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SMOOTH PURSUIT WITH SAMPLED VISUAL INPUT: COMPARISON TO CONGENITAL NYSTAGMUS*

INTRODUCTION

The evaluation of the ability to pursue a moving target with one's eyes (smooth pursuit) requires that both the target and eye velocities be measured. The quotient of eye velocity over target velocity is defined as the smooth-pursuit «gain» and is the best measure of the performance of the smooth pursuit subsystem. This is a relatively simple procedure to carry out in a laboratory equipped with the proper eye-movement measuring equipment and usually poses no problems when studying a normal human or animal. However, when the functioning of the smooth pursuit subsystem is evaluated in a subject with ocular motor abnormalities, the method is subject to error if not applied properly. To understand both the sources of error and methods of avoiding them, a careful review of the definition of gain is necessary.

THE DEFINITION OF «GAIN»

First, let us consider a simple representation of the ocular motor system with only one input and one output (see Figure 1a). Here, the definition of smooth-pursuit gain is the same as that given above.

$$G_{sp} = \dot{\Theta}_E / \dot{\Theta}_T. \qquad \text{(Eq. 1)}$$

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If the only eye movements produced are entirely in response to the target velocity (referred to as «causality»), Eq. 1 will yield the gain of the smooth pursuit subsystem. This restriction excludes any other eye movements, such as: saccades to correct position errors; compensatory, vestibuloocular reflex (VOR) movements due to head acceleration; or optokinetic reflex (OKR) movements due to a moving background. In the lab, these sources of confounding eye movements can be controlled. When the data contain saccades, they can easily be excluded *before* computing gain; vestibular and optokinetic movements are not easily separated from pursuit and care must be taken to prevent their intrusion by stabilizing both the subject's head and the visual background. To describe these considerations more formally, Figure 1b shows a system with multiple inputs. For this more realistic representation of the ocular motor system,

$$G_{sp} = \dot{\Theta}_E / \dot{\Theta}_T = \dot{\Theta}_{E_{sp}} / \dot{\Theta}_T \quad \text{only when,} \quad \dot{\Theta}_{E_{sac}} = \dot{\Theta}_{E_{vor}} = \dot{\Theta}_{E_{okr}} = 0. \qquad \text{(Eq. 2)}$$

For a normal subject, we need only ensure that the respective inputs to the other ocular motor subsystems (Θ_T, $\ddot{\Theta}_H$, and $\dot{\Theta}_B$) are maintained at zero to ensure that the measured output velocity is causally related to the input target velocity and the calculation of smooth pursuit gain given by Eq. 2 will be correct. The gain of any system is only well-defined when the measured output is solely produced by the input to which it is being related in the calculation (i.e., causality is preserved).

Now let us consider what happens when, despite keeping all other inputs at zero, a given ocular motor system produces an output unrelated to the pursuit input. In the first (trivial) case, with target velocity equal to zero but eye velocity not zero, the gain function is undefined; we cannot divide the measured output eye velocity by zero. When we apply an input target velocity to this system, we can divide the measured output eye velocity by the target input velocity but the resulting «gain» (given by Eq. 2) cannot be interpreted as smooth-pursuit gain. The condition that the output be causally related to the input is not met. When, in the real world, does this become a problem? Consider the case (shown in Figure 1c) where the subject exhibits a spontaneous nystagmus ($\dot{\Theta}_{E_n}$) due to an internal nystagmus signal ($\dot{\Theta}_n$), either acquired or congenital. Regardless of the location of the source of this nystagmus, the resulting spontaneous eye movements are not causally related to a smooth-pursuit input. When a target is presented for that individual to pursue, the measured eye movement will still contain components due to the source of the nystagmus; the corresponding quotient of eye to target velocity cannot be equated to smooth-pursuit gain.

That is,

$$G_{sp} \neq \dot{\Theta}_E / \dot{\Theta}_T \quad \text{since,} \quad \dot{\Theta}_E = \dot{\Theta}_{E_{sp}} + \dot{\Theta}_{E_n}. \qquad \text{(Eq. 3a)}$$

Similarly,

$$G_{vor} \neq \dot{\Theta}_E / \dot{\Theta}_H \quad \text{since,} \quad \dot{\Theta}_E = \dot{\Theta}_{E_{vor}} + \dot{\Theta}_{E_n} \qquad \text{(Eq. 3b)}$$

and

$$G_{okr} \neq \dot{\Theta}_E / \dot{\Theta}_B \quad \text{since,} \quad \dot{\Theta}_E = \dot{\Theta}_{E_{okr}} + \dot{\Theta}_{E_n}. \qquad \text{(Eq. 3c)}$$

Unfortunately, there have been many instances where the simple quotient of eye velocity to target velocity, given by Eq. 3, has been misrepresented as the gain of either the smooth pursuit, VOR, or OKR subsystems of individuals with nystagmus. In all such cases, the requirement of causality between input and output was not met. The resulting «gain» calculations yielded meaningless numbers and conclusions reached based on such calculations could not be supported by the data.

