A Robust, Normal Ocular Motor System Model with Latent/Manifest Latent Nystagmus and Dual-Mode Fast Phases

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INTRODUCTION

Latent/manifest latent nystagmus (LMLN) is a specific type of infantile nystagmus that occurs subsequent to strabismus in some patients.[1,2] The amplitude of LMLN usually follows Alexander's law, increasing as the fixating eye moves into abduction and decreasing in adduction.[1] The slow phases of LMLN may be either linear or of decreasing velocity in the same patient.[3] Depending on the slow-phase velocity, LMLN fast phases may cause the target image to fall either within (foveating) or outside (defoveating) the foveal area.[3] Higher slow-phase velocities precipitate defoveating fast phases.[4] Also, as presaccadic slow-phase velocities grow, fast-phase amplitudes follow.

We present a dual-mode, ocular motor system model capable of producing normal saccades and both foveating and defoveating fast phases in LMLN. Additionally, the model contains a mechanism by which linear slow phases undergo the transition to decreasing velocity slow phases. The model includes programmable Alexander's law behavior and fixation conditions (binocular, right- and left-eye viewing), allowing simulation of the idiosyncratic characteristics of a broad spectrum of individuals with LMLN.[5]

MATERIALS AND METHODS

The model was built using the Simulink component of MATLAB (The MathWorks, Natick, MA) and is of modular design, consisting of subsystems thought to be required for accurate ocular motor control. The model also contains distributed delays that duplicate those known to exist from neurophysiological studies.

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The saccadic system responds to abrupt changes in target position and is capable of making short-latency (130-msec) corrective saccades, based on efference copy of position motor commands.

The internal monitor (IM) is essential for this model; the functions it performs have been required by all our past models of ocular motor function and *dysfunction*.[6–11] It uses afferent signals from the retina and efferent signals from the brainstem to accurately reconstruct target position and velocity and to distinguish them from eye position and velocity in the presence of motor instabilities. It calculates saccadic motor commands for voluntary and corrective saccades and for fast phases, and controls what portion of the saccadic pulse should be integrated. Provision is also made for Alexander's law variation of nystagmus slow phases.[9,11] Without such abilities, we contend that the ocular motor system could not function in the presence of either nystagmus or saccadic instabilities, let alone under normal circumstances.

To generate a *foveating* fast phase, the output of the neural integrator is compared with a desired eye-position signal, and the difference is compared to a position–signal error threshold. If this error exceeds the threshold, a saccade proportional to the error is generated. When the slow-phase velocity exceeds the velocity threshold (4°/sec), a *defoveating* fast phase is generated. The transition from foveating to defoveating saccades in the model is based on phase-plane data from LMLN subjects that show a significant difference in the presaccadic velocities for the foveating and defoveating cases. Some subjects have a region of overlapping slow-phase velocities where either foveating or defoveating fast phases can occur. This can be simulated by a change in the position-error threshold.

Previous studies correlated fast-phase magnitude with pre- and post-saccadic velocity.[4,12] The linear relationship between the magnitude and post-saccadic velocity suggested that an unintegrated, or "stepless," pulse was being employed. The postsaccadic velocities indicated that the pulse was not totally unintegrated, and the data suggested that the fast-phase generator produces a pulse width and height for a saccade of a relatively small size. To generate the decreasing velocity profiles of LMLN slow phases additional mechanisms were required: increasing the pulse to produce saccades greater than necessary to foveate the target leads to a larger unintegrated pulse, which is summed with the output from the neural integrator, producing a decreasing velocity slow phase.

RESULTS

The nystagmus of persons with MLN (both eyes open) contains linear slow phases and foveating fast phases throughout most gaze angles. When the model simulates a small gaze-angle Alexander's law effect, even though slow–phase velocity increases as the fixating eye abducts, the fast phases remain foveating until the eye is far into abduction. With a larger gaze-angle effect, slow-phase velocity increases more rapidly as the eye moves into abduction. When the velocity exceeds 4°/sec, the fast phases become larger and defoveating, and the slow phases exhibit a decreasing velocity profile. Importantly, neither type of MLN interferes with the accuracy of the saccadic subsystem, which can still make corrective saccades as necessary.

The nystagmus of individuals with LN (one eye occluded) contains decreasing velocity slow phases and defoveating fast phases throughout most gaze angles.

When a small gaze-angle Alexander's law effect is simulated, even though slow-phase velocity decreases as the fixating eye adducts, the fast phases remain defoveating except in extreme adduction. With a larger gaze-angle effect, the slow-phase velocity decreases more rapidly as the fixating eye adducts. As a result, the slow phases drop below 4°/sec at a more central gaze angle, causing smaller foveating fast phases and linear slow phases. As before, normal saccadic behavior is preserved.

CONCLUSIONS

We constructed a computer model of the normal ocular motor system that can also simulate LMLN. We demonstrated that an internal monitor could make use of afferent retinal and efferent motor information to detect changes in target position and to accurately differentiate target position and velocity from internally generated eye position and velocity (e.g., resulting from LMLN). In addition, we demonstrated that when slow-phase velocity exceeded 4°/sec, the foveating fast phases became defoveating, and the resulting slow phases decreased in velocity due to unintegrated portions of the fast-phase pulses. Finally, we demonstrated that if slow-phase velocity increased as gaze was directed in the abducting direction of the fixating eye (due to Alexander's law), that would ultimately cause the switch from foveating to defoveating fast phases.

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