Analysis of human vergence dynamics

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Disparity vergence is commonly viewed as being controlled by at least two mechanisms, an open-loop vergence-specific burst mechanism analogous to the ballistic drive of saccades, and a closed-loop feedback mechanism controlled by the disparity error. We show that human vergence dynamics for disparity jumps of a large textured field have a typical time course consistent with predominant control by the open-loop vergence-specific burst mechanism, although various subgroups of the population show radically different vergence behaviors. Some individuals show markedly slow divergence responses, others slow convergence responses, others slow responses in both vergence directions, implying that the two vergence directions have separate control mechanisms. The faster time courses usually had time-symmetric velocity waveforms implying open-loop burst control, while the slow response waveforms were usually time-asymmetric implying closed-loop feedback control. A further type of behavior seen in a distinct subpopulation was a compound anomalous divergence response consisting of an initial convergence movement followed by a large corrective divergence movement with time courses implying closed-loop feedback control. This analysis of the variety of human vergence responses thus contributes substantially to the understanding of the oculomotor control mechanisms underlying the generation of vergence movements.

Keywords: oculomotor, dynamics, vergence, binocular, eye movements, convergence, divergence, anomaly

Citation: Tyler, C. W., Elsaid, A. M., Likova, L. T., Gill, N., & Nicholas, S. C. (2012). Analysis of human vergence dynamics. *Journal of Vision*, *12*(11):21, 1–19, http://www.journalofvision.org/content/12/11/21, doi:10.1167/12.11.21.

Introduction

Vergence control

The present study focuses on the issue of binocular vergence movements and the analysis of their control mechanisms when driven by large-field changes in disparity. Disparity vergence is commonly viewed as being controlled by at least two mechanisms, an open-loop vergence-specific burst mechanism analogous to the ballistic drive of saccades, and a closed-loop feedback mechanism controlled by the disparity error (Krishnan & Stark, 1977; Hung, Semmlow, & Ciuffreda, 1986; Semmlow, Hung, & Ciuffreda, 1986; Semmlow, Hung, Horng, & Ciuffreda, 1993; Erkelens, 2011). The time course of the vergence-specific burst is an order of magnitude slower than the saccadic time course for the same amplitude of motion of each eye.

The existence of a burst component of disparity vergence movements, though initially contentious (Robinson, 1971; Krishnan & Stark, 1977), was established by studies such as that of Mays, Porter, Gamlin, and Tello (1986) and Gamlin and Mays (1992), which showed that the change in eye position (eye velocity) was directly proportional to the firing rate of the motorneurons driving the eye muscles involved (Figure 1). Since the vergence signals underwent a sigmoid change in position, the motorneuron burst matches the roughly Gaussian or gamma-function form of the velocity trace. It is now well established that the change in midbrain motorneuron firing rate is linearly proportional to the vergence

doi: 10.1167/12.11.21 Received February 7, 2012; published October 25, 2012

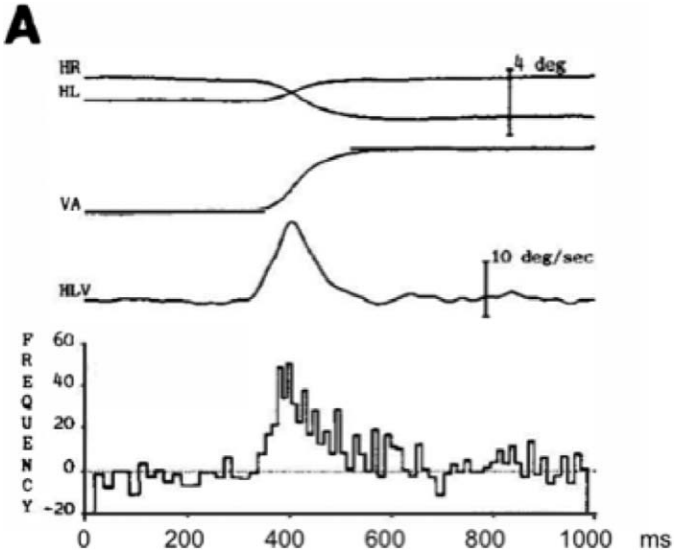

Figure 1. Monkey vergence response reproduced from Gamlin and Mays (1992), showing the right and left horizontal eye position traces (HR, HL), the difference trace of vergence amplitude (VA), the corresponding vergence velocity trace (HLV), and the concomitant change in firing rate of a midbrain medial rectus motorneuron recorded in the oculomotor nucleus during the movement. Note the similarity between the waveform of the velocity trace and motorneuron firing burst, which both have relatively long durations of about 200 ms.

velocity (with a gain of 1–5 spikes/s per °/s across the cell population; Mays et al., 1986; Gamlin & Mays, 1992). Thus, vergence velocity may be regarded as an effective proxy for the firing rate of the underlying motorneurons.

The presence of the closed-loop component of disparity vergence control is consistent with the idea that the vergence waveform beyond the peak velocity should approximate the form of an exponential decay function, due to the proportional reduction in the error signal as the eye approaches the target positions (Robinson, 1971; Zee, Fitzgibbon, & Optican, 1992). There are many cases in which the data approximate such an exponential decay. (Here we use the term "exponential" in the qualitative sense of a continuously decreasing velocity as the eyes asymptote to the final position, without attempting to assess how well it conforms to a mathematical exponential of the form e^{-x}).

The presence of two processes feeding into the motorneurons is further implied by the "dual mode theory" of Hung et al. (1986) and Semmlow and Yuan (2002), based on an independent components analysis of the variations in the disparity vergence responses, implying that vergence movements consist of a rapid transient component and a slower sustained component. However, it should be noted the assumption of independent components analysis is that the components are invariant in temporal waveform (varying only in their relative amplitudes), although it is well known that vergence movements have variability of their temporal parameters, which could give a spurious result of an initial transient component under the incorrect assumption of temporal invariance. The independent components analysis is not, therefore, by itself sufficient evidence of a separate transient component to disparity vergence.

Recent work on the optimal control theory of saccadic dynamics suggests that the saccadic waveform is tuned to jointly minimize the duration and accuracy of each saccade (Harris & Wolpert, 1998, 2006; Tanaka, Krakauer, & Qian, 2006; Xu-Wilson, Zee, & Shadmehr, 2009). At least for small saccades, this theory predicts a time-symmetric velocity function for the saccadic waveform, approximating a half-cosine waveform. Based on the empirical results of the dual-mode theory, we take this symmetry to be the signature of the transient component.

If valid, this dual mode analysis implies that the open-loop component of vergence movements would have the same waveform structure as a saccade, although far slower in time course. This concept suggests that the waveform of the open-loop component should be time-symmetrical (in the sense that the waveform velocity, or temporal derivative of the waveform, has symmetrical rise and fall times). This symmetric form is driven by a (slow) burst of activity in the oculomotor neurons driving the vergence movement and is formally distinguishable from the exponential concept of the closed-loop component, which implies a time-asymmetrical waveform as the feedback progressively reduces the error to zero. We will use this difference in time-symmetry of the two components as a key to their relative predominance in our subject populations.

Types of vergence dynamics

In terms of peak velocity, it is well established that there is a "main sequence" function of peak velocity versus amplitude for disparity vergence eye movements, very similar to that for saccades (Rashbass & Westheimer, 1961; Erkelens, Van Der Steen, Steinman, & Collewijn, 1989; Hung, Ciuffreda, Semmlow, & Horng, 1994). The summary data of the latter study show a roughly linear increase in vergence velocity with amplitude up to about 2°, with a progressive saturation of the curve for larger amplitudes. For reference, the slope of the linear portion is about 7 °/s.

