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# Glissadic Overshoots Are Due to Pulse Width Errors

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• **Glissades are the slow, gliding eye movements often appended to the end of human saccadic eye movements. They have been used as an aid in diagnosing disease states, eg, multiple sclerosis and vascular lesions. Glissades are a consequence of a mismatch between the sizes of the pulse and step components of the pulse-step motoneuronal controller signals. This physiological and simulation study shows that glissadic overshoot is caused by pulse width errors and not by pulse height errors. This implies that the CNS can control the firing frequencies and recruitment of motoneurons more precisely than it can control the duration of the high-frequency motoneuronal saccadic burst.**

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Glissades are the slow, gliding movements sometimes appended to the end of normal human saccadic eye movements. Their frequency of occurrence in normal subjects is increased by fatigue.[1]

Several recent papers[2-5] have suggested that the existence of glissades could be used to aid in diagnosing internuclear ophthalmoplegia. In these patients, there is a glissadic undershooting of the adducting eye and a concommitant glissadic overshooting of the abducting eye. Sometimes there is abduction nystagmus. However, normal subjects can also exhibit these same eye tracking patterns.[6] So there is clinical interest in the etiology of glissades, as well as the evident physiological interest.

The cause of glissades has been the subject of previous speculation. Daroff and his group[7-9] named glissades and suggested that they were zero-latency, monocular corrective movements and products of a prenuclear feedback system. Later, it was suggested that a glissade was probably due to an error in the magnitude of the firing rate, comprising the pulse portion of the motoneuronal pulse-step controller signal.[10,11] This motoneuronal control signal consists of two portions: first, the phasic or pulse component, characterized by a high-frequency burst of motoneuronal activity for the agonist and an inhibition of motoneuronal activity for the antagonist; and secondly, the tonic or step component, the steady state neuronal firing rate. For normal-sized saccades, the duration of the pulse component is about one half the duration of the saccade.[12] This pulse component is responsible for the large forces that rapidly move the eye from one position to another. The step component holds the eye in its new position. Bahill et al[13] showed that glissades could be caused by errors in either the pulse or the step portions of the motoneuronal controller signal. This allowed a 13-fold way for saccadic-glissadic interactions (Fig 1). This present report investigates the neurophysiological origins of one of these 13 possibilities: glissadic overshoot due to a pulse error (type H, Fig 1).

## MATERIALS AND METHODS

Computer models are often used to study complex neurological systems because it is easier, cheaper, safer, and quicker to manipulate internal parameters of a model than to alter the internal parameters of a human or an animal. Furthermore, directions for new neurophysiological experiments can usually be suggested by the modeling results. The modeling data of this report were performed on the Reciprocal Innervation Model for human eye movements (Fig 2).[14] This model was justified qualitatively by comparing shapes of human and model saccadic and vergence eye movements, quantitatively by comparing the Main Sequence (peak velocity, magnitude, duration) relationships of human and model saccadic and vergence eye movements, analytically by doing a sensitivity analysis, and heuristically by simulating eye movements that the model was not designed to simulate.[14,15] The model has faithfully emulated all eye movements that we have attempted to simulate.[13,16,17] Therefore, confidence in this model is justified.

The photoelectric method of eye movement measurement[16] was used for gathering the human data. An important part of the data analysis was the definition of saccadic magnitude. For overshooting saccades, we defined saccadic magnitude as the angle transversed from the original starting position to the peak of the overshoot (from foot to peak). The instrumental bandwidth extended from 0 to at least 500 Hz. The system was linear for 20 degrees. However, computer processing and careful calibration allowed accurate quantification of larger saccades. Saccades as small as three minutes of arc have been recorded on this equipment.

