

Dynamic and Static Violations of Hering's Law of Equal Innervation*

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ABSTRACT

Hering's Law of Equal Innervation treats the double eye as a single organ. Normal humans often execute saccadic eye movements that are dynamic violations of Hering's Law. These infractions are produced by differences in the neural controller signals sent to each eye and are exemplified by monocular movements, such as dynamic overshoot, glissades, and double saccades; these dynamic violations occur more frequently in fatigued subjects. In contrast to dynamic violations, static violations of Hering's Law are usually indicative of pathological conditions.

Hering's Law of Equal Innervation is an important clinical tool, which has been useful to optometrists, ophthalmologists, and physiologists for over a century. A simple statement of Hering's Law is that, during eye movements, corresponding muscles of each eye receive equal innervation.¹⁻³ Hering's Law is not an immutable law; it is often violated by normal humans. Most of these infractions are dynamic violations of Hering's Law, because they occur during saccadic eye movements.

This paper presents 2 categories of Hering's Law violations: static and dynamic. The static violations, which are rare in normals but common in patients, are usually indicative of pathology, while the dynamic violations (closely spaced saccades; overlapping saccades; low-velocity, long-duration saccades; dynamic overshoots; oblique saccades; and glissades) occur in normal humans. Hering's Law is a unifying

concept for explaining these variations in the hyperfine structure, the detailed shape information of saccadic eye movements. Some of these violations have been reported previously, but most are reported for the first time in this paper. The previous reports of these violations have treated them as isolated, enigmatic phenomena; we now have united them by their classification as Hering's Law violations.

METHODS

Eye movements were measured with the photoelectric technique similar to the one described by Bahill *et al.*⁴ The exceptions were that the infrared illumination came from light-emitting diodes mounted on a spectacle frame and that differentiation was usually performed with an RC circuit. Mounting the infrared light sources on the spectacle frame reduced the effects of head movements and allowed the use of a simple chin rest and head rest in place of a bite bar. The instrumental bandwidths were 500 and 70 Hz, respectively, for the eye position and the eye velocity records in Figs. 3 and 4.⁵ The bandwidths for eye position, eye velocity, and target position were 70, 25, and 10 Hz, respectively, in the other figures. The system was normally linear over a 15-degree range of eye movement. Since most naturally occurring human saccades are 15 degrees or less in magnitude,⁶ attempts were seldom made to measure larger eye movements. The clinician must possess some skill and experience with any method of measuring eye movement, so that artifactual results may be eliminated from the data. We were particularly fortunate that eye blink and head movement artifacts were easy to identify in our records and were therefore immediately eliminated from our data.

Vertical eye movements were measured in our Neuro-optometry Clinic with either the infrared photodiode method⁷ or with electro-oculography (EOG). One disadvantage of using the EOG technique was that the corneo-retinal potential varied long after illumination changes ceased.

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For instance, a step change in illumination could produce oscillations which doubled the EOG magnitude with a period of about 1 hr.⁸ Consequently, for the infrequent times when we used EOG, the patients were kept in the laboratory with fairly constant illumination for 1 hr or more before the measurements were taken. An alternate procedure was to keep the recording sessions very short and only use data where identical calibrations were recorded before and after each experimental run. All of the records of this report were derived from the infrared photodiode method of eye movement measurement.

RESULTS

Dynamic Violations of Hering's Law

When the eyes moved between 2 points, dynamic violations of Hering's Law were often recorded. The pathway taken by each eye was usually different. Since these violations were a consequence of the movements of the eyes, they were categorized as dynamic violations. We show 6 basic types of dynamic violations of Hering's Law in the saccades of normal subjects: closely spaced saccades; overlapping saccades; low-velocity, long-duration saccades; dynamic overshoots; oblique saccades; and glissades. These monocular movements appeared in the dominant and the nondominant eye, in leftward and rightward saccades, and in abducting and adducting saccades.

Hering's Law violations that we found included double saccades, 2 saccades with less than the classical 200-msec intersaccadic interval. They were either closely spaced saccades (Fig. 1) or overlapping saccades (Fig. 2). Because the velocity profiles for each saccade overlapped one another, they were called overlapping saccades. As can be seen from these figures, double saccades were monocular phenomena; they appeared in only 1 eye at a time. Fatigue^a increased the frequency of occurrence of double saccades; however, normal unfatigued subjects also, on occasion, executed double saccades.^{9, 10} Double saccades were not the result of instrumental artifacts, because a measurement artifact would not extend the duration of the movement but would merely insert a discontinuity into the records (see "Discussion"). The saccades of Figs. 1, 2, and 8 were executed by the same normal subject within a 3-min period. They show that double saccades occurred in either eye.

^aWe use the term "fatigue" in a very general manner, encompassing sensory adaptation, CNS habituation, and muscular fatigue.

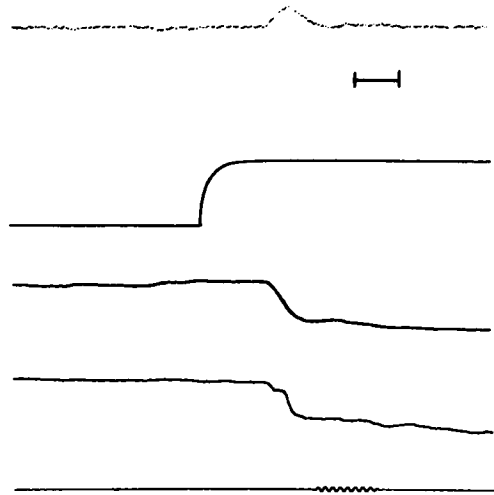


FIG. 1. Shown as functions of time, from top to bottom, are velocity of the left eye, target position, horizontal position of the left eye, horizontal position of the right eye, and the timing marker. The target movement was a 10-degree step displacement that took less than 1 msec for completion. The slowness of the target position record was due to a slow, low-bandwidth channel on the strip chart recorder; the eye movement channels had larger bandwidths (see "Methods"). Leftward movements are represented by upward deflections in the eye position records and downward deflections in the target position record. The timing trace has a 60-Hz burst each second. The calibration mark below the velocity trace represents 100 msec. All of the figures of this report have a similar format, unless otherwise noted. Hering's Law was violated during this eye movement; there were closely spaced saccades in the movement of the right eye and a low-velocity, long-duration saccade in the movement of the left eye. These are signs of fatigue in normal subjects.