It is also well known that disparity vergence is not always perfectly matched between the two eyes, i.e., with matching time-functions in opposite directions in the two eyes (Collewijn, Erkelens, & Steinman, 1995).

Instead there is often reported to be an asymmetrical saccade in one eye (even when the target motion is perfectly symmetrical), which may be seen as the vergence system employing a biased strategy to achieve a faster acquisition of the vergence target (Coubard & Kapoula, 2008).

The third well-known disparity vergence behavior is seen for the situation of asymmetrical vergence to a laterally off-center target, in which case a uniocular saccade is typically used to bring the eyes to the average position required by the target endpoint, with a slow convergence movement (which should have the exponential waveform) to complete the motion (Collewijn et al., 1995). Conversely, Horng, Semmlow, Hung, & Ciuffreda (1998) reported that the initial open-loop component of disparity vergence could be much reduced or absent in the divergence direction, although this conclusion was based on recordings from only two subjects, and other studies have not reported such a convergence/divergence imbalance.

Two kinds of disparity vergence movement are excluded from this account. One is the case of symmetrical vergence saccades with the normal saccadic time course but in opposite directions in the two eyes. This form of saccadic eye movement is considered to be specifically prohibited in the conventional account, although the explicit mechanism of the prohibition is obscure.

Vergence latencies

This account of the vergence dynamics would be incomplete without a consideration of the vergence latencies, which are generally included as a free parameter in the cited models of vergence control. It is noteworthy that the vergence latencies are quite similar to those of saccades, at around 200 ms. Any differences reported in the literature are likely to be attributable to sampling differences among the subject groups and the details of the method used to specify the time of onset of the respective movements. On this view, the latency for the initiation of both saccades and vergences is attributable to the cortical processing of the sensory stimulation providing the impetus for the eye movements, i.e., the computational processes specifying the required amplitude and direction of the movements and the decision to initiate the movements (since they are essentially voluntary in character and can be suppressed if necessary). Once initiated, the control for the activation is passed to the oculomotor control systems of the brainstem and cerebellum, which evidently treat the two kinds of oculomotor requests very differently, but it is likely that the cortical processing is basically similar in the two cases.

Rationale

Based on the foregoing overview, the main goal of the present paper is to reassess the status of symmetrical vergence movements and the extent to which they are achieved either by the open-loop (burst) or the closed-loop (exponential) component of the neural control system. This reassessment was achieved by evaluation of the temporal asymmetry of the vergence velocity profiles around the peak velocity for a large number of subjects. The exponential decay form of the closed-loop model should have a highly asymmetric profile, while the open-loop burst model should be close to symmetric (similar to that for typical saccades; Harris & Wolpert, 1998). The temporal asymmetry criterion is thus a good indicator of the underlying mechanism of vergence control and is quantified in the form of a temporal asymmetry index (see Methods).

A further goal of the paper is to evaluate the form of the vergence dynamics to full-field targets. In the world, when an object moves toward or away from the eyes or when we move through the world, large regions of texture typically stimulate the retina. However, many oculomotor studies use only small targets to stimulate the vergence system. Here we evaluate the disparity vergence component of such large field stimulation, showing that anomalous forms of vergence eye movements are in fact encountered at an unexpectedly high frequency.

Methods

Recruitment

This study involved a recruited base of 68 participants (59% female) from a nonacademic online marketplace for the normative study of oculomotor dynamics, who passed the exclusion criterion of having no clinical history of brain or ocular abnormalities, including traumatic brain injury events. All recruitment and experimental procedures in this study adhered to the Declaration of Helsinki.

The participants met the criteria of letter acuity of 20/40 or better in both eyes (Bailey-Lovie chart, mean left eye [LE] denominator :23 ± 5, mean right eye [RE] denominator :24 ± 6) and of passing a random-dot stereopsis test at a disparity of 4 arcmin. The ages ranged from 19–60 (mean :36 ± 12).

Oculomotor procedures

Binocular eye movements were recorded with the Visagraph III binocular infrared limbal eye tracker

(Compevo AB, Stockholm, Sweden), which has a sampling rate of 60 Hz and a typical noise level of ~3 arcmin standard deviation in each eye for live human recordings (as assessed from the variability during fixation periods in the most stable participants). This assessment provides a net vergence noise level of ~3 arcmin after the four-point elliptical (third-order) smoothing applied to the eye movement traces. Of course, the measured variability would also take into account the physiological variation in the fixation capability and oculomotor stability of each participant. This variation would typically be much larger than the irreducible measurement error, but we note that many participants achieved calibrated mean standard errors of the repeated vergence signals close to the estimated optimal level of about 3 arcmin (see Figures 3 through 11).

Horizontal position calibration series

To calibrate the linearity of the recorded position function, a 0.4° cross-hair monocular fixation target underwent two randomized sets of horizontal position shifts over the range from −16° to 16° for each eye, with button presses indicating when fixation was accurate at each position. The full set of points was fitted with a third-order polynomial to provide a linear calibration of the horizontal position separately for each eye.

Rapid horizontal disparity vergence jumps

Binocular eye movements were recorded while the 30° × 30° noise field incorporating a 1° central fixation target that underwent 2° horizontal square-wave disparity changes every 2–3 s, with random jitter over 1 s from a uniform distribution to avoid predictability of the onset time. The minimum interval of 2 s allowed comfortable completion of repeated normal vergence movements.

Oculomotor time series analysis

The vergence (LE minus RE) signal waveforms were extracted from a period around the times of the instantaneous transitions of the stimulus in a window from 1 s prior to the transition to 2.5 s after the transition. The sets of converging eye movement transitions were analyzed separately from diverging eye movements. Each event response was rezeroed by removing the mean value over the 100 ms preceding the transition. Nonrepresentative individual responses were excluded from the analysis by iteratively removing responses whose mean squared error over time from the mean across nonexcluded responses was beyond 2 standard deviations of the mean error. (In no case were more than three responses excluded under this procedure.)

The average nonexcluded vergence signal was numerically differentiated to derive the vergence velocity signal for each direction of eye movement for each subject. To account for noise and drift, the full duration of the vergence movements was defined as the time from the last crossing of 5% of the peak velocity prior to the time of the peak to the first crossing of 10% of the peak velocity after the peak. (The limits are asymmetric because the onset variance was smaller than the offset variance as a consequent of the respective distances from the zeroing region.) The portions of the velocity traces beyond these limits are blocked out to focus the presentation on the degree of symmetry *during* the primary vergence movements.

Temporal asymmetry

Temporal asymmetry of the velocity trace was defined by computing the ratio of the postpeak area minus the prepeak area to the total area of the vergence interval defined from the velocity trace. In principle, this temporal asymmetry index has a value of 0 for a time-symmetric waveform and a value of 1 for a pure exponential waveform. In practice, the smoothing applied to the waveform reduces the maximum value for the pure exponential response after the filtering of the waveforms, so we defined a Normalized Temporal Asymmetry Index (γ) as the ratio of the empirical temporal asymmetry index to the theoretical temporal asymmetry index for a filtered exponential decay. (Note that a waveform with an asymmetry sharper than the exponential form could have $\gamma > 1.0$).

Statistical analysis

The statistical analyses were performed by *t*-tests. Unless otherwise noted, significant results are reported at level of $p < 0.01$. Results reported as nonsignificant did not pass the criterion of $p < 0.05$.