The normalitive human data, small dots in the Main Sequence diagrams, and the experimental details for their collection have been previously reported.[13,16] They were collected from one normal subject. Data from seven other subjects were subsequently collected. This later data could be superimposed on the original data. No other subject was studied over such a large range and in such detail. The glas-

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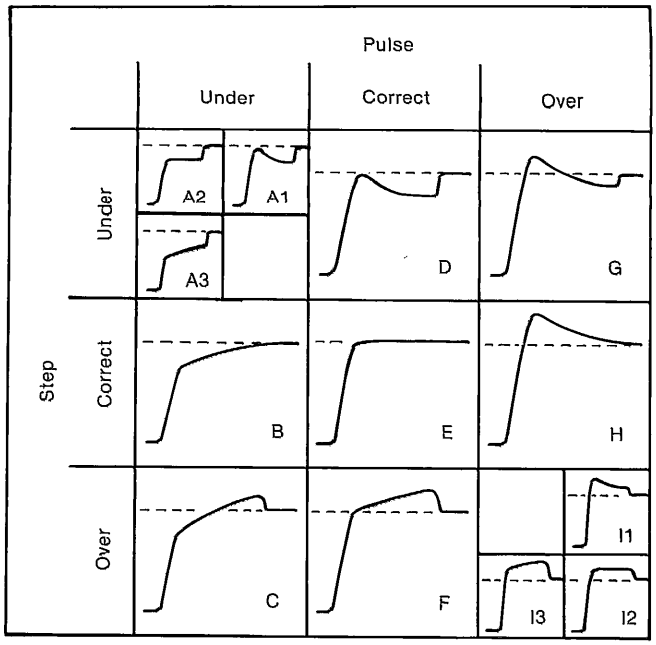


Fig 1.—Thirteen-fold way, showing all saccadic-glissadic interactions that are possible due to mismatches of pulse and step portions of motoneuronal controller signals. These illustrations were made to clearly demonstrate the glissades that are slow gliding movements after main large saccade. Naturally occurring glissades in unfatigued normal subjects are usually not this large.