Fatigue also caused slowing of individual saccades. This sometimes occurred in one eye before the other as shown in Figs. 1 and 2. In this experimental trial, the subject's saccades of the left eye became low-velocity, long-duration saccades after about 10 min, while her saccades of the right eye were still normal at the end of a half-hour. Thus, the third demonstrated violation of Hering's Law was the slowing down of individual saccades.

Dynamic overshoot resulted when an eye went beyond its final target position and then returned with a quick saccadic movement which lasted 15 to 20 msec and had a velocity of up to 200 degrees/sec (the duration and velocity of the return phase depended upon the magnitude of the return phase). Dynamic overshoot was often a monocular phenomenon. Fig. 3 shows saccades with the right eye dynamically

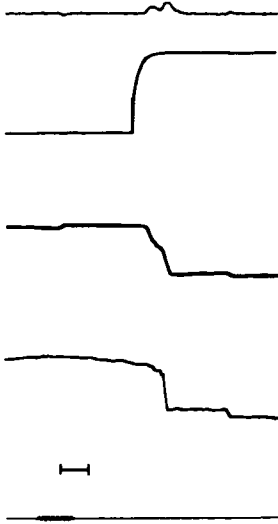


FIG. 2. Overlapping saccades in the movement of the left eye. The velocity profiles of the 2 saccades in the left eye seem to overlap. The left eye is slowed down due to the sluggishness of the saccades and to the presence of the double saccade. Therefore, the left eye starts 50 msec before and comes to rest 10 msec after the right eye. The target position trace was retouched to indicate the onset of target motion.

overshooting and the left eye not dynamically overshooting, but rather glissadically overshooting. The neurological control signals which produced these 2 types of saccades were quite different.^{4, 11, 12} In Fig. 3, the saccade with dynamic overshoot was slower than the saccade without it, because of the double saccade in the movement with dynamic overshoot. The effects of this double saccade can be seen best in the acceleration plots.

Saccadic eye movements were seldom linear or straight. Even saccades between 2 points on a vertical line (called purely vertical saccades) or between 2 points on a horizontal line seldom moved the eye in a straight line through XY space. Fig. 4 shows a typical, purely vertical saccadic eye movement. It had a small, transient, orthogonal, horizontal component. These horizontal components and, therefore, the trajectories of the eye positions in space varied from saccade to saccade. Using after-image techniques similar to those used by von Helmholtz,¹³ we have shown that the trajectories of the 2 eyes were usually different.

The glissades shown in Fig. 5 demonstrated the 6th and final example of dynamic violations of Hering's Law. Glissades¹⁴ were slow, drifting

movements that were often attached to the end of saccadic eye movements. Their velocities decayed exponentially to zero, and their maximum velocities were functions of the size of the glissades.¹⁵ The examples shown in Fig. 5 were typical. Glissades were 1 to 2 degrees in magnitude, had durations of 100 to 500 msec, and had velocities of about 5 degrees/sec. They were usually monocular and occurred more often in fatigued subjects.

The 6 types of dynamic violations of Hering's Law that have been presented are closely spaced saccades; overlapping saccades; low-velocity, long-duration saccades; dynamic overshoots; oblique saccades; and glissades. They were all associated with the hyperfine structure of saccadic eye movements and occurred in normal humans.



FIG. 3. Dynamic overshoot in the saccade of the right eye. Shown as functions of time, from top to bottom, are the position of the left eye, the position of the right eye, velocities of the 2 eyes, and accelerations of the 2 eyes. Leftward movements are represented by upward deflections. Each record is 500 msec in duration. The change in static eye position was 10.0 degrees. The left eye had a maximum velocity of 510 degrees/sec and a peak positive acceleration of 42,000 degrees/sec². These saccades were recorded directly on a computer disk memory and were plotted out in an XY plotter using a computer slow-down routine. The instrument bandwidth for the eye position records was 500 Hz. The high-frequency noise is so prominent in the records of the left eye is artificial and was not low-pass filtered out.

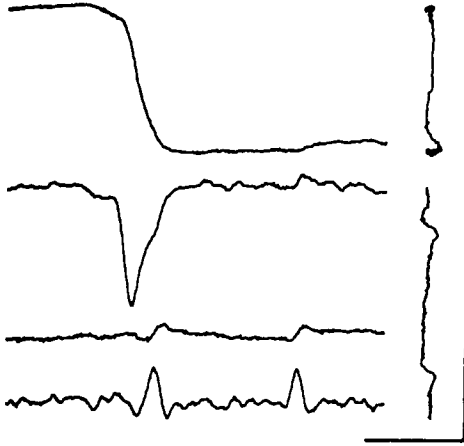


FIG. 4. Trajectory curvature illustrated with a downward saccade having a small, transient, horizontal component. The display shows in the left column, from top to bottom, vertical eye position, vertical eye velocity, horizontal eye position, and horizontal eye velocity; at the top of the right column is shown eye position in space, or the X-Y trajectory; the bottom of the right column is the horizontal position versus time record rotated on its side and aligned with the X-Y trajectory above. The calibrations shown represent 4 degrees, 150 degrees/sec, and 100 msec. Rightward (temporal) and upward eye movements are represented by the upward deflections. Although this is only a monocular recording, our psychophysical studies have shown that the trajectory curvatures are different for the 2 eyes.