Group categorization analysis

The disparity vergence dynamics estimates were not homogeneous across the population, but had a surprising variety of response characteristics. In order to formalize the categorization process, we took the approach of finding approximately Gaussian groupings of the duration parameter of the participants' vergence responses. The first step was to restrict the analysis to

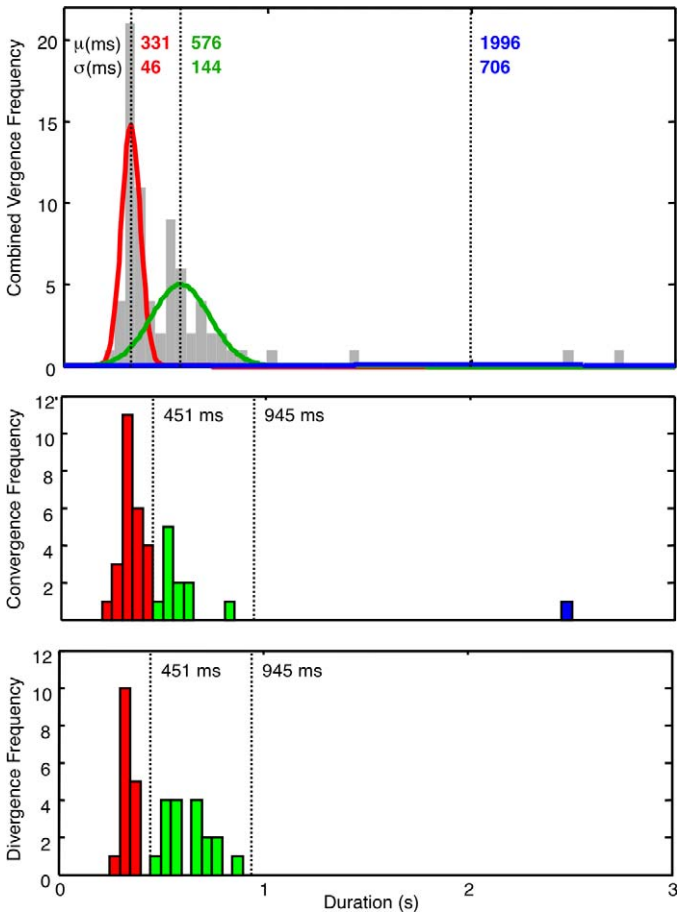

Figure 2. Top panel: Gaussian mixture model cluster analysis of all well-formed vergence response durations, showing the fit for the optimal 3-Gaussian case (red, green, and blue curves), with respective mean ± parameters. Center panel: Distribution of convergence duration clusters with cluster boundaries at 451 and 945 ms. Bottom panel: Distribution of divergence duration clusters, similarly.

the population of well-formed responses by weeding out all unreliable responses and those that did not achieve an amplitude criterion of a least half the disparity demand of 2° within an onset latency of 500 ms. Fifty-four individuals (77%) met these criteria for well-formed responses.

The next step was to combine all the vergence durations (convergence or divergence) into a single group and apply a Gaussian mixture cluster routine to determine the optimal number of Gaussian clusters to categorize the durations. The optimization error was specified according to the Bayesian Information Criterion (Schwarz, 1978) over fits from 1 to 12 Gaussians, which reached a minimum with a three-Gaussian model. The three Gaussians best fitting the distribution are shown in Figure 2. (Note that the blue curve for the long-duration fit is close to the duration axis.) The cluster boundaries between adjacent distributions were determined from the upper 1% cutoffs of each Gaussian, which occurred at 451 and 945 ms, defining three duration ranges: typical, mid, and slow vergence durations (T, M, and S, respectively). These boundaries were then applied separately to the convergence and divergence duration distributions, respectively, to form nine dual-criterion categories in principle.

A final category that will be specified in detail in Results had divergence responses that did not pass the initial screen because they began by consistently converging prior to initiating the divergence movement (Anomalous Compound Divergence). This behavior implied that the divergence latency failed the screening criterion of <500 ms, although the divergence velocities once initiated were within or close to the typical range of ~10 °/s.

Results

Disparity vergence dynamics

Before analyzing the results in detail, the quantitative indices of median latency, duration, peak velocity, and temporal asymmetry, together with their ± standard errors of the means (*SEM*), are presented in tabular form in Table 1. The proportion of subjects falling into each category is tabulated in the first row of the table.

Typical convergence/divergence responses

The "typical" response group is defined as those individuals with vergence movements in the short-duration range, up to about 450 ms in duration, for both the convergence and divergence directions. Because atypical vergence behavior was more common in the divergence than the convergence direction, the typical vergence behavior was treated in two ways. To define the typical convergence performance parameters for a Typical Convergence (All) group, mean convergence responses meeting the short-duration criterion of 451 ms were pooled across the population, regardless of divergence performance. Similarly, mean divergence responses meeting the short-duration criterion of 451 ms were pooled for a Typical Divergence (All) group, regardless of convergence performance. But the main categorization was performed on individuals rather than responses, and for this purpose the individuals with typical responses in both the convergence and divergence directions were identified to form a Typical Both category.

For Typical Convergence (All) group, the median convergence latency was 252 ± 27 ms with a typical

	Typical conv (all)		Typical div (all)		Typical both		Typical conv (only)		Typical div (only)	
N (%)	62		32		22		28		10	
	Median	*SEM*			Median	*SEM*	Median	*SEM*	Median	*SEM*
Convergence										
Median latency (ms)	252	27			252	37	275	40		
Peak velocity (deg/s)	9.5	1.5			10.3	2.8	8.1	1.4		
Duration (ms)	333	12			330	18	341	17		
Templ asymm	0.17	0.18			0.09	0.06	0.16	0.32		
Divergence										
Median latency (ms)			283	32	317	35			283	32
Peak velocity (deg/s)			−9.6	1.5	−9.1	1.9			−7.9	1.5
Duration (ms)			337	10	317	12			317	10
Templ asymm			0.16	0.05	−0.06	0.06			0.16	0.05

Table 1. Vergence dynamics parameters for the vergence groupings. conv = convergence; div = divergence; ACD = anomalous compound divergence; Templ asymm = temporal asymmetry; SEM = standard error of mean.

peak velocity of 9.5 ± 1.5 °/s and median total duration of 333 ± 12 ms. The values for the Typical Divergence (All) group were not significantly different, with a median convergence latency of 283 ± 32 ms, a typical peak velocity of -9.6 ± 1.5 °/s and median total duration of 337 ± 10 ms. The convergence values, drawing from the larger group of 62% of the population, may therefore be taken as the typical high-performing vergence behavior, since the other groupings all had longer convergence durations.

Typical vergence responses

The Typical (Both) response group is defined as those individuals with vergence movements for both the convergence and divergence directions in the short-duration range up to 451 ms in duration. Examples of convergence movements for three individuals from the typical convergence/divergence group are shown in Figure 3. For convergence responses in this typical group, the median latency was 252 ± 37 ms, a typical peak velocity of 10.3 ± 2.8 °/s, and median total duration of 330 ± 18 ms, none of which are significantly different from the values for the larger group of typical convergence responses. Also, the velocity values are not significantly different from the corresponding velocity on the vergence main sequence summarized by Hung et al. (1994).

Typical divergence responses

In the group of individuals with typical response dynamics for both convergence and divergence movements, typical divergence responses had the following characteristics: latency of 317 ± 35 ms, typical peak velocity of −9.1 ± 1.9 °/s, and a total duration of 317 ± 12 ms. Examples are shown in Figure 4. These values indicate that none of the dynamic parameters for the typical divergence response are significantly different from those for the convergence movements. These parameters are again different by an order of magnitude from those of typical saccades (see Figure 4), indicating that the participants did not typically resort to saccadic strategies to achieve the divergence targets.