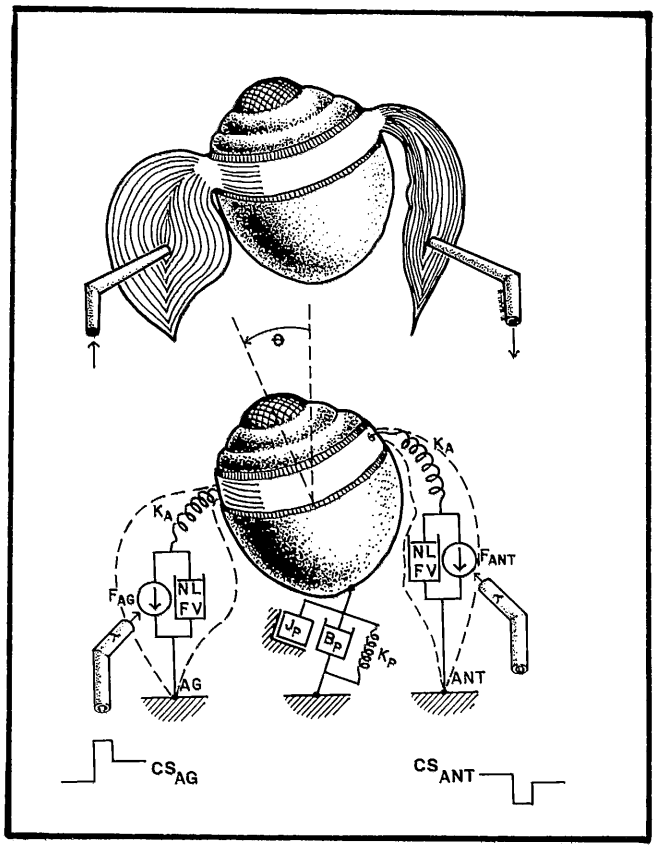


Fig 2.—Two reciprocal innervation models for human eye movement system—Descartes' 1626 model and our 1976 model. The top figure shows Descartes' basic concept of reciprocal action of extraocular muscles: Descartes thought that muscles were like balloons; when inflated, they would be short and fat, when deflated, they would be long and thin. Pipes, representing nerves, were used to pump fluid in and out of muscles. Shortening of agonist together with lengthening of antagonist produces eye movements. Bottom figure shows ideal mechanical elements used for modelling plant. Globe and surrounding tissues were modelled by effective inertia ($J_p$), a viscous element ($B_p$) and a passive elasticity ($K_p$). Each muscle was modelled by active state tension generator, $F_{AG}$ for the agonist and $F_{ANT}$ for the antagonist, nonlinear dashpot (NL-FV) representing nonlinear force-velocity relationship, series elasticity ($K_A$), and parallel elasticity that was combined with passive elasticity of globe to form ($K_p$). Active state generator converts oculomotoneuronal firing into force through first order activation-deactivation process. Controller signals ($CS_{AG}$ and $CS_{ANT}$) represent aggregate activity of all of motoneurons in agonist and antagonist motoneural pools.

sadic data are for two subjects: the original subject and one subsequent subject. Informed consent was obtained after the experiment and the equipment was explained to the subject.

## RESULTS

There are two obvious ways of making the pulse portion of the motoneuronal controller signal too large, thus mismatching the pulse and step components of the controller signal and producing type H glissadic overshoot: the pulse could be either too wide or too high. Both of these possibilities were tried in the model. The resulting mismatched saccades had similar qualitative shapes but different quantitative Main Sequence Measurements.

Figure 3 shows (with dotted lines) ideal 10-degree saccades, with no mismatch of the pulse and step components and (with solid lines) mismatched 12-degree saccades with 2-degree glissades appended. These mismatched saccades were produced by making the pulse portions of the agonist controller signals too large. For simplicity, the antagonist controller signals were not altered. The final eye positions were the same because the tonic firing rates of the motoneurons (the step portions of the controller signals) were the same. The mismatched saccade (solid lines) of Fig 3-PW was produced by increasing the pulse width of an ideal 10-degree saccade from 20 to 26 msec. This pulse width was the only parameter that was different in the controller signals for the 10- and 12-degree saccades of Fig 3-PW. The mismatched saccade (solid lines) of Fig 3-PH was produced by increasing the pulse height of an ideal 10-degree saccade from 87 to 120. The pulse height was the only parameter that was different in the two controller signals of Fig 3-PH. Thus, Fig 3 shows that eye movements that qualitatively look like human glissades could be created by making either the pulse width or the pulse height too large. However, quantitatively, these eye movements differed: the peak velocity of the pulse width mismatched saccade (Fig 3-PW) was smaller than the peak velocity of the pulse height mismatched saccade (Fig 3-PH), although they were both 12-degree saccades. In order to gain further insight into the neurological control of saccades, these mismatched