Violation of Hering's Law Associated with Pathology

Certain neurological disease states are often characterized by eye movement syndromes involving glissades. For example, in dynamic internuclear ophthalmoplegia, also called the syndrome of the medial longitudinal fasciculus, the adducting eye falls short of its final target position and completes the movement via a glissade. Simultaneously, the abducting eye overshoots the target and glissades back to the final position. Sometimes there is abduction nystagmus.¹⁶⁻¹⁸ If the terms abducting and adducting are interchanged in the previous sentences, then abduction internuclear ophthalmoplegia is described.¹⁹ Fig. 6 shows eye movements similar to those reported in internuclear ophthalmoplegia. However, the saccades of Fig. 6 were executed by a clinic patient who lacked other confirmatory signs and symptoms needed to establish the clinical diagnosis of internuclear ophthalmoplegia and were therefore considered only as an unexplained pattern of adduction lag. We have found 3 (out of 40)

clinical patients showing these pseudo-internuclear ophthalmoplegia patterns, as well as 2 of this paper's authors who exhibited similar saccadic-glissadic patterns on particular days. Fig. 7 shows the eye movements of one of the authors (T. B.), illustrating abduction lag and adduction nystagmus. These records are remarkably similar to the records of a patient with abduction internuclear ophthalmoplegia.¹⁹ However, this author has neither before nor since exhibited pseudo-internuclear ophthalmoplegia patterns. Therefore, we caution against overinterpreting similar saccadic-glissadic patterns into a diagnosis of internuclear ophthalmoplegia.

Static Violations of Hering's Law

Sometimes in the middle of a recording session the eyes saccaded in the same direction but with different magnitudes, producing static violations of Hering's Law (Fig. 8). Three similar static violations were recorded during the same half-hour session. The causes of these random, unexplained, Hering's Law violations were difficult to determine. We could neither explain why a normal subject made these aberrant eye movements nor could we predict their occurrence. The eye movements shown in Fig. 8 include both dynamic and static violations. That is, not only are the dynamic saccadic movements different, but the static eye position levels are also different.

Fig. 9 shows the eye movements of a 69-year-old woman with nystagmus of recent onset. The frequency of her nystagmus was usually about 4 Hz, but the type of nystagmus varied: sometimes no nystagmus would be present, sometimes the nystagmus would be pendular, sometimes the nystagmus would be sawtooth, and sometimes it would violate Hering's Law (Fig. 9). In this record every other saccade was a violation of Hering's Law, since the saccades moved the eyes alternatively in the same and then in opposite directions. Since the trajectories and the static eye positions were disjunctive on alternate saccades, the movements of Fig. 9 clearly show both static and dynamic violations of Hering's Law.

DISCUSSION

Dynamic Violations and the Hyperfine Structure of Saccades

A pulse-step controller signal must be produced by the extraocular motoneurons in order to produce a saccadic eye movement. The pulse is the high-frequency burst of motoneuronal firing that moves the eye rapidly from one point

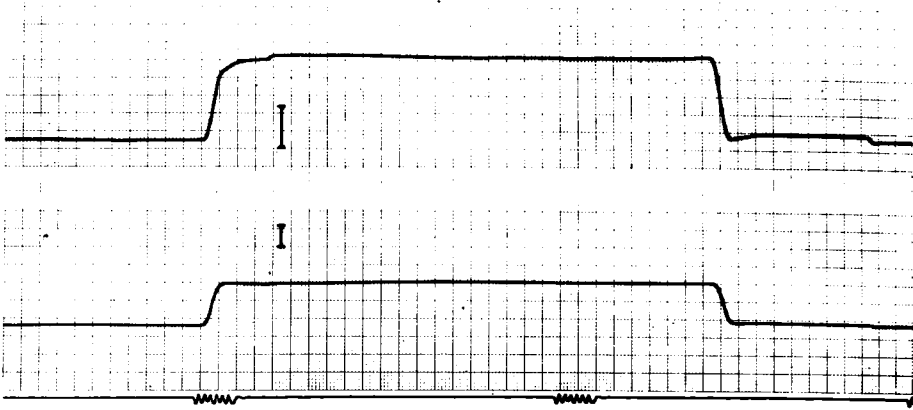


FIG. 5. Glissadic undershoot (first saccade) and glissadic overshoot (second saccade) exhibited in the saccades of the left eye (top trace) of a normal subject. The calibrations represent 10 degrees for each eye.

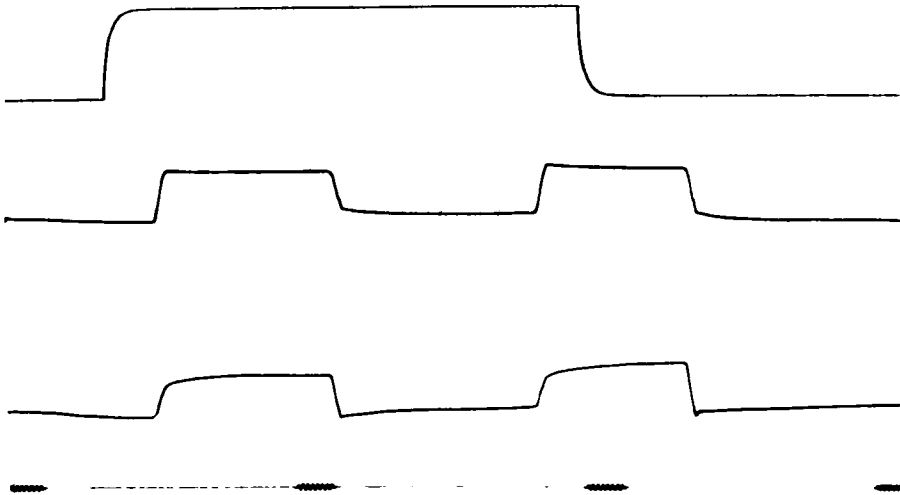


FIG. 6. Pseudo-internuclear ophthalmoplegia records showing glissadic undershoot of the adducting eye and simultaneous glissadic overshoot of the abducting eye, a syndrome often associated with internuclear ophthalmoplegia. Note that the eye movement patterns are the same for eye movements made: (1) after the target movement by a normal latency, (2) to a blank point on the screen in anticipation of target movement, and (3) back to the target again.

to another. The step is the tonic level of innervation that holds the eye in its new position. Variations in this pulse-step controller signal give rise to the hyperfine structure of saccadic eye movements and to the dynamic violations of Hering's Law shown in Figs. 1 to 3 and 5 to 7.