Measurement of «gain» in the presence of nystagmus

How then can the function of the smooth pursuit (or VOR or OKR) subsystem be evaluated in individuals with spontaneous nystagmus? As Eq. 3 shows, even when the contributions from the other subsystems are eliminated, the measured eye velocity is *not* the causal output of the subsystem whose gain is being determined. In our studies of smooth pursuit and the VOR in an individual with congenital nystagmus (CN), we developed several *unrelated* methods that produced meaningful gain functions for both (7,8).

The *first* method depends on the existence of a foveation period in each cycle of the nystagmus waveform and was initially demonstrated graphically (5). During fixation of a stationary target, the eye velocity during CN foveation periods is very low and is usually zero for some time. If, during smooth pursuit, we form the quotient of the measured, non-zero eye velocity to target velocity *only* during these repetitive foveation periods, the resulting gain will be that of the smooth pursuit subsystem; eye velocity due to the CN is approximately zero. That is,

$$G_{sp} = \dot{\Theta}_E / \dot{\Theta}_T = \dot{\Theta}_{E_{sp}} / \dot{\Theta}_T \quad \text{where,} \quad \dot{\Theta}_{E_n} = 0. \qquad \text{(Eq. 4a)}$$

The values calculated for such «foveation-period gains» were normal and supported the hypothesis that the smooth pursuit subsystem functions normally in individuals with CN who have good foveation periods (4,6). We also applied this method to the VOR and found normal gains (7). For the VOR,

$$G_{vor} = \dot{\Theta}_E / \dot{\Theta}_H = \dot{\Theta}_{E_{vor}} / \dot{\Theta}_H \quad \text{where,} \quad \dot{\Theta}_{E_n} = 0. \qquad \text{(Eq. 4b)}$$

For the OKR,

$$G_{okr} = \dot{\Theta}_E / \dot{\Theta}_B = \dot{\Theta}_{E_{okr}} / \dot{\Theta}_B \quad \text{where,} \quad \dot{\Theta}_{E_n} = 0. \qquad \text{(Eq. 4c)}$$

In each of the above cases, knowledge of the portion of the CN waveform that corresponded to the foveation periods was necessary to choose that portion of the eye-movement record during which the gains could be calculated. A rough approximation to this method, resulting from simply averaging the eye signal and then calculating the gain, was shown to be very inaccurate (8).

The *second* method involves the use of phase planes, which are plots of position vs. velocity. We found that during fixation of a stationary target the phase plane of eye movement in CN contained portions of the trajectories (cusps) corresponding to the foveation periods that remained within a «foveation window» (9). This window was bounded by ±0.5° (the foveal radius) and ±4.0°/sec. If an individual with CN pursued a moving target perfectly, the phase plane of retinal-image error (eye motion – target motion) should be equivalent to that of fixation. This was found to be the case (8). Retinal-error phase planes also contained the cusps of foveation within the foveation window. Using this method to assess the VOR, gaze phase planes also contained cusps of foveation, indicating a normal VOR (7).

The *third* method we developed requires that the frequency spectrum of the spontaneous oscillation be separable from that of the input signal (pursuit, VOR or OKR) (7). For CN, where the nystagmus is usually 3 Hz or greater, gains can be calculated for the low-frequency inputs (usually less than 2 Hz) commonly used in assessing pursuit, VOR or OKR. Position signals were transformed with a fast Fourier transform (FFT) program after bias and trend had been digitally removed. Gain and phase were then computed from the cross- and autospectral densities of the FFT signals. A triangular (Bartlett) window was used to weight each frequency bin. The VOR gains calculated in this way were normal (7). Smooth pursuit or OKR gains can also be evaluated by this method.

Studies of individuals with CN whose foveation periods are roughly 50 msec or longer have shown normal pursuit (5,8,10) and VOR (7) gains. It has also been demonstrated that when there are no foveation periods (or, very short ones), one cannot assess the pursuit gain from the slope of the slow-eye-movement signal (5). How then, can one determine the smooth-pursuit gain in these individuals? Foveation-period gain cannot be calculated using Eq. 4 if there are no reasonably long foveation periods. Retinal-error phase planes might show some small cusps in the foveation window if there are short-but-measurable foveation periods. The method most applicable in these cases is the third, i.e., frequency separation of the CN and the input signal.

Implications of low «gain» in the presence of nystagmus

The final question I wish to address is, «Even if gain is properly measured in an individual with nystagmus, does a low gain necessarily imply that the subsystem in question (pursuit, VOR or OKR) is deficient?» Or, is it possible that the subsystem itself is normal but the measured low gain is due to a substandard visual input? To answer this question, we need to know how the normal pursuit system responds to diminished visual input. Fortunately, pursuit measurements have been made in normals whose visual input was degraded by providing tachistoscopally illuminated moving targets (1,3). The ability of the individual to accurately judge target speed was impaired to a degree dependent on the duration and interval of the flashed illumination. This resulted in diminished smooth-pursuit gains. When normals were presented with targets flashed for only 10 msec (much less than usual CN foveation-period durations) at intervals of 320 msec, their gains were approximately 0.85 and when flashed for pulse durations between 10 and 160 msec at an interval of 640 msec (approximately double that of a typical CN period), their gains varied from 0.5 to 0.8 respectively. Because this paradigm was performed on subjects with normal smooth pursuit, the low gains measured could *not* be attributed to deficient pursuit systems.