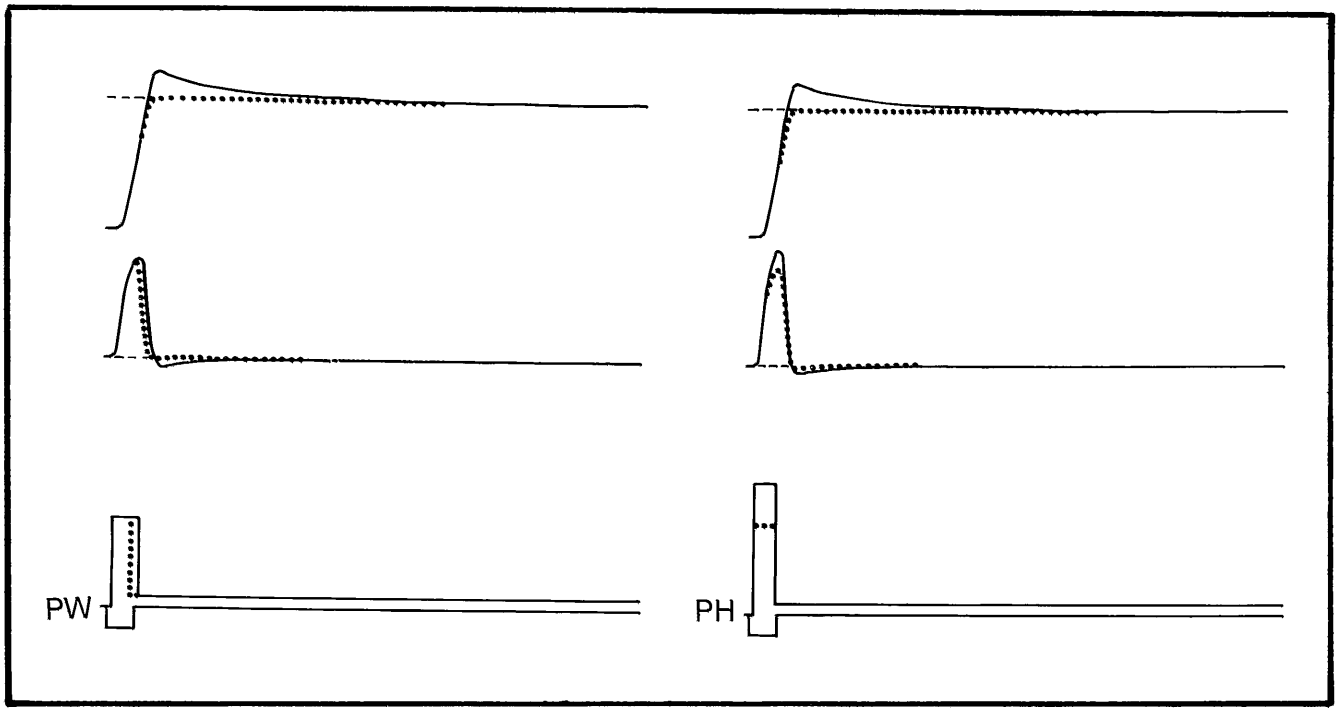


Fig 3.—Ideal 10-degree saccades (dotted lines) and mismatched 12-degree saccades, with 2-degree glissades appended (solid lines). Mismatched saccades were created by increasing only pulse width (PW) or only pulse height (PH) of agonist controller signals. Shown from top to bottom, as functions of time, are eye position, eye velocity, and the pulse-step motoneuronal controller signals that represent average firing rates of all motoneurons in agonist and antagonist motoneuronal pools. For simplicity, no time delay is shown between onset of motoneuronal activity and onset of movement, and corners of motoneuronal controller signals are not rounded by activation and deactivation time constants. Each record represents 500 msec.