Often both eyes of fatigued subjects did not complete the transition from one target position to the next with 1 large, smooth saccade; one eye executed either 2 closely spaced saccades (Fig. 1) or 2 overlapping saccades (Fig. 2). As few as 30 saccades of 50-degree magnitude or 1000 saccades as small as 10 degrees were

usually sufficient to produce these signs of fatigue.¹⁰ Double saccades were not the result of instrumental artifacts, because in all cases in which we accepted the results as being double saccades we had recorded smooth single saccades both before and after the double saccades. Double saccades are a manifestation of an amplitude decomposition of the movement and not of a temporal decomposition. In other words, if a 10-degree saccade is decomposed into two 5-degree saccades, then two 5-degree pulse-step controller signals must be sent to the extraocular muscles. This will result in two 35-msec, 5-degree saccades for a total move-

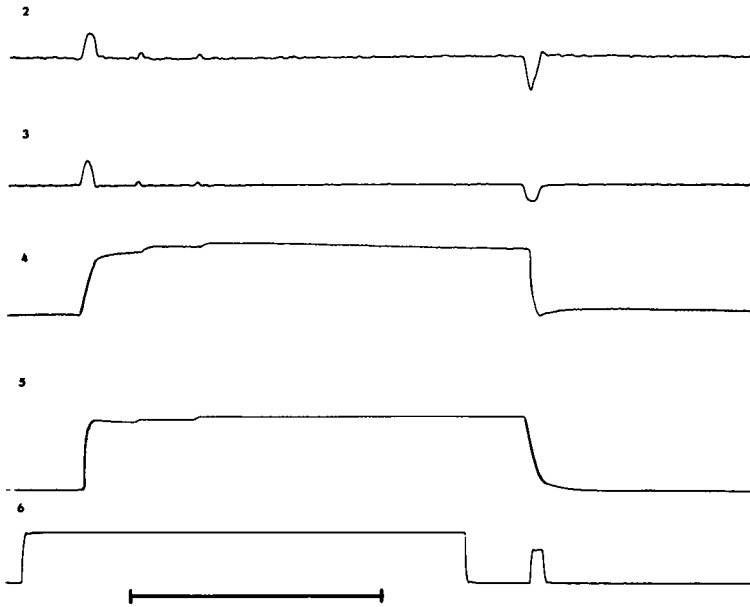


FIG. 7. Shown from top to bottom are left eye velocity, right eye velocity, horizontal position of the left eye, horizontal position of the right eye, and the target position. The target jumps are physiologically large: 20 degrees. The calibration mark represents 1 sec. The strip chart recorder used curvilinear paper. The final pulse shown in the target position record was ignored by the subject. Leftward movements are represented by upward deflections. Pseudo-abduction internuclear ophthalmoplegia records showing abductor glissadic undershoot, concomitant adductor glissadic overshoot, and adductor nystagmus in the right eye. Although this is similar to tracking patterns described for patients with abduction internuclear ophthalmoplegia, these eye movements were executed by a normal subject.



FIG. 8. Static violations of Hering's Law made by a normal subject.

ment duration of 70 msec. This is much longer than the 40-msec duration of a normal 10-degree saccade. Merely introducing a pause into the middle of the pulse of a 10-degree pulse-step controller signal will not produce the same long duration of movement that two 5-degree pulse-step controller signals will. This amplitude decomposition is discussed in greater detail by Bahill and Stark.¹⁰

Dynamic overshoot is produced by role reversals at the end of the pulse-step controller signal. That is, the primary saccade agonist has a pause of about 10 msec, and the primary saccade antagonist simultaneously has a brief

burst of motoneuronal firing.^{4, 11} This burst in the primary saccade antagonist and pause in the primary saccade agonist drive the eye back to the final position with a small saccade. The peak velocity versus magnitude and the duration versus magnitude relationships (Main Sequence diagrams)²⁰ for the return phase of the dynamic overshoot are the same as for other types of saccades.⁴

Glissades are produced by mismatches between the pulse and step components of the motoneuronal controller signals; these mismatches may be due to errors in either the pulse or the step components.¹⁵ As an example of a pulse error glissade, suppose the pulse component is too large, due to either having too wide a pulse, recruiting too many motoneurons, or firing the motoneurons at too high frequencies, then the pulse commands a larger eye movement than the step: the first saccadic portion of the movement drives the eye beyond its final target position, and the movement is completed with a glissade back to the final position. However, saccades with this type of glissadic overshoot have slightly lower peak velocities than equal sized saccades without glissades. This fact, in conjunction with modeling studies,