CN frequencies usually range between 2.5 and 6 Hz (corresponding to periods of 400 and 167 msec respectively) and foveation-period durations range between 10 or 20 msec to more than 400 msec; nominal values would be 3 Hz and 80 msec. If, to facilitate comparison between normals and individuals with CN, we equate pulse duration to foveation-period duration and pulse interval to CN period, the normal data for flashed moving targets suggest that an individual with nominal values for CN frequency and foveation-period duration should have a normal pursuit gain. We know that, due to waveform changes, the foveation periods of an individual with CN may decrease as gaze angle is changed. This can, in light of the above, result in diminished pursuit gain at different gaze angles. More importantly, different individuals with CN exhibit foveation periods whose durations may differ by substantial amounts. This also may be reflected in variations in smooth-pursuit gain despite the integrity of their smooth pursuit systems.

Concluding remarks

Diminished smooth-pursuit gains in normals result from lowering the durations of visual input. It also appears that the same may be true of the lowered durations of CN foveation periods. If these latter gains are the same as those of normals for equivalent tachistoscopic illumination, the most parsimonious conclusion is that the measured smooth pursuit system in individuals with CN

is normal (already demonstrated for CN with good foveation periods) but that pursuit gains may diminish as foveation-period durations diminish (as do the gains of normals). Although the studies that could provide a definitive answer have not yet been performed, this latter explanation is, in my opinion, more probable than the unsubstantiated claims of deficient or «reversed» pursuit. The latter has no data to support it; on the contrary, all published eye-movement records of smooth pursuit in individuals with CN and good foveation periods show accurate pursuit in the correct direction. Published eye-movement records from CN waveforms without foveation periods do not allow assessment of smooth-pursuit function, good or bad (5); claims to the contrary, notwithstanding.

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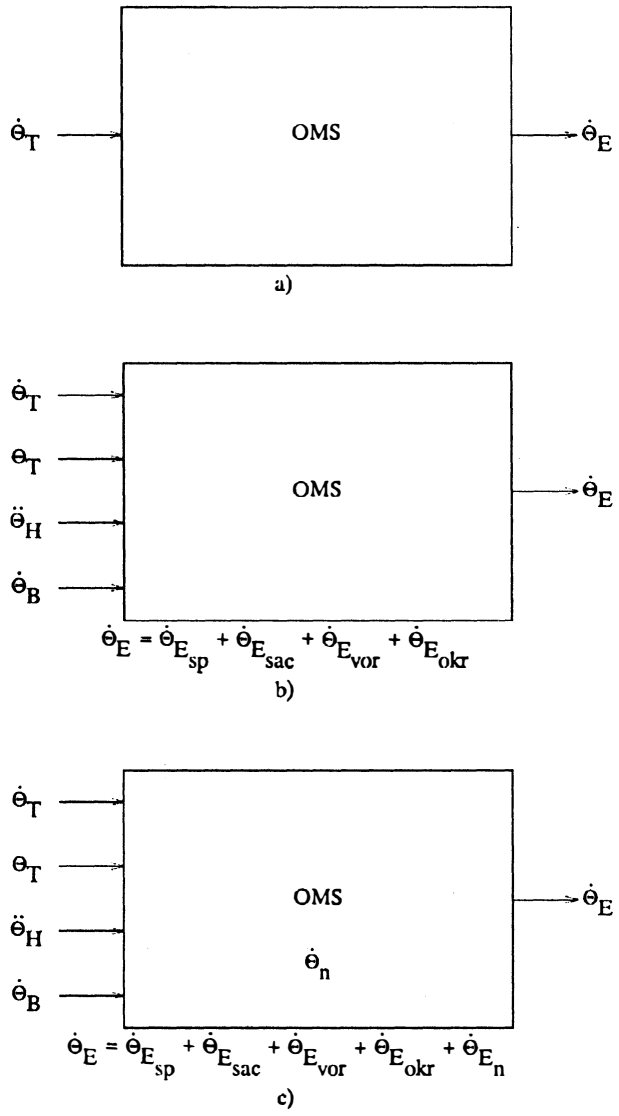

Fig. 1. Representations of the ocular motor system (OMS). A simple, single-input model is shown in a), a multiple-input model in b), and a multiple-input model with spontaneous nystagmus in c). Θ – position, $\dot{\Theta}$ – velocity, $\ddot{\Theta}$ – acceleration, T – target, H – head, B background, E – eye, sp – smooth pursuit, sac – saccade, vor – vestibulooocular reflex, okr optokinetic reflex, and n – nystagmus. Thus, $\dot{\Theta}_{E_n}$ is the eye velocity due to nystagmus.

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