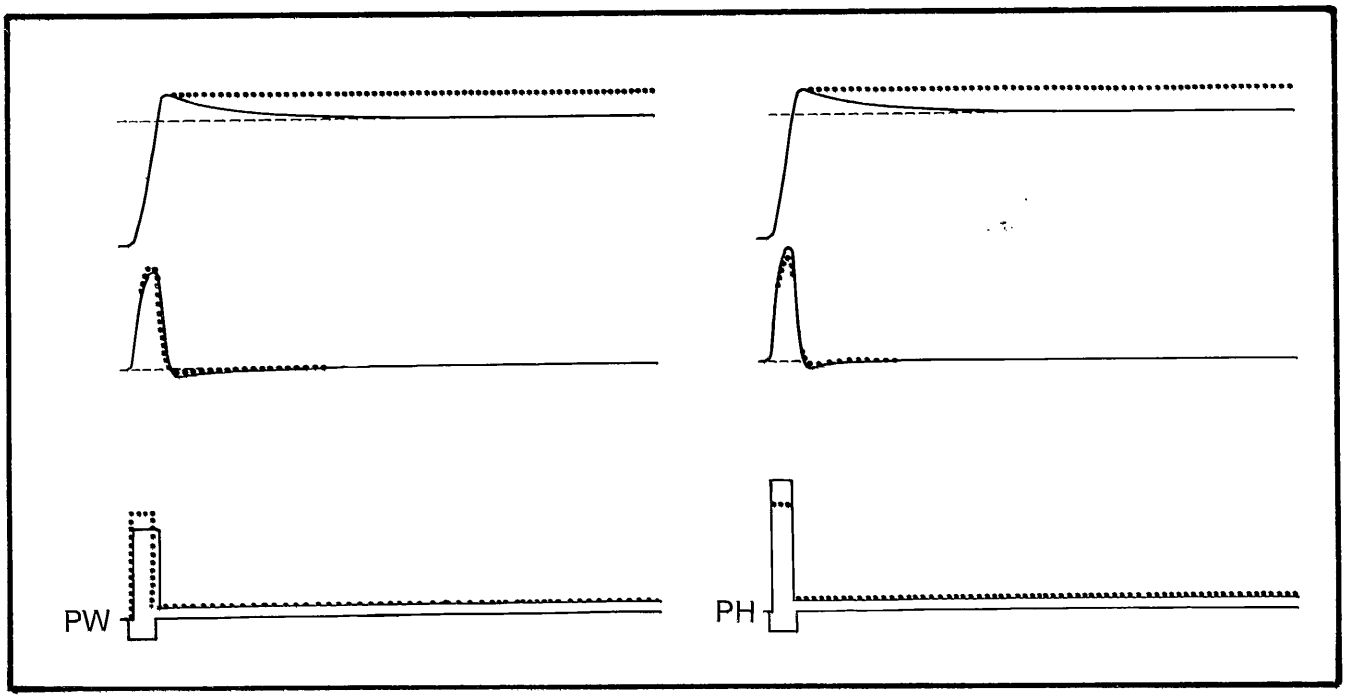


Fig 4.—Ideal 12-degree saccades (dotted lines) and same mismatched 12-degree saccades, with 2-degree glissades appended (solid lines). Velocity traces show that pulse width errors (PW) produce smaller saccadic peak velocities, while pulse height errors (PH) produce larger saccadic peak velocities.