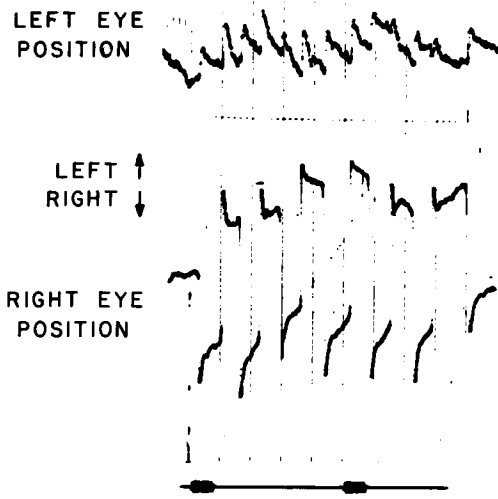


FIG. 9. Every other saccade made by this patient with nystagmus is a violation of Hering's Law. This sequence of saccades was recorded with only the left eye viewing a stationary fixation target. The gains of the 2 channels are not the same. The saccades are about 5 degrees in magnitude.

suggests that an error in pulse size which produces glissadic overshoot is probably the result of a pulse width error and not a pulse height error.²¹ This may help to unravel the underlying pathology which produces internuclear ophthalmoplegia. Small saccades have smaller peak velocities than do large saccades, a relationship called the Main Sequence. The adducting saccades with glissadic undershoot in patients with internuclear ophthalmoplegia are smaller than the simultaneous abducting saccades and, therefore, should have smaller peak velocities, as pointed out by Metz.¹⁸ It has not yet been shown whether saccades with glissadic undershoot have larger or smaller peak velocities than equal-sized saccades without glissades. This could either accentuate or minimize the results noted by Metz.

The adducting nystagmus of the right eye seen in the abduction pseudo-internuclear ophthalmoplegia records of Fig. 7 is not a true nystagmus, but merely the result of alternating glissades to the right and corrective saccades to the left. Large saccades in normals are usually hypometric: they have static undershoot that is ameliorated by a corrective saccade about 150 msec after the primordial saccade. For the first saccades shown in Fig. 7, each eye's step portion of the pulse-step controller signal codes for a change in eye position that is smaller than the change in target position, and this produces static undershoot in both eyes. The pulse signal for the abducting (left) eye codes for a saccade

that is even smaller than the change in eye position coded for by the step. This causes a leftward glissade: the glissade and the subsequent corrective saccade are in the same direction. The pulse signal for the adducting (right) eye codes for a saccade that is larger than the change in eye position coded for by the step, but smaller than the change in target position; this causes a rightward glissade, and the glissade and the subsequent corrective saccade are in opposite directions. This produces the nystagmoid pattern seen in the adducting (right) eye. The slow phases in this pseudo-nystagmus are exponential in shape, like all glissades.^{10, 15, 18} The peak velocity of each subsequent glissade becomes smaller, until the actual eye position matches the motoneuronally coded eye position.

The fact that the step portion of the motoneuronal controller signal is usually the same for both eyes, while the pulse portion usually differs, gives support to the differentiator hypothesis^{15, 22} for formation of the motoneuronal controller signal. This states that the input signal to the saccadic, time-optimal, pulse-step controller signal generator is the step, and that this step is differentiated and summed with itself to produce the composite pulse-step motoneuronal controller signal.

Dynamic violations of Hering's Law are produced by variations in the pulse portions of the pulse-step controller signal. These variations are not due to variations in peripheral nerves or muscles, because dynamic violations in normals are not affected by the lateral direction of the saccades. Furthermore, patients with pathology who exhibit dynamic violations usually have brain stem lesions rather than peripheral injury.

Previous Investigators Have Intimated the Occurrence of Dynamic Violations of Hering's Law.

In 1901 Dodge and Cline²³ stated that the durations of large (30 degree) rightward and leftward saccades were different. Therefore, the 2 eyes were not in simultaneous motion throughout the excursion. Krauskopp *et al.*²⁴ have stated that the correlation coefficient between the magnitudes of small fixation saccades in the 2 eyes ranged from only 0.76 to 0.91. Smith *et al.*²⁵ stated that the times at which the eyes attain their maximum velocities may differ by as much as 5 msec. Goodwin and Fender²⁶ and, later, Fricker and Sanders²⁷ cross-correlated target position to the position of each eye and stated that the time delay to the peak value was usually different for the 2 eyes. Pickwell,²⁸ without showing records, and Stark,²⁹ without explaining but showing records, demonstrated that in eye movements during changes in asym-

metric vergence, the saccade of the non-dominant eye was larger. Weber and Daroff¹⁴ named glissades and demonstrated their monocularly; previously, Stark²⁹ had shown a glissade as an example of a violation of Hering's Law. All of these authors demonstrated only dynamic violations of Hering's Law, for only the pulse portions of the pulse-step controller signals were different.

Artifactual Considerations

Artifacts in eye movement records may be produced by Hering's Law violations. EOG is a noisy technique for measuring eye movements. Part of this noise comes from jaw muscle potentials. However, if the 2 EOG electrodes are placed so that an electrode is at the outer canthus of each eye, jaw muscle potentials will be picked up by both electrodes. When the difference of the 2 electrode voltages is taken, the jaw muscle potentials will be subtracted out, and therefore the EOG records will be less noisy. Many investigators have used this technique.³⁰⁻³⁶ In fact, one investigator³⁷ used this averaging technique with infrared photodiodes. If Hering's Law were never violated, then the average eye movement measured with this technique would be very similar to the actual movements of each eye. However, we have shown that the movements of the 2 eyes may start at different times (Fig. 2), may end at different times (Figs. 1, 2, 3, and 5), may have different velocities (Figs. 1, 2, and 7), and may have different shapes, due to either closely spaced saccades (Fig. 1), overlapping saccades (Fig. 2), dynamic overshoot (Fig. 3), or glissades (Figs. 5, 6, and 7). Therefore, we view with caution the records derived by averaging the movements of the 2 eyes.