Temporal asymmetry

Across the group of typical subjects, the Normalized Temporal Asymmetry Index for the group (62%) of typical convergence response rates had the value of $\gamma = 0.17 \pm 0.18$, not significantly greater than zero. Because this superordinate grouping has some heterogeneity, we may evaluate the asymmetry for the smaller group with typical responses in both directions, for which the asymmetry had the tighter range of $\gamma = 0.09 \pm 0.06$, again not significantly different from zero. We may conclude that, for this widefield texture stimulus at the disparity convergence amplitude of 2°, there is no significant positive temporal asymmetry in the convergence response.

The Normalized Temporal Asymmetry Index was similar for the divergence responses of the superordinate group with typical divergence response rates. The net asymmetry was again small, at $\gamma = 0.16 \pm 0.05$, though significantly positive in this group. For the smaller group with typical responses in both vergence directions, the divergence asymmetry was, however, not significantly different from zero, $\gamma = -0.06 \pm 0.06$. Thus it seems that, in the fully typical individuals of the Typical Both group, divergence responses do not inherently have a different temporal form from

Mid both		Mid conv (only)		Mid div (only)		Slow vergence		ACD (div)		ACD (conv)	
12		10		22		6		18		(18)	
Median	*SEM*	Median	*SEM*	Median	*SEM*	Median	*SEM*	Median	*SEM*		
198	25	144	52			300	33	233	14		
7.3	1.6	6.0	1.1			8.1	1.1	10.2	1.2		
538	25	585	17			316	47	317	33		
0.27	0.11	0.64	0.26			−0.77	0.12	−0.84	0.15		
200	54			211	43	266	95	650	110	133	18
−5.0	1.3			−5.9	4.4	−3.8	0.2	−10.3	2.9	5.5	1.0
700	25			567	23	1433	516	554	59	483	45
0.01	0.57			−0.20	0.16	0.67	0.24	1.07	0.18	−0.91	0.22

Table 1. Extended.

convergence responses (Horng et al., 1998), although convergence/divergence differences will become apparent as the less typical groupings are analyzed.

To introduce the properties of temporal asymmetry, we have to anticipate somewhat the grouping results, so refer to the relevant sections for full details of those responses. The first case (Figure 5A) is an example of strong asymmetry for both convergence and divergence, from the "mid both" vergence response category (see Table 1). The range of the analysis is described in Methods. Note the separation between the forward time (green curves) and reverse time (gray curves) plots of the velocity plots, defining the waveform asymmetry. The second case (Figure 5B) is from the group of slow divergence responses with typical convergence responses. Note the tight overlap of the convergence velocity traces in contrast to the strong temporal asymmetry of the divergence traces. The third quartet of panels (Figure 5C) is a typical case from the Anomalous Compound Divergence group, in which the divergence response is reliably preceded by an inappropriate convergence movement. Rather than analyzing the responses to the convergence and divergence stimuli (as in the previous panels), here we analyzed the convergence and divergence *components* of the compound divergence movements. While the divergence component has a positive temporal asymmetry similar to the other cases, the convergence component shows a strongly *negative* value of temporal asymmetry, implying a more gradual onset than offset time-course. The interpretation of these asymmetry features is left to the Discussion.

Vergence/saccadic interactions

Before considering the role of vergence/saccadic interactions, we include here an analysis of the saccadic responses of the same subject group (and on the same timescale) with Figures 3 and 4 as a basis for comparison (Figure 6). The saccadic stimulus was a 1.25° circle/cross combination jumping between horizontal positions 10° to the left and right of primary (straight-ahead) position, with a temporal delay randomized over a flat distribution between 2 and 3 s. The average saccades over 12 repeats show the typical latencies, similar to those of the typical vergence group (316 ± 68 ms), and typical saccadic durations of about 80 ms (see Bahill, Clark, & Stark, 1975). This information is included to provide reassurance that the saccadic dynamics recorded under the present conditions are, as expected, very different from the vergence dynamics (about 4 times shorter duration, even though their amplitude was ~10 times larger in each eye).

We note that many subjects exhibit a habitual pattern of a slight saccadic undershoot followed by a corrective saccade, with an almost invariant pattern across the 12 repeats (shown as the barely-visible pink penumbra around the purple time courses in Figure 6). The timing of the corrective saccade was 173 ± 33 ms with a peak velocity of 38 °/s, similar to the values found in the original studies of this phenomenon (Becker & Fuchs, 1969; Becker, 1972).

In general, saccadic activity should not be expected to be a strong component of the symmetrical vergence response. In practice, the typical case is shown in Figure 7, where symmetrical vergence is accompanied by self-corrective microsaccades that often have a boxcar form with a saccade following the vergence movement and a correction a few hundred ms later. They represent the subjects' saccadic refixations within the 1.25° fixation target. The saccades in Figure 7 are conjunctive (lower trace), averaging out to a level that is undetectable relative to the prevailing noise level in the raw vergence difference waveform (upper trace). In this example, the