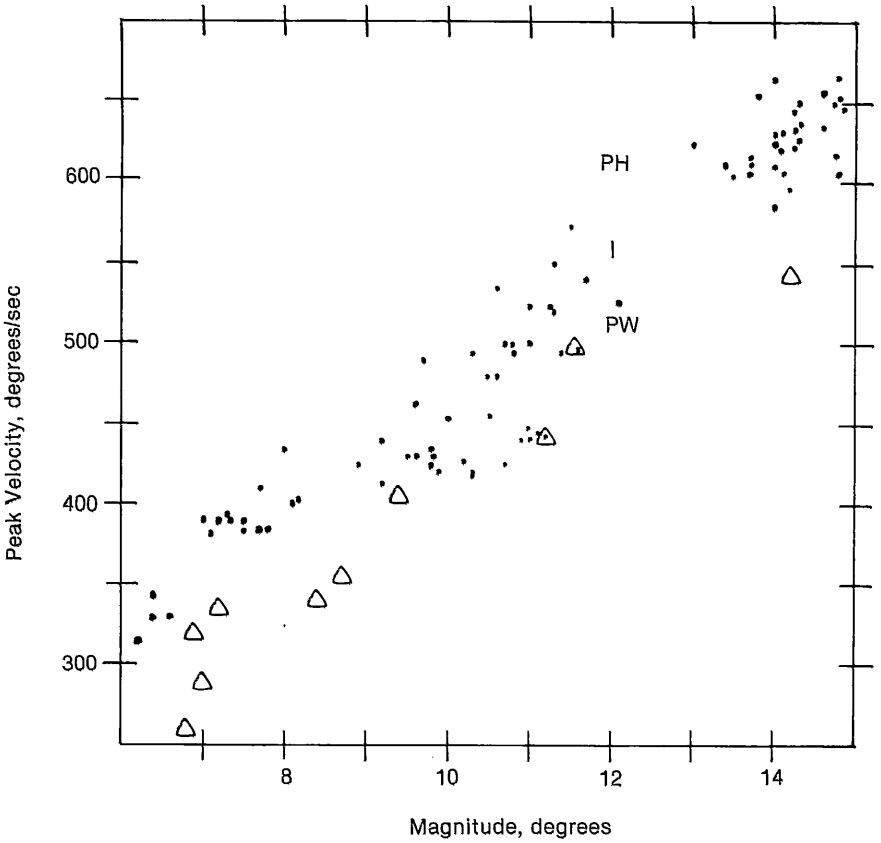


Fig 5.—Linear Main Sequence diagram showing that naturally occurring human saccades with glissadic overshoot (type H of Fig 1) have reduced saccadic peak velocities just as pulse width mismatched saccades from model have. Shown are normal human saccades (dots), human mismatched saccades (triangles), model pulse width mismatched saccades (PW), model pulse height mismatched saccades (PH), and model ideal 12-degree saccades (I).

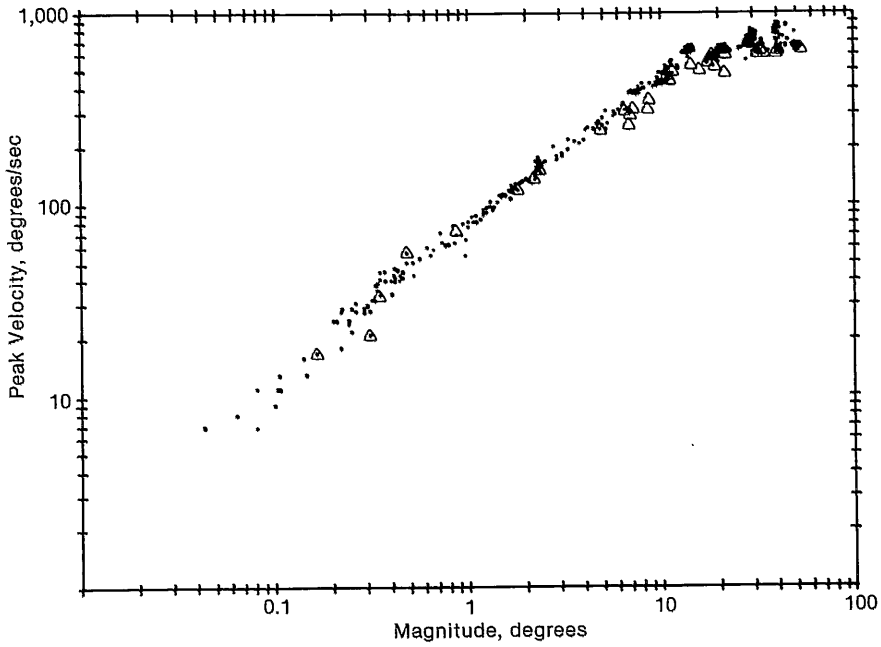


Fig 6.—Log-Log Main Sequence diagram showing that over range of 5 to 50 degrees, naturally occurring human saccades with glissadic overshoot (type H), shown with triangles, have reduced peak velocities.

12-degree saccades were next compared to ideal 12-degree saccades. Figure 4 demonstrates this comparison.

Figure 4 shows (with dotted lines) ideal 12-degree saccades, and (with solid lines) the same movements as the solid lines of Fig 3, ie, mismatched 12-degree saccades, with 2-degree glissades appended. The important traces in Fig 4 are the velocity traces. The peak velocity of the 12-degree pulse width mismatched saccade (Fig 4-PW) was 518 degrees/sec, which was smaller than the 560 degrees/sec peak velocity of an ideal 12-degree saccade. In contrast, the peak velocity of the 12-degree pulse height mismatched saccade (Fig 4-PH) was 612 degrees/sec, which was larger than the 560 degrees/sec peak velocity of an ideal 12-degree saccade.

These peak velocities are indicated with the symbols PW, PH, and I on the linear peak velocity Main Sequence diagram of Fig 5. Normal human saccades are indicated with dots. Therefore, actual human mismatched saccades with glissadic overshoot (type H) should have either unusually low peak velocities, if they were produced by pulse width errors, or else exceptionally high peak velocities, if they were produced by pulse height errors.