Static Violations of Hering's Law

Since the vertical muscles have different actions as functions of horizontal position⁸ (e.g., when the eyes are abducted 39 degrees, the obliques have no vertical action¹), static violations of Hering's Law often occur when the eyes are not near primary position. Therefore, Hering's Law treats the 4 vertical muscles as 1 group. Furthermore, for abduction of more than 23 degrees from primary position, the vertical recti reverse their cyclorotary functions.² Thus, assignment of yoke muscles even as a foursome would not be consistent with Hering's Law. Hering³⁸ acknowledged these difficulties. He treated the 4 vertical muscles as 1 group, but he did not have their innervational relationships change as functions of horizontal eye position. Thus, he did not suppose that a variable yokedness occurred, which might have compensated

for the changed effectiveness of the vertical recti and obliques as functions of horizontal position. However, he did suggest that the fan-like insertions of the extraocular muscles into the globe would have an influence in minimizing the variable effectiveness of the recti and obliques. Further consequences of removing the eyes from primary position are discussed in the appendix of this paper.

Hering's Law Is a Valuable Clinical Tool.

Static violations of Hering's Law are produced by patients with static internuclear ophthalmoplegia. These patients are for all practical purposes unable to use versional eye movements to adduct the affected eye into the nasal field.¹ In contrast, certain patients manifest secondary deviations which are a direct consequence of obeying Hering's Law of Equal Innervation. For example, a patient may have had a paretic right lateral rectus due to either muscle, 6th nerve, or motoneuronal pathology. When viewing the target with the normal left eye, the covered paretic right eye would be pointed nasalward showing decreased abduction, a primary deviation. When viewing the target with the paretic right eye, the covered left eye would be pointed nasalward showing an exaggerated overcompensated adduction, a secondary deviation which is greater than the primary deviation.¹ The increased innervation sent to the right lateral rectus to compensate for its weakness is accompanied, as predicted from Hering's Law, by an increased innervation to its yoke muscle, the healthy left medial rectus. This produces the larger secondary deviation.

Although dynamic violations of Hering's Law frequently occur in normal subjects, particular patterns of dynamic violations may have clinical significance. For example, patients with dynamic internuclear ophthalmoplegia show adductor glissadic undershoot and concomitant abductor glissadic overshoot. This syndrome is caused by lesions in the medial longitudinal fasciculus, usually due to multiple sclerosis if bilateral, or vascular accidents if monocular.

Summary

Static violations of Hering's Law of Equal Innervation are usually indicative of pathological conditions, while dynamic violations usually are not. Normal subjects, especially when slightly fatigued, frequently execute closely spaced saccades, overlapping saccades, slow fatigued saccades, curved oblique saccades, dynamic overshoots, and glissades. Because these phenomena are monocular, the eye movements are dynamic violations of Hering's Law.

APPENDIX—THE 39° PARADOX

Static violations of Hering's Law are much less common than dynamic violations. The few static violations that we have presented have been either rare examples in normals or records taken from pathological patients. However, there are possibly 2 additional static violations of Hering's Law which may occur for vertical saccades with the eyes moderately abducted. They are elucidated by the 39-degree paradox. The primary feature of this paradox is that the forces available from the vertical muscles are functions of the horizontal position; therefore, a set of controller signals appropriate for an abducted eye would be inappropriate for an adducted eye. Hering understood this and elaborated his rule so as to treat the 4 vertical muscles as 1 group. (We discuss this idea later.) The secondary feature of the paradox shows that small vertical saccades executed with an eye 39 degrees abducted would produce either cyclorotary disparities or static violations of Hering's Law.

In this hypothetical experiment the subject looked at a target which abducted his right eye by 39 degrees and then made small vertical saccades about this point. With the right eye abducted 39 degrees, its superior and inferior oblique muscles were pure cyclorotators and contributed *no* force for elevation or depression;¹ the small upward saccades were executed entirely by the superior rectus agonist. To make an upward saccade, the innervation to the right superior rectus must have increased, but in this position this would also produce a small amount of excyclorotary force.² Either excyclorotation of the right eye existed due to this excyclorotary force of the right superior rectus, or an increase of innervation to the right superior oblique compensated for this excyclorotary force.

Let us first assume that there was no net excyclorotation of the right eye. (We have not been able to observe it visually.) In order to counteract the excyclorotary force of the right superior rectus, the innervation to the right superior oblique must have increased. Meanwhile, during this eye movement, what has happened to the left eye, which has been adducted about 39 degrees? (It would be adducted by more than 39 degrees for near targets. For example, it would be adducted by 42 degrees for targets 57.3 cm away from the midline of the subject's forehead.) To make the upward saccade, the innervation to both the left superior rectus and the left inferior oblique must have increased, and by Descartes' Law of Reciprocal Innervation,³⁹ the innervation to the left inferior rectus and the left superior oblique must have

decreased. This is in contradiction to Hering's Law, which predicts that if the innervation to the right superior oblique increased, then the innervation to its yoke muscle, the left inferior rectus for versional eye movements, must have also increased. (This theoretical violation holds not only when the eye is abducted by 39 degrees, but also when the eye is abducted by any amount between 23 and 51 degrees. For at 23 degrees abducted, the superior rectus changes from an incyclorotator to an excyclorotator, while its yoke muscle, the inferior oblique, does not change its action.)

Could this contradiction be due to our assumption of increased innervation to the right superior oblique in order to produce zero cyclorotation of the right eye? To check this, let us suppose that the innervation to the right superior oblique was zero for the upward saccade, for this would have allowed excyclorotation of the right eye. Then, according to Hering's Law, innervation to the yoke muscle, the left inferior rectus, must have been zero. Therefore, the vertical saccade in the left eye must have been executed solely by the left inferior oblique, which would have produced upward movement and excyclorotation of the left eye. With excyclorotation of both eyes, there would have been cyclorotary disparity and violations of Listing's Law, but not necessarily of Donders' Law. Therefore, this solution would not have alleviated the dilemma.