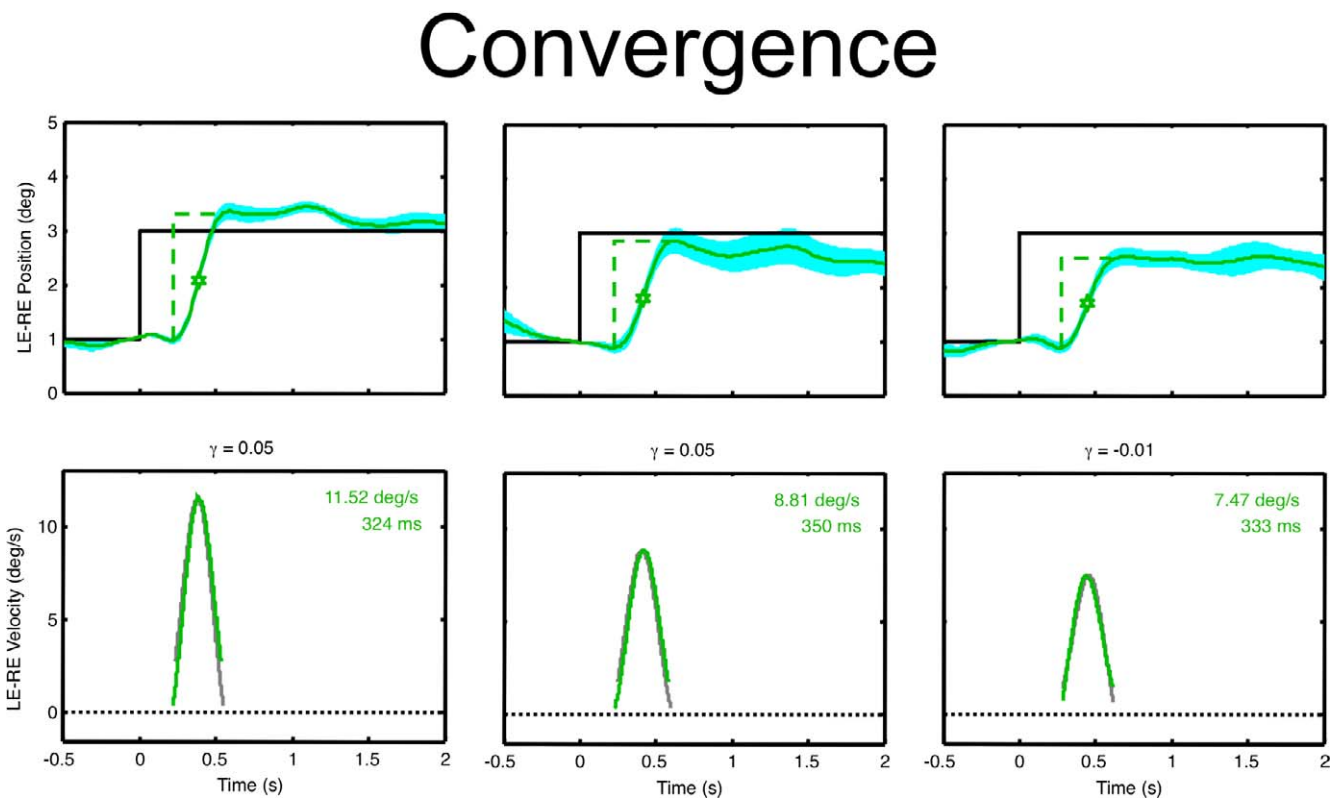

Figure 3. Upper plots: Examples of typical mean convergence responses for three individuals (green curves, each averaged over 10–12 responses). Black line indicates the stimulus disparity change event. Gray dotted lines show the estimated amplitude and duration of each vergence movement. Cyan region represents the ±1 *SEM* range of the variability over the 10–12 repeats for each trace. The star marks the point of peak velocity. Lower plots: Corresponding velocity profiles for the same average responses (color curve), together with the time-inverted velocity profile (light gray curve) aligned with the peak of each response to illustrate its degree of temporal asymmetry, quantified above each velocity panel. Note the high degree of symmetry (near-zero asymmetry) of these typical examples. Peak velocity and duration values (green annotations) are estimated from the velocity trace as described in Methods. The vergence movements are repeatable with a standard error of a few arcmin.

saccades are mainly to the right for both convergence and divergence movements, but there was no consistent pattern across subjects. As can be seen in the averaged vergence responses of Figures 3 and 4, there is no evidence of a consistent saccadic component contributing to the vergence responses; the vergence durations are of the order of 320 ms (see Table 1), with no hint of a coordinated saccadic jump anywhere along the averaged vergence waveforms (Figures 3 and 4).

Midrate vergence responses

As indicated in Figure 2, about one third of the sample had vergence responses of up to twice the duration of the typical rate in one or both vergence directions. According to the cluster analysis, these responses formed a distinct category with median durations of ~450–950 ms, some for convergence, some for divergence, and some for both. Of those with midrate vergences in both directions (12% of the sample; see Figure 8), the median durations were 538 ± 25 ms and 700 ± 25 ms for convergence and divergence, respectively, significantly longer than for the typical vergence cases. The peak velocities were significantly lower than for the typical groups, at 7.3 ± 1.6 °/s and −5.0 ± 1.3 °/s, as might be expected from the significantly longer durations. The median latencies, on the other hand, were at the low end of the typical range, at 198 ± 25 ms and 200 ± 54 ms, indicating that there was nothing abnormal about the initiation processes. The midrate vergence parameters for the other two groups, those with midrate vergence in one direction but not the other, were not significantly different from those of the "both" group with two exceptions: (a) that the Normalized Temporal Asymmetry Index of divergence for the Midrate Convergence group was significantly higher than for the Midrate Both group (or for the Typical group), and (b) that the divergence duration for the Typical group was significantly shorter than for the Midrate Both group (though significantly longer than typical; see Table 1).

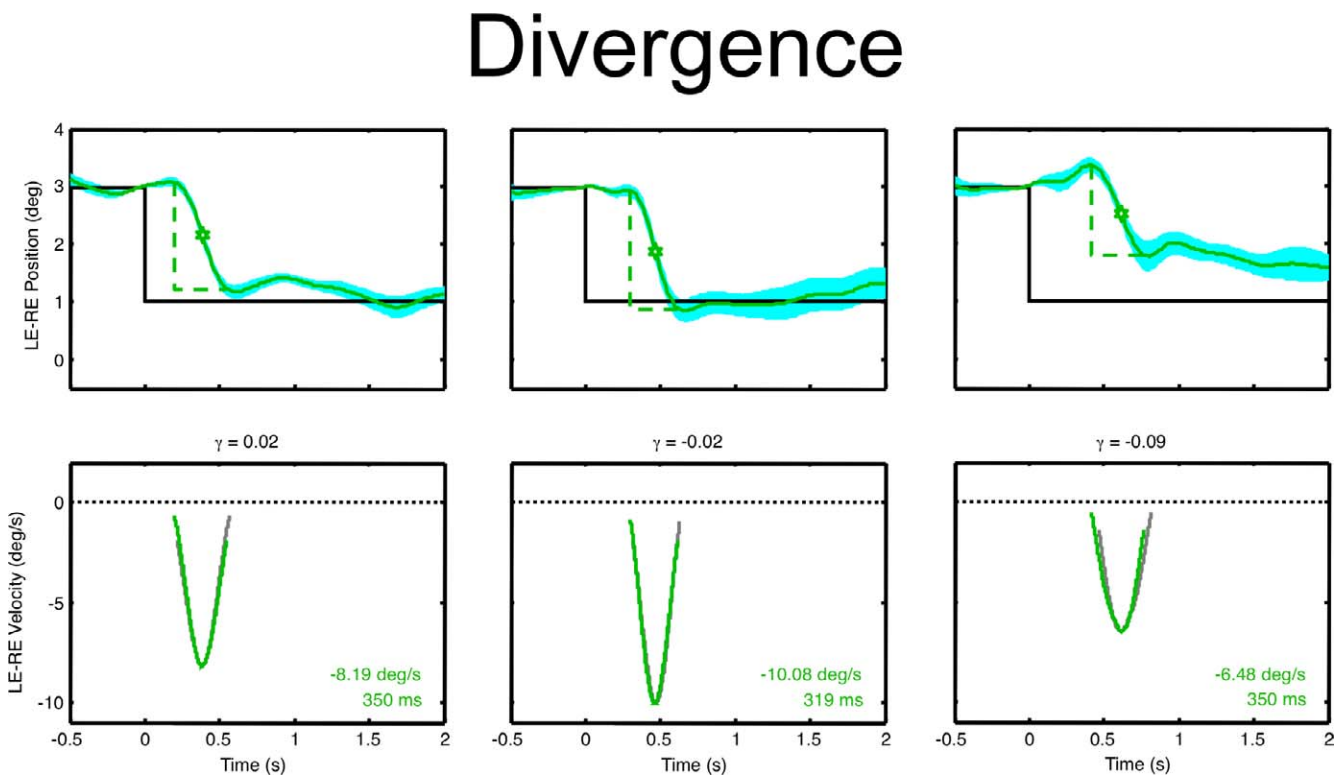

Figure 4. Examples of typical mean divergence responses and velocity traces for the same sample of typical subjects and plotted with the same conventions as Figure 3. Note the high degree of symmetry (near-zero asymmetry) of these typical examples of divergence.

Fast convergence/slow divergence

A subgroup of four individuals of this sample of nonTBI and nonstrabismic individuals had significantly slower vergence velocities (duration >945 ms) in either the convergence or divergence direction, although none of the sample had such slow responses in both directions. Since only one was a slow convergence case, that one does not qualify as a group, so we will only consider the group statistics for the slow divergence group (Figure 9). The convergence responses all fell in the typical range with a median latency of 300 ± 33 ms, a total duration of 316 ± 47 ms, and a typical peak velocity of 8.1 ± 1.1 °/s, all of which are similar to those of the typical group. The divergence responses had much slower responses of −3.8 ± 0.2 °/s with correspondingly longer durations of 1433 ± 516 ms, but typical latencies of 266 ± 95 ms.

