes the yoking normally seen.

Observations and Discussion

Studies of normal humans and monkeys have tended to reinforce the concept of tight yoking in binocular animals. However, some studies have demonstrated striking disconjugacies in normal responses. Normal saccades may be disconjugate (requiring glissades) or one eye may exhibit a dynamic overshoot (Weber and Daroff, 1971; Weber and Daroff, 1972). Asymmetric vergence can result in disconjugate saccades (Kenyon et al., 1980; Enright, 1986), unequal magnification spectacles cause differential saccadic gain adjustment (Erkelens et al., 1989; Lemij and Collewijn, 1991; Lemij and Collewijn, 1991; Kenyon et al., 1980; Zee et al., 1992) and saccades to unequal disparities also cause unequal saccades in the two eyes (Findlay and Harris, 1993). Enright concluded that the *step* of neural innervation was "generated seperately for each eye" and independently of the pulse driving a saccade (Enright, 1992). Zee et al. developed several models to simulate saccade-vergence interaction (Zee et al., 1992); two of them utilized independent burst neurons for each eye.

Furthermore, studies of abnormal eye movements have provided many examples of disconjugate eye movements. In strabismus, tropic eyes typically do not mimic the movements of the fixating eye nor do their latent or congenital nystagmus waveforms duplicate those of the fixating eye. In spasmus nutans, each eye oscillates independently

of the other (Weissman et al., 1987). In achiasmatic dogs, uniocular saccades and uniocular nystagmus waveforms are seen (Williams and Dell'Osso, 1993); the same may be true in human achiasma (Apkarian et al., 1993). These data from both normals and those with abnormalities suggest that current models for ocular motor control are inadequate representations of the actual system.

The inability of UYC (or even BYC) system models to duplicate the ocular motor responses of binocular mammals suggests that their ocular motor systems evolved from the bilateral, independent control (BIC) systems seen in chameleons. Each eye in a BIC model is driven by its own retinal input and, therefore, each muscle is driven by its own pulse generator-neural integrator combination. If mammalian ocular motor systems evolved from the chameleon, the same architecture should be preserved. Since binocularity imposes tight yoking, an overlay is needed to drive the independently controlled eyes in a conjugate manner. One way this could be accomplished is shown in Figure 1. This model contains bilateral, yoked, independent control (BYIC). Both eyes in the BYIC model are driven by a conjugate error signal derived from the errors seen by the individual eyes. The steering diodes indicate anatomical separation of retinal error signals in the two directions; all signals on both sides of the brain stem are positive. Models with the architecture of Figure 1 can produce the types of conjugate responses seen in most normals. The actual architecture of the yoking circuitry in binocular animals should allow the ocular motor system to function as either a BIC or BYIC system (or some combination of the two), depending on the type of input supplied. This architecture has yet to be worked out.

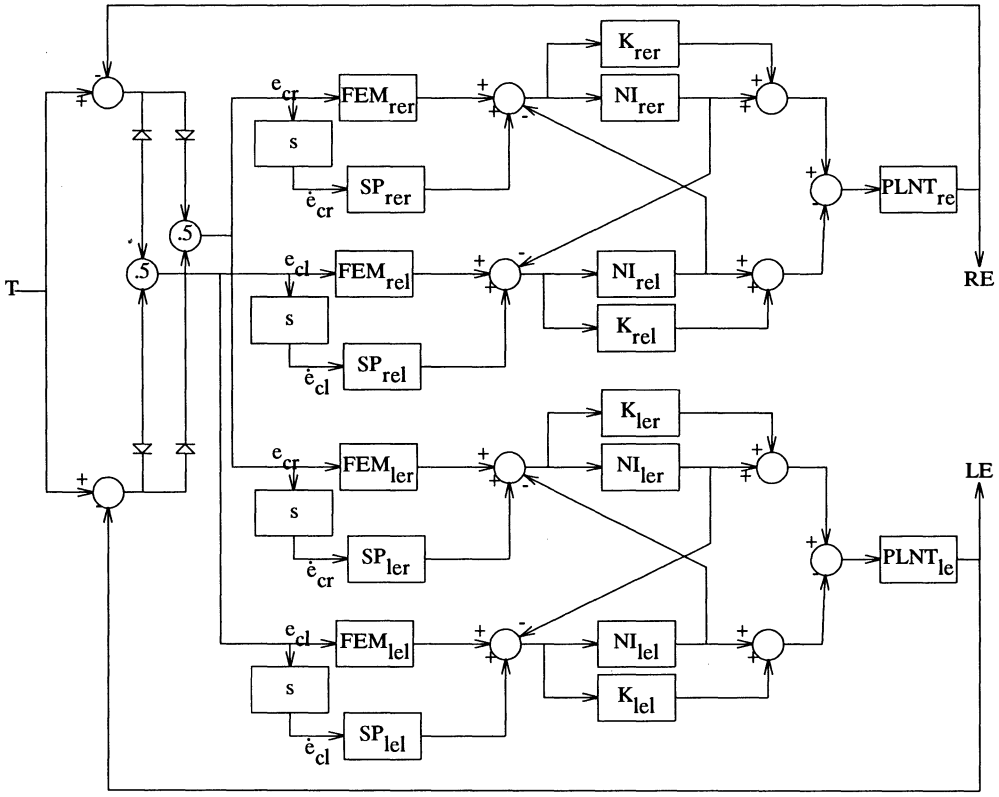

Figure 1. Illustration of bilateral, yoked, independent control architecture in a model containing both the (saccadic) fast eye movement (FEM) and smooth pursuit (SP) subsystems. T-target, c-conjugate, e-retinal error, s-differentiator, r-right, l-left, re/RE-right eye, le/LE-left eye, PLNT-plant, NI-neural integrator.

Finally, let us consider the question of where the yoking might be accomplished. Even in the chameleon, yoking is needed for the vestibuloocular reflex. A possible site for that yoking may be at the level of the interneurons of the VI Nerve. Since a conjugate response to head movement is needed, a yoking mechanism is required. Both the saccadic and smooth pursuit subsystems act on monocular retinal inputs allowing uniocular responses. In mammals, the yoking also controls saccades and smooth pursuit. Possible deficits in yoking that would exist in achiasmatic dogs and humans, human infants with spasmus nutans and humans (monkeys) with strabismus may exist at this site.

Before more specific neuronal connections can be hypothesized with confidence, anatomical data are required to answer several questions arising from the architecture in Figure 1. In the chameleon (and presumably higher mammals), is there a direct connection for saccades (i.e., burst neurons and common neural integrator neurons) and pursuit to the III Nerve or is the internuclear neuron pathway used? Are there connections between the motoneurons and internuclear neurons in the VI Nerve nucleus? The block diagram provided suggests a possible architecture from which working models of various subsystems can be made. The redundancy and specificity of function should allow demonstration of a wide range of behavior with minimal adjustment.

Abnormalities producing grossly disconjugate eye movements can be easily simulated using the independent control of each eye released by a deficiency in the yoking overlay. The implications of independent control of each eye coupled with the essential bilateral brain stem architecture are far-reaching. They suggest that each individual *muscle* is driven by independent populations of neurons (burst cells, neural integrator cells, etc.). The agonist muscles of each eye are usually coordinated (yoked) but may function independently if the task dictates or if binocularity did not develop.

Conclusions

A truly robust model of ocular motor control requires incorporation of both the bilateral nature of brain stem organization and the independent control needed to make disconjugate or uniocular eye movements. Such a model should be able to duplicate the many responses (normal and abnormal) possible from the neurophysiological system.

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