

# Vision and neural control of movement

October, 1st

14H00-14H45



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## Modeling Oculopalatal Tremor

OPT develops some time after a lesion in the brain that interrupts inhibition of the IO by the deep cerebellar nuclei (DCN). Over time the IO gradually becomes hypertrophic, and its neurons enlarge and develop abnormal soma-somatic gap junctions. However, results from several experimental studies have confounded the issue, because they seem inconsistent with a role for the IO in OPT, or because they ascribe the tremor to other brain areas. Here we look at 3D binocular eye movements in 15 OPT patients, and compare their behavior to the output of our recent mathematical model of OPT. This model has two mechanisms that interact to create OPT: an oscillator in the IO, and a modulator in the cerebellum. Here we show that this dual mechanism model can reproduce the basic features of OPT, and plausibly refute the confounding experimental results.

Oscillations in all patients and simulations were aperiodic, with a complicated frequency spectrum showing dominant components from 1 - 3 Hz. The model's synchronized IO output is too small to induce noticeable ocular oscillations, requiring amplification by the cerebellar cortex. Simulations show that reducing the influence of the cerebellar cortex on the oculomotor pathway reduces the amplitude of ocular tremor, makes it more periodic and pulse-like, but leaves its frequency unchanged. Reducing the coupling among cells in the IO decreases the oscillation's amplitude until they stop (at  $\sim 20\%$  of full coupling strength), but does not change their frequency.

The dual-mechanism model accounts for many of the properties of OPT. Simulations suggest that drug therapies designed to reduce electrotonic coupling within the IO or reduce the disinhibition of the cerebellar cortex on the DCN could treat OPT. Preliminary clinical tests of several drugs are consistent with our hypothesis.

14H45-15H30



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### Causality attribution biases oculomotor responses

When viewing one object move after being struck by another, humans perceive that the action of the first object "caused" the motion of the second, not that the two events occurred independently. Although established as a perceptual and linguistic concept, it is not yet known whether the notion of causality exists as a fundamental, preattentive "Gestalt" that can influence predictive motor processes. Therefore, eye movements of human observers were measured while viewing a display in which a launcher impacted a tool to trigger the motion of a second "reaction" target. The reaction target could move either in the direction predicted by transfer of momentum after the collision ("causal") or in a different direction ("noncausal"), with equal probability. Control trials were also performed with identical target motion, either with a 100 ms time delay between the collision and reactive motion, or without the interposed tool. Subjects made significantly more predictive movements (smooth pursuit and saccades) in the causal direction during standard trials, and smooth pursuit latencies were also shorter overall. These trends were reduced or absent in control trials. In addition, pursuit latencies in the noncausal direction were longer during standard trials than during control trials. The results show that causal context has a strong influence on predictive movements.

15H30-16H00

Coffee break

# 16H00-16H45



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## The influence of cues and stimulus history on the ocular pursuit response to randomized target motion

In responses to pseudo-random target motion, smooth pursuit gain and phase change with the range of frequencies comprising the stimulus. We provide evidence that this effect may be related to combined effects of stimulus timing history and target deceleration cues. Human subjects pursued pseudo-random stimuli composed of concatenated, alternating (left/right) segments of variable duration (RD). Segments were (a) ramps: velocity  $\pm 15$  deg/s or (b) half-cycle sinusoids: peak velocity  $\pm 23.5$  deg/s. RDs were randomized within 4 ranges (300-720 ms; 420-840 ms; 600-1020 ms; 840-1200 ms), each range comprising 8 RDs. Despite randomisation of RD and absence of deceleration cues in concatenated ramps, anticipatory reversals occurred prior to each direction change. Averaging of responses with identical RD within each range showed that eye velocity started to decline in anticipation of target reversal at a constant time from the start of the ramp, irrespective of actual ramp duration. However, the anticipatory decline occurred progressively later as mean RD of the range increased. Regression analysis suggested that timing of successive anticipatory reversals was probably derived from a running, weighted average of prior stimulus reversals. In concatenated sine responses, where deceleration cues were present, timing of reversal was proportional to RD for the longest range stimuli (840-1200 ms) but, surprisingly, was almost constant for the shortest range (300-720 ms). Thus, it appeared that deceleration cues could be used to predict direction-changes during long duration stimuli, whereas during short duration (i.e. high frequency) reversals there was insufficient time to respond to such cues and the system resorted to the use of timing history to predict turnaround. Frequency analysis of both sine and ramp responses suggested that combined effects of deceleration cues and timing history may explain the non-linear gain and phase characteristics of pseudo-random pursuit.

16H45-17H30



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## Multi-sensory integration across reference frames

Much of the sensory information available to the brain is redundant. Redundant information gets combined to reduce errors and enhance performance. This is done by weighting sensory input with respect to how reliable it is. However, these sensory signals are coded in very different ways and relative to different frames of reference. It has been hypothesized that in order to compare or combine different signals, they first need to get transformed into a common reference frame. In addition, there is evidence that any transformation that occurs in the brain has a cost, which is reflected in added noise to the transformed signals. Since the reference frame transformations themselves rely on noisy signals, this transformation cost should be signal-dependent.

Here, we set out to investigate if reference frame transformations add signal-dependent noise and if so, how such noisy transformations affect multi-sensory weighting. We show that both perception and action are affected by signal-dependent noise in a way that can be understood by stochastic reference frame transformations. As a result, transformed signals are less reliable, which results in lower multi-sensory weights. The bigger the transformation that has to occur, the less the transformed signal was weighted, which validates the above hypotheses. We believe that this is evidence that the brain has online knowledge of the sensory and transformation statistics and uses this information in an optimal fashion.