The asymmetry assessment can be used to evaluate the hypothesis that the slow responses derive from a missing open-loop vergence system (see Discussion). The Normalized Temporal Asymmetry Index for the slow vergence responses were different for the convergence and divergence directions. In the convergence direction, the temporal asymmetry had the significantly negative value of $\gamma = -0.77 \pm 0.12$, while for the divergence direction, it was significantly positive at $\gamma = 0.67 \pm 0.24$ (and thus significantly higher than the typical value for divergence responses).

Anomalous compound divergence response

A substantial subgroup of nine individuals from this study (with no strabismus or reported traumatic brain injury [TBI]) showed a profound inability to make prompt disparity-driven divergence movements. Instead, the divergences were preceded by an initial convergence movement before the required divergence could take place. A raw 60 s vergence record of the 12 repeats of the convergence/divergence cycles for one such subject is shown in Figure 10. Note that, on this 60 s time scale, a saccade appears as an almost-vertical line in the trace, of which only three are visible (at 17, 36, and 52 s). The time courses of the anomalous vergence movements (the 2 s spike after each divergence stimulus) were nonsaccadic, with typical vergence dynamics (compare Figures 4, 5, and 7).

Averages of convergence and divergence responses over 12 events (see Methods) are plotted for three such subjects in Figure 11. The upper panels show that the convergence movements were within the typical range, with average latencies of 233 ± 14 ms, average durations of 317 ± 33 ms, and average velocities of 10.2 ± 1.2 °/s, none of which are significantly different

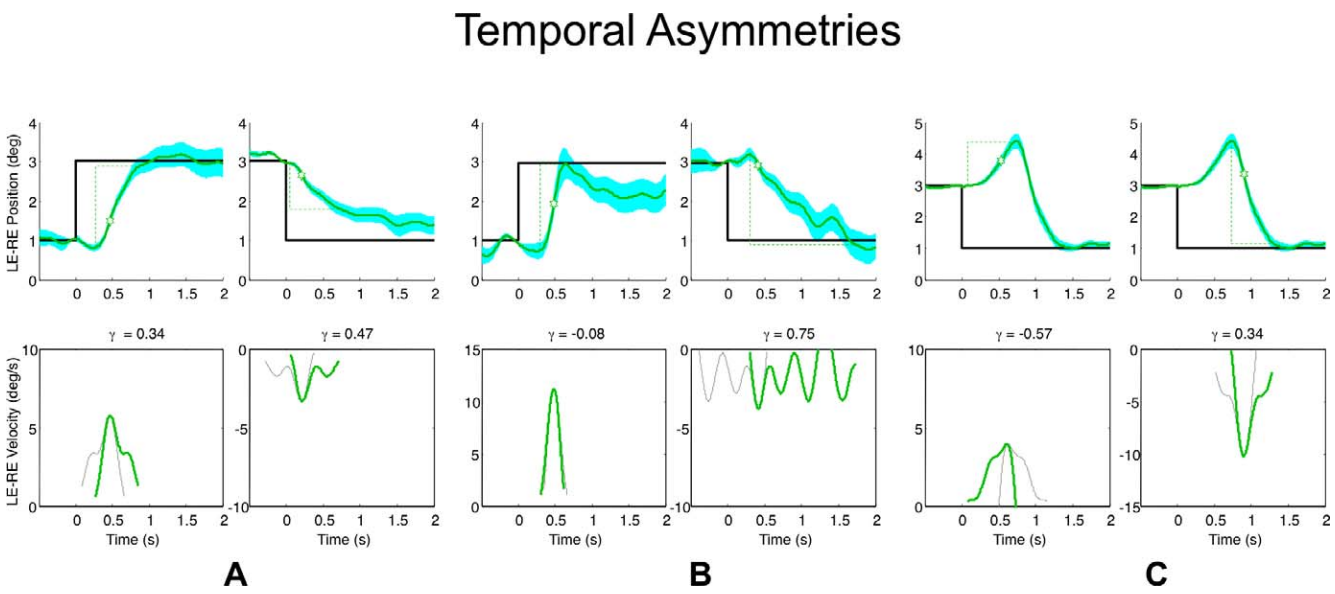

Figure 5. Examples of typical mean vergence responses and velocity traces for responses showing temporal asymmetries, plotted with the same conventions as Figure 3. (A) Convergence/divergence example from the Mid Both category. (B) Convergence/divergence example from the Slow Divergence Only category. (C) Divergence example from the Anomalous Compound Divergence category, analyzing the *same divergence response* for asymmetry separately in the two vergence directions.

from the typical values. The one parameter that did differ from the typical value was the temporal asymmetry of $\gamma = -0.84 \pm 0.15$.

Only when this convergence movement was completed did the eyes begin a divergence movement, usually immediately, but in one case after about a second's delay. In most cases, the divergence movement was longer than the normal divergence movement (554 ± 59 ms), sufficient to correct for the initial convergence to bring the eyes close to the required vergence posture at the typical vergence velocity (10.3 ± 2.9 °/s). These divergence movements had the largest values for temporal asymmetry of any vergence movement ($\gamma = 1.07 \pm 0.10$) indicating that they were on average close to the exponential form, which implies that the open-loop burst mechanism for divergence was inactivated in these individuals.

Discussion

As expected, the parameters of the typical vergence dynamics were clearly distinct from those of the saccadic system. If we consider the main sequence parameters for a saccade of the size of the present vergence movements (1° in each eye), it should have an expected duration of 20 ms and a velocity of 40 °/s (i.e., [200 °/s at 5°] / 5; Bahill, Clark, & Stark, 1975), while the typical vergence responses had the total duration of about 330 ms and a typical peak velocity in each eye of about 5 °/s for both the convergence and divergence directions (making a total vergence velocity of about 10 °/s). Thus, the vergence impetus is an order of magnitude less efficient than the saccadic, requiring a neural drive of about 20 times longer duration for the same amplitude of ocular displacement.[1]

We may expect the total energy needed to move each eye during the vergence movement to match that of saccadic eye movements. Accordingly, Gamlin and Mays (1992) found that medial rectus motorneurons in the midbrain had about the same integrated firing rate for saccadic and vergence movements of the same amplitude (4°). Interestingly, if we divide the saccadic velocity by the duration factor of 1/20 between saccades and vergences, this total energy hypothesis predicts a peak vergence velocity of 40/20 or 2 °/s, which is within a factor of 2 of the measured velocity. Since these motorneurons are the final common pathway for vergence and saccadic movement control, it makes sense that they should approximately adhere to the principle of the same total energy for a given eye movement, corresponding to the same total number of motorneuron spikes, despite the order of magnitude difference in the durations of the eye movements. The factor of 2 discrepancy for vergence relative to versional movements has been validated in direct muscle force measurements by Miller, Davison, and Gamlin (2011).