There is one more logical possibility for innervation of the right superior oblique: during the vertical saccade, the innervation to the right superior oblique may have decreased. Thus, there would have been excyclorotary forces due to *both* the right superior rectus and the right inferior oblique. This would have produced very large excyclorotation, because the right inferior oblique would have been acting in a plane where its total force would have been cyclorotary. Thus, there would have been very large excyclorotation of the right eye. Meanwhile, what would have happened in the left eye? The left superior rectus would have produced upward movement and incyclorotation, while the left inferior oblique would have produced upward movement and excyclorotation. These cyclorotary forces would have tended to cancel each other out: the left eye would have had little cyclorotation, while the right eye would have had a large excyclorotation. This also would have produced cyclorotary disparity and therefore would not have solved our dilemma.

So, it seems that small vertical saccades with one eye abducted 39 degrees produced irreconcilable problems for Hering's Law of Equal Innervation. There are at least 3 solutions to this problem. First, there is the answer Ewald

Hering³⁸ used in 1868. He did not use the term yoke muscles. Instead, he treated the vertical recti and the vertical obliques as a single group of muscles with variable interactions between them. Secondly, we could assume that the eyes actually do have different cyclorotation and that this cyclorotatory disparity is ameliorated by neural processing. Kertesz and Jones⁴⁰ and G. van den Brink (personal communication) have suggested the cyclofusion is accomplished by neural signal processing. Third, we could ignore innervation completely and restate Hering's Law in terms of eye movements, as Hering did in 1879: "The movements of the two eyes are equal and symmetrical".⁴¹⁻⁴²

If Hering's original solution is used, then Hering's Law could be stated as follows: *Tonic innervations of the horizontal and vertical groups of muscles of both eyes usually change by the same amount either in the same direction for versional eye movements, or in opposite directions for vergence eye movements.*

We believe that this statement of Hering's Law is in keeping with what Hering believed. It requires that the 4 vertical muscles be treated as a group and not individually or even as pairs of muscles. In studies of oblique eye movement trajectories,^{7, 43} no evidence could be found that the 4 vertical muscles did not act as a single group, (i.e., for the independence of the oblique and vertical recti muscles). Interestingly, Nakayama⁴⁴ has adopted a similar scheme in order to explain the implementation of Listing's Law. He also showed that neither Listing's Law nor Descartes' Law of Reciprocal Innervation held during sleep. We have similarly shown here that fatigue increases the frequency of Hering's Law violations.

We have shown that Hering's Law does not apply during eye movements. Analogously, Westheimer and McKee⁴⁵ have shown that Donder's Law does not hold during smooth pursuit eye movements.

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REFERENCES

1. Cogan, David, Neurology of the Ocular Muscles, Charles C Thomas, Springfield, Ill., 1948, pp. 10, 52, 57-60, 92.
2. Alpern, Mathew, Movements of the eyes, in The

- Eye, Vol. 3, edited by Hugh Davson. New York, Academic Press, 1962, pp. 36, 91.
3. Adler, Francis H., Physiology of the Eye. The C. V. Mosby Co., St. Louis, 4th ed. 1965, p. 384.
4. Bahill, A. Terry, Michael Clark, and Lawrence Stark, Dynamic overshoot in the saccadic eye movements is caused by neurological control signal reversals, *Exp. Neurol.*, 48: 107-122, 1975.
5. Bahill, A. Terry, Neurological Control and the Hyperfine Structure of Saccadic Eye Movements. Doctoral Dissertation, Department of Electrical Engineering and Computer Science, University of California, Berkeley, 1975.
6. Bahill, A. Terry, Deborah Adler, and Lawrence Stark, Most naturally occurring human saccades have magnitudes of 15 degrees or less, *Invest. Ophthalmol.*, 14 (6): 468-469, 1975.
7. Bahill, A. Terry, and Lawrence Stark, Neurological control of horizontal and vertical components of oblique saccadic eye movements. *Math. Biosci.*, 27: 287-298, 1975.
8. Taumer, R., J. Hennig, and D. Pernice, The ocular dipole—A damped oscillator stimulated by the speed of change in illumination. *Vision Res.*, 14: 637-645, 1975.
9. Bahill, A. Terry, Karen Bahill, Michael Clark, and Lawrence Stark, Closely spaced saccades, *Invest. Ophthalmol.*, 14 (4): 317-321, 1975.
10. Bahill, A. Terry, and Lawrence Stark, Overlapping saccades and glissades are produced by fatigue in the saccadic eye movement system, *Exp. Neurol.*, 48: 95-106, 1975.
11. Bahill, A. Terry, Michael R. Clark, and Lawrence Stark, Computer simulation of overshoot in saccadic eye movements. *Computer Programs in Biomedicine*, 4 (4): 230-237, 1975.
12. Bahill, A. Terry, and Lawrence Stark, The high-frequency burst of motoneuronal activity lasts about half the duration of most saccadic eye movements. *Math. Biosci.*, 26 (3/4): 319-323, 1975.
13. von Helmholtz, Hermann, Helmholtz's Treatise on Physiological Optics, Vol. 3, with notes by von Krish, edited by J. P. C. Southall. New York, Dover Publications, Inc., 1962, p. 153.
14. Weber, R. B., and R. B. Daroff, The metrics of horizontal saccadic eye movements in normal humans, *Vision Res.*, 11: 921-928, 1971.
15. Bahill, A. Terry, Michael R. Clark, and Lawrence Stark, Glissades—Eye movements generated by mismatched components of the saccadic motoneuronal control signal, *Math. Biosci.*, 26 (3/4): 303-318, 1975.
16. Dell'Osso, Louis F., David A. Robinson, and Robert B. Daroff, Optokinetic asymmetry in internuclear ophthalmoplegia, *Arch. Neurol.*, 31: 138-139, 1974.
17. Fricker, Stephen J., and James J. Sanders, Velocity and acceleration statistics of pseudorandomly timed saccades in humans, *Vision Res.*, 15: 225-229, 1975.
18. Metz, Henry S., Saccadic velocity measurements in internuclear ophthalmoplegia, *Am. J. Ophthalmol.*, 81 (3): 296-299, 1976.
19. Kommerell, Guntram, Internuclear ophthalmoplegia of abduction. *Arch. Ophthalmol.*, 93: 531-534, 1975.