Human saccades with glissadic overshoot actually had unusually low peak velocities. Figure 5 shows normal human saccades, indicated with dots, and human mismatched saccades (type H) with glissadic overshoot due to oversized pulse components, indicated with triangles. The peak velocities of these mismatched saccades were smaller than the peak velocities of normal saccades, just as in the pulse width error mismatched saccades of the model.

This implies that in normal human eye movements with glissadic overshoot due to the pulse being too large, the error is caused primarily by the brain's mistake in computing the pulse width and not the pulse height. One implication of this is that normal human glissades are not caused by peripheral disturbances but are caused by the CNS circuits that produce the pulse width.

Figure 6 shows the more usual log-log Main Sequence diagram, with normal and mismatched (type H) human saccadic eye movements plotted. It confirms that in humans without disease, saccades with glissadic overshoot had smaller than normal peak velocities for saccades in the range of 5 to 50 degrees. For smaller saccades, this may not be true.

## COMMENT

Our theory that glissades are caused by pulse width errors has been demonstrated only for large saccades with glissadic overshoot (type H) in normal subjects. However, three cases have been reported where the patients may have used central adaptation to compensate for a peripheral deficit (a unilateral abducens nerve paralysis).[18] The resulting eye movements of these patients look like saccades with glissadic overshoot (type H). The findings of our report would imply that these patients had changed the pulse by making a pulse width adaptation (they also changed the step size). It is also probable that the characteristic eye movement patterns of patients with intranuclear ophthalmoplegia are manifestations of CNS pulse width adaptations compensating for pathological states.[19] Generalizing, the CNS control of duration is more flexible than the control of motoneuronal frequency or recruitment. This produces more errors of duration in normal subjects but also allows for adaptive control in pathological patients (perhaps via the cerebellum).

This prediction that pulse width and not pulse height errors are the cause of glissadic overshoot provides insight into the neurophysiological control circuits for saccadic eye movements. It implies that it may be more difficult for the CNS to accurately control the motoneuronal burst duration (pulse width), than the motoneuronal firing frequencies and number of motoneurons recruited (pulse height).

In the sensitivity analysis of the model,[14] it was also shown that errors in the pulse width and not the pulse height should be responsible for glissadic overshoot. This sensitivity analysis individually varied each of the model's internal parameters and recorded the effects on an ideal 10-degree saccade. It showed that increasing the pulse height from its nominal value produced increases in both saccadic amplitude and saccadic peak velocity. However, increases in the pulse width produced an increase in only saccadic amplitude. These are precisely the findings of this present report.

These reports have shown that glissadic overshoots (type H) could be due exclusively to pulse width errors but not exclusively to pulse height errors. The possibility that they could be due to a combination of pulse height and pulse width errors was found to be unlikely but was not ruled out by the data of these reports.

The sensitivity analysis also showed, however, that decreasing either the pulse width or the pulse height from their values for an ideal 10-degree saccade produced changes in both saccadic amplitude and peak velocity. This fact in conjunction with the data of Fig 6 implies that the suggestion that glissadic overshoots are caused exclusively by pulse width errors is valid only for saccades larger than about 5 degrees.

Fatigue increases the frequency of occurrence of glissades and also slows down saccades. So, it must be considered that some of the slowing of saccades seen in the data of Fig 6 could have been due to fatigue. We tried to minimize this possibility first by using data from periods when the subject was not exhibiting obvious signs of fatigue and second, by studying glissadic overshoot. It is primarily glissadic undershoot and not glissadic overshoot that becomes more prevalent in fatigued subjects.

In pathological patients also, mismatched saccades with glissadic undershoot (type B) are the most common type of saccadic-glissadic interaction. The obvious extension of this research is to evaluate the other 12 types of saccadic-glissadic interactions (particularly type B) and to show how they can explain the neurophysiological behavior observed in certain disease states.

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