This energetics analysis leaves unanswered the question of why the duration of vergence movements should be so much longer than that of saccades. Presumably, it is not evolutionarily adaptive, because the slowness of vergence movements leaves significant

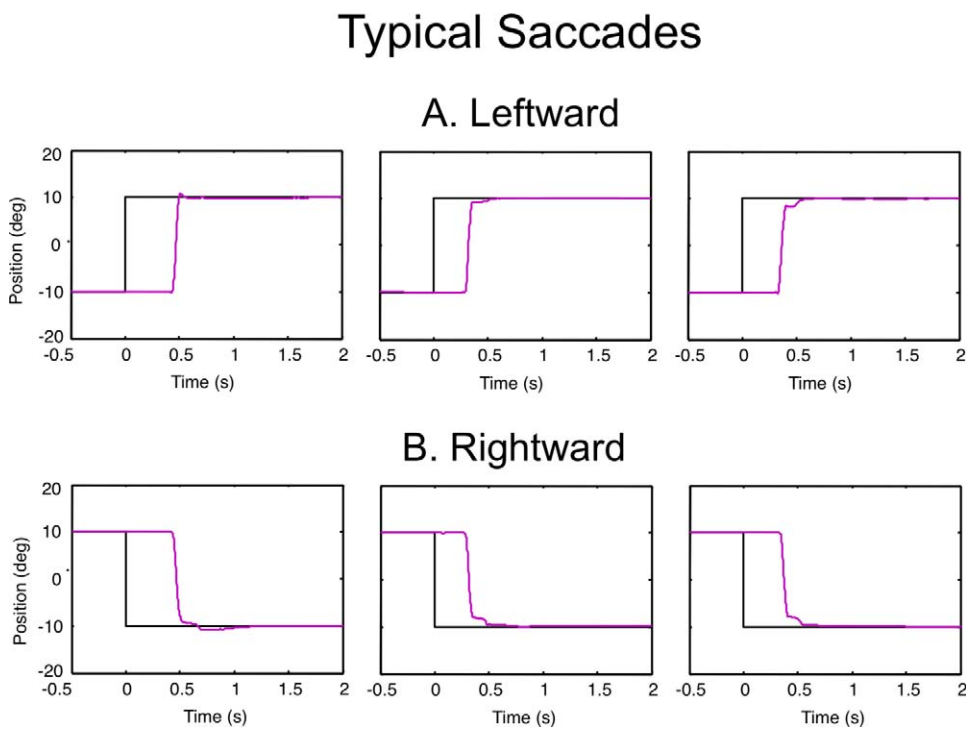

Figure 6. Position and velocity traces for leftward (upper panels) and rightward (lower panels) saccades averaged over 12 repeats for the subjects of Figures 3 and 4, showing the similar latencies and much faster time courses in relation to the vergence responses. As distinct from the vergence responses, the average saccades were obtained by first aligning the responses to the time of the initial deviation in amplitude from the baseline level, then setting the averaged waveform to the average latency for the group in each case. The *SEM* as a function of time is shown as the pink shaded region around the traces. The fact that this error region is barely visible reveals the high consistency of the saccadic performance on this compressed time scale, including the corrective saccade following the main saccade in most cases.

time for diplopia to become evident, which constitutes an undesirable failure of object coherency whenever large vergence movements are made. Instead, the slowness may derive from the physiology of the vergence control pathway, which is routed through the cerebellum rather than having a direct cortical/brainstem drive (Gamlin, 1999, 2002). Perhaps there is something in the integrative nature of cerebellar processing that requires a longer integration time than is available through the direct drive.

A key factor determining the slowness of vergence movements may well be the *inhibitory cross-coupling* via inhibitory burst neurons projecting via the medial longitudinal fasciculus (Strassman, Highstein, & McCrea, 1986) between the pairs of brainstem nuclei that control the ipsilateral motor neuron activation for both the lateral and medial rectus muscles (in the abducens and oculomotor nuclei, respectively). Presumably, this mechanism has evolved to inhibit the contralateral motorneurons to the corresponding muscle type in order to maximize the conjugate (parallel) pairing of the muscle activations required for saccades. Based on the relative durations of vergence and saccades, our specific proposal to account for the vergence dynamics would be that this contralateral inhibition is 90% effective or more, reducing the drive to the eye muscles for the symmetric activation of vergence movements to 10% or less of the level for the conjugate activation of saccades. As a result, the vergence system has to generate the drive for more than 10 times longer duration to achieve the same amplitude of symmetric vergence movements as conjugate saccades, in accord with the equivalence of the time-integrated signals reported by Gamlin and Mays (1992).

Waveform asymmetry analysis

This study introduces an index of temporal asymmetry that assesses the time-symmetry of the vergence velocity trace. This Normalized Temporal Asymmetry Index (symbolized as γ) was designed to evaluate interpreting the temporal symmetry results in terms of the dual-mode model reviewed in the Introduction, consisting of an *open-loop* burst component driving disparity vergence movements and a *closed-loop* vergence tracking component corresponding to the step-response minimization of the error signal generated by the disparity step. The closed-loop response to a step change in the tonic vergence signal is expected to

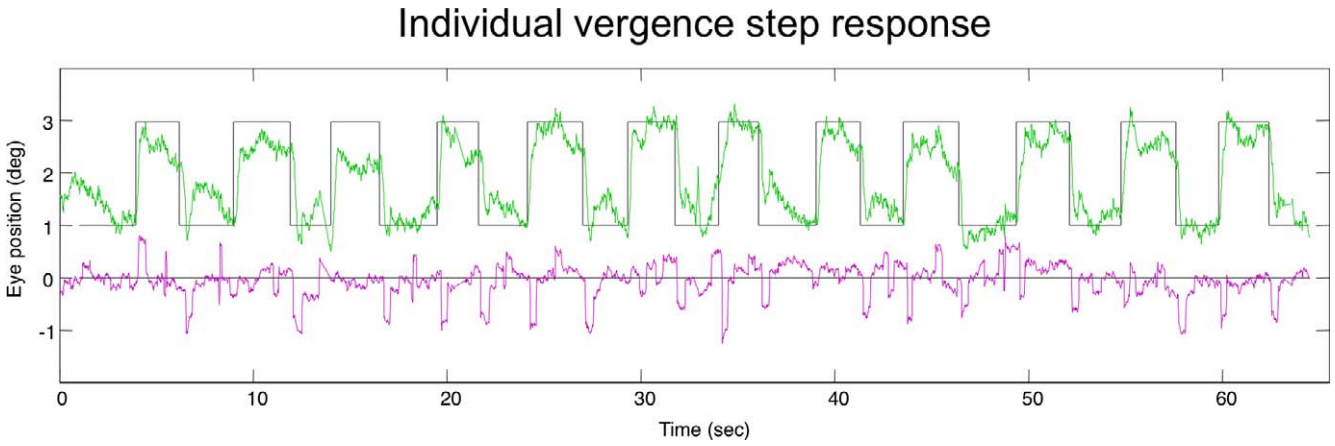

Figure 7. Example of the saccades occurring during a vergence stimulation series of 2° vergence jumps over 12 irregular cycles (black line). The vergence signal of the difference in position between the two eyes (green curve) shows an approximate match to the stimulus disparity with a delay of a few hundred ms. The mean position of the two eyes over time (magenta curve) shows general stability around the mean stimulus position (zero disparity) overlaid with microsaccades of less than 1° amplitude, which often occur at or after the completion of the vergence responses.

conform to an exponential time course (Robinson, 1971; Zee, Fitzgibbon, & Optican, 1992), while the open-loop response should have a more symmetric form derived from an open-loop burst mechanism (Gamlin & Mays, 1992). This model generates a Normalized Temporal Asymmetry Index value of zero for the time-symmetric open-loop response and $\gamma = 0.6$ – 0.9 for the exponential time course of closed-loop vergence eye movements. (The full asymmetry of $\gamma = 1.0$ would correspond to a pure exponential waveform under our recording conditions, but there is always some smoothing of the onset rise time that reduces the index below 1.0.)