20. Bahill, A. Terry, Michael R. Clark, and Lawrence Stark, The Main Sequence, a tool for studying human eye movements, *Math. Biosci.*, 24: 191-204, 1975.
21. Bahill, A. Terry, Frederick K. Hsu, and Lawrence Stark. Development, Sensitivity Analysis, and Predictions of a Reciprocal Innervation Model for Saccadic and Vergence Eye Movements. A paper presented at the Seventh Annual Pittsburgh Conference on Modeling and Simulation, Pittsburgh, Penn., April 26-27, 1976.
22. Barmack, Neal H., Saccadic discharges evoked by intracellular stimulation of extraocular motoneurons, *J. Neurophysiol.*, 37 (3): 395-412, 1974.
23. Dodge, Raymond, and Thomas Cline, The angle velocity of eye movements, *Psychol. Rev.*, 8 (2): 145-157, 1901.
24. Krauskopp, John, Thomas N. Cornsweet, and Lorin A. Riggs, Analysis of eye movements during monocular and binocular fixation, *J. Opt. Soc. Am.* 50 (6): 572-578, 1960.
25. Smith, Karl U., Jack Schmidt, and Vernon Putz, Binocular coordination: Feedback of synchronization of eye movements for space perception, *Am. J. Optom.*, 47 (9): 679-689, 1970.
26. Goodwin, A. W., and D. H. Fender, The interaction between horizontal and vertical eye-rotations in tracking tasks. *Vision Res.*, 13: 1701-1712, 1973.
27. Fricker, Stephen, and James J. Sanders, Internuclear ophthalmoplegia and associated abnormalities in eye motion timing (differential delays), *Neurology* 25 (3): 281-285, 1975.
28. Pickwell, L. D., Hering's Law of equal innervation and the position of the binoculus, *Vision Res.*, 12: 1499-1507, 1972.
29. Stark, Lawrence, The control system for versional eye movements, in *The Control of Eye Movements*, edited by P. Bach-y-Rita, C. C. Collins and J. Hyde, New York, Academic Press, 1971, pp. 376, 377.
30. Bizzi, E., R. E. Kalil, P. Morasso, and V. Tagliacolo, Central programming and peripheral feedback during eye-head coordination in monkeys, in *Cerebral Control of Eye Movements and Motion Perception*, edited by J. Dichgans and E. Bizzi, S. Karger, Basel, No. 82 in *Bibliotheca Ophthalmol.*, Series, pp. 220-232, 1972.
31. Levy-Schoen, Ariane, and Jeanine Blanc-Garin, On oculomotor programming and perception, *Brain Res.* 71: 443-450, 1974.
32. Stern, J. A., D. A. Bremer, and J. McClure, Analysis of eye movements and blinks during reading: Effects of Valium, *Psychopharmacologia*, 40: 171-175, 1974.
33. Becker, W., and H. Klein, Accuracy of saccadic eye movements and maintenance of eccentric eye positions in the dark, *Vision Res.*, 13: 1021-1034, 1973.
34. Taumer, R., K. Mie, and G. Kommerell. Three kinds of reaction mechanisms of the human saccadic system, in *Biocybernetics*, edited by H. Drischel and P. Dettmar. Veb Gustav Fischer Verlag Jena, 1972, Vol. 4, pp. 236-242.
35. Brockhurst, Robert J., and Kurt S. Lion, Analysis of ocular movements by means of an electrical method. *AMA Arch. Ophthal.*, 46: 311-318, 1951.
36. Lisberger, S. G., A. F. Fuchs, W. M. King, and L. C. Evinger, Effect of mean reaction time of saccadic responses to two-step stimuli with horizontal and vertical components, *Vision Res.*, 15: 1021-1026, 1975.
37. Barmack, Neal H., Dynamic visual acuity as an index of eye movement control. *Vision Res.*, 10: 1377-1391, 1970.
38. Hering, Ewald, *The Theory of Binocular Vision*. Translated by B. Bridgeman, edited by B. Bridgeman and L. Stark. New York, Plenum Press, 1977.
39. Ciuffreda, Kenneth J., and Lawrence Stark. Descartes' law of reciprocal innervation. *Am. J. Optom.*, 52 (10): 663-673, 1975.
40. Kertesz, Andrew, and Richard Jones, Human cyclofusional response, *Vision Res.*, 10: 891-896, 1970.
41. Duke-Elder, Sir Stewart, and Kenneth Wybar, *Ocular Motility and Strabismus*, in *System of Ophthalmology*, Vol. VI, edited by S. Duke-Elder. London, Henry Kimpton, 1973, p. 125.
42. Davson, Hugh, *The Physiology of the Eye*, 3rd ed. New York, Academic Press, 1972, p. 331.
43. Bahill, A. Terry, and Lawrence Stark. Dynamic independence of horizontal and vertical components of oblique saccadic eye movements, *Arch. Ophthalmol.*, in press.
44. Nakayama, Ken, Coordination of extraocular muscles, in *Basic Mechanisms in Ocular Motility and Their Clinical Implications*, edited by G. Lennerstrand and P. Bach-y-Rita. New York, Pergamon Press, 1975, pp. 193-207.
45. Westheimer, Gerald, and Suzanne McKee. Failure of Donders' law during smooth pursuit eye movements, *Vision Res.*, 13: 2145-2153, 1973.

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