A Robust, Normal Ocular Motor System Model with Congenital Nystagmus (CN) Including Braking and Foveating Saccades

L.F. Dell'Osso[1-3] and J.B. Jacobs[1,3]

Ocular Motor Neurophysiology Lab, VAMC[1] and Departments of Neurology[2], and Biomedical Engineering[3], Case Western Reserve University, Cleveland, Ohio, U.S.A.

Summary

The pendular waveforms of CN are quite complex, composed of a sustained sinusoidal oscillation punctuated by foveating and/or braking saccades. Previously we demonstrated how these quick phases could be generated by the same mechanism as voluntary saccades.[1] We propose a computer model for CN based upon the normal ocular motor control system. The model accurately simulates CN-patient ocular motility data. It provides a hypothetical explanation for the conditions that result in sustained pendular oscillation and the rules for the saccadic components that shape this underlying oscillation into the well-known family of pendular CN waveforms: pendular, pseudopendular, pendular with foveating saccades, and pseudopendular with foveating saccades. As is the case for normal physiological saccades, position error determines the saccadic amplitudes of foveating saccades, whereas braking saccades are stereotypical, velocity-driven, and their amplitudes not dependent on visual information.

Introduction

Congenital nystagmus (CN) is an involuntary oscillation of the eyes towards and away from the point of fixation. CN waveforms can be either pendular or jerk, and the slow-phase portion of the latter tends to take the form of an increasing-velocity (or "runaway") exponential, though

approximately linear slow phases can be found in some uncommon waveforms.[2]

Braking saccades are small, automatic (i.e. non-visually triggered), stereotyped fast phases that appear in some CN waveforms (pseudopendular (PP), pseudopendular with foveating saccades (PP_{fs}), pseudocycloid (PC), pseudojerk (PJ), bidirectional jerk (BDJ), and triangular (T)).[2] Braking saccades act to oppose the runaway slow phase, slowing the eye, and in some cases reversing its direction.[3, 4] These saccades are triggered by velocity efference signals; when the eye's velocity exceeds some critical velocity, (e.g., 4°/sec—the retinal slip velocity leading to decreased visual acuity), there is cause to attempt to arrest the eye's runaway.

It is our hypothesis that complex CN waveforms are actually created by a simple oscillation of the smooth pursuit system and shaped by the interposition of braking and foveating saccades. There is evidence that CN may start, as seen in early infancy in some individuals,[5, 6] as almost purely slow-phase movements, and as the ocular motor system develops during the first months of life, saccades begin to appear leading to the establishment of familiar CN waveforms.

Materials and Methods

The model was designed and implemented using the Simulink component of MATLAB, a control systems simulation package capable of performing simulations in both continuous and discrete time. The model is of modular, hierarchical design, consisting of functional building blocks thought to be required for accurate ocular motor control. The model uses distributed delays duplicating those known to exist from neurophysiological studies.

At the simplest level, our model is in essence a smooth pursuit (SP) system and a saccadic system whose behaviors are coordinated by an "Internal Monitor" (IM), that receives inputs from both subsystems (position and velocity efference copy) and sends control signals back to them. Because eye-movement data have shown that CN patients are not so different from normals, our SP and saccadic subsystems are capable of normal behavior, as well as being able to simulate many common clinical abnormalities.

SMOOTH PURSUIT SYSTEM: We selected the Robinson[7] model because of its simplicity of design that nonetheless yields realistic results. A defining feature of Robinson's model is the damped oscillation ("ringing") that occurs with the onset of pursuit. The source of this ringing is in the pre-motor circuitry sub-block. The frequency of the oscillation depends on the length of a delay in the feedback branch of this block. To induce the model into *sustained* oscillation, the gain must

be raised above its original value of 1.1. To use this SP system in an eye-position-based model, the plant was changed from a single pole to a double pole. This was necessary to achieve more realistic eye trajectories when combined with the saccadic portion of the model.

SACCADIC SYSTEM: The heart of the pulse generator is based on a local resettable neural integrator (RNI)[8, 9] with pulse height and width non-linearities. Saccadic pulses output through the common neural integrator and ocular motor neuron to the two-pole plant. The RNI is part of the circuit that determines saccade duration; when the RNI resets, the saccade ends. Saccadic durations are based on a combination of published physiological data[10, 11] and personal observation. Pulse amplitude was determined by an adaptive algorithm that varied the magnitude of the pulse until the peak amplitude of the eye matched the intended target position. An exponential function was then fit to these magnitudes.

INTERNAL MONITOR: The Internal Monitor (IM) is the "brains" of this model, performing all the logic and computation necessary to insure proper smooth pursuit velocities, saccades and neural integrator control, among other functions. The IM has a long history in ocular motor models.[12-16] The IM makes use of visual signals from the retina, as well as position and velocity efference signals available in the brainstem. Using this information, delayed appropriately, it is possible to reconstruct target position and velocity independent of any confounding "noise," e.g., CN or latent/manifest latent nystagmus (LMLN). The model can then respond appropriately to target changes, providing proper commands to the SP and saccadic systems.

Results

Because we propose that the ocular motor system in CN is essentially normal, it is necessary to demonstrate that the model can perform behaviors seen in normals and some commonly seen basic pathologies, such as saccadic dysmetrias, gaze-evoked nystagmus (GEN), muscle-paretic nystagmus of myasthenia gravis (MG) and LMLN. Furthermore, the presence of the internal oscillations should not interfere with the goal-directed operation of the system.

SACCADES: Saccades from <1° to approximately 17° are accurately executed in one movement. Larger saccades show characteristic hypometria followed by a short-interval (130 msec) corrective saccade. The model correctly responds to target-position changes occurring at any time.

PURSUIT: The model is capable of accurately pursuing ramps ranging from very low to moderately high velocities. There is an initial pursuit latency of 125 ms after which the eye, off-target, matches target

velocity; at 225 ms a catch-up saccade puts the eye onto the target. Since the overall SP gain is 0.95, there are several more catch-up saccades made over the duration of the stimulus.

COMBINED SACCADES & PURSUIT: In addition to the above behaviors that demonstrate separate functioning of the SP and saccadic systems, the model is also generates accurate responses to combinations of step and ramp stimuli.

EVOLUTION OF CN WAVEFORMS: CN progresses from the simple, initial underlying pendular oscillation. Enabling only braking saccades, the model does not attempt to achieve foveation and the PP oscillation remains symmetric around the point of fixation. Enabling only foveating saccades yields the pendular with foveating saccades (P_{fs}) waveform. The foveating saccades make use of reconstructed eye position error to foveate the target, shifting the oscillation so it is no longer symmetric about the target. The periods following the foveating saccades can then make a useful contribution to visual acuity, as they are both within the fovea and of low enough retinal slip velocity. Finally, when braking and foveating saccades are both active, the result is the complex PP_{fs} waveform. An important behavior is the spontaneous reversal of foveating and braking saccade direction. This is known as *bias reversal*; it is commonly seen in CN and was not specifically designed into the model. Bias reversal is due to small variations in the timing of braking/foveating saccade generation.

Conclusions

We constructed a computer model of the normal ocular motor system that also simulates saccadic dysfunctions, GEN, MG, and LMLN. To it, we added the ability to simulate CN based on the interposition of braking and foveating saccades into an underlying pendular oscillation of the SP system.

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