The neural signals of saccadic burst neurons, on the other hand, are not exponential in form but are largely symmetrical in time in the amplitude range of most vergence movements (Becker & Fuchs, 1969; Becker, 1972; Bahill, Clark & Stark, 1975; Harris & Wolpert, 1998, 2006; Tanaka, Krakauer, & Qian, 2006; Xu-Wilson et al., 2009). Thus, although in principle saccades could have the exponential form characteristic of a closed-loop continuous feedback system, in practice they approximate the time-symmetric form of a fully ballistic drive for amplitudes within the vergence range of up to about 10°. Since the velocity/time function of an eye movement is a reasonable proxy for the underlying motorneuron burst that drives it (Figure 1), we may operationalize the symmetry of the velocity/time plot as an indicator of the degree of involvement of a neural burst of activation in the generation of any given eye movement. Based on the weight of the evidence, then, we will take perfect symmetry ($\gamma = 0$) as implying that the eye movement is entirely driven by a neural burst, while large asymmetries of $\gamma > 0.5$ will be assumed to imply a strong dominance by the closed-loop step response.

The above analysis provides an interpretation of the fact that the Normalized Temporal Asymmetry Index was approximately zero for the large group (62%) of typical convergence movements and for the smaller group (32%) of typical divergence movements, under the present stimulation conditions of a widefield texture stimulus at a vergence amplitude of 2°. This result may be taken as suggesting that the typical vergence system response may be driven by an open-loop burst mechanism comparable to that of the saccadic system, although weaker by an order of magnitude (as indicated by the respective durations of about 400 versus 20 ms, respectively, for typical vergence and saccadic movements at this amplitude; cf. Figures 3, 4, and 6). A similar result can be seen in the large vergence movements of Erkelens et al. (1989). This analysis through the Normalized Temporal Asymmetry Index thus tends to validate the dual-mode model not only for typical convergence, but also for typical divergence responses (Table 1). Atypical forms with high positive and even high negative symmetries are encountered in some of the groups, but in the typical range defined by durations <450 ms, the temporal asymmetry remains close to zero, similar to that of the typical convergence responses. Thus, we infer that the optimal divergence response is not fundamentally different in control dynamics from the convergence response—both are well described by the dual-mode theory of a combination of fast open-loop and more accurate closed-loop control mechanisms.

Latencies

Before discussing the detailed properties of each of the other subgroups, a general comment on the

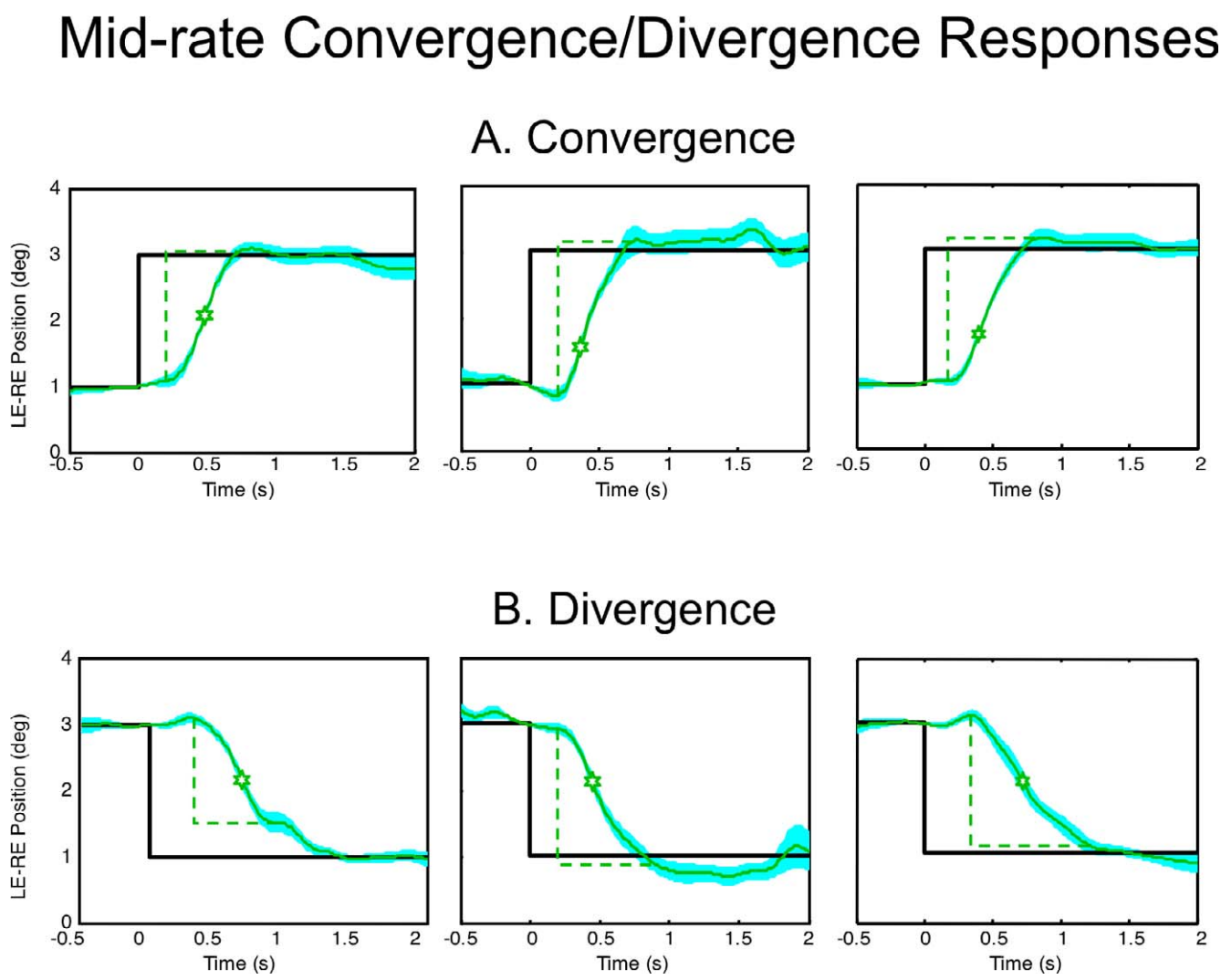

Figure 8. Examples of typical mid-rate convergence responses and velocity traces plotted with the same conventions as Figure 3.

latencies is in order. For the four groups with typical response behavior, the latencies had similar values, of 252 and 317 ms and 275 and 283 ms, for the pairings of convergence and divergence movements, respectively (see Table 1). Similarly the midrate latencies, though shorter (at 198 and 200 ms and 144 and 211 ms, respectively), failed to exceed the statistical criteria for significant differences. Thus, the atypical groups did not have significantly longer latencies than the typical vergence group, all being between about 150 and 320 ms, with *SEM*s of 25–50 ms. Similar results are evident for the Slow Divergence group. This relative invariance in the latencies gives assurance that the atypical groups were not suffering from the kinds of general neurological problems or impaired cortical decision-making capabilities that affect saccadic latencies, suggesting instead that the slowing and anomalous behavior in theses cases was specific to their vergence control mechanisms once the movement was triggered.

Slow divergence with typical convergence movements

A significant subgroup of the participants had slow divergence dynamics in conjunction with typical convergence dynamics. One subject had the reverse combination: typical divergence dynamics combined with slow convergence dynamics. The same diversity was reported among the small group of four subjects studied by Erkelens et al. (1989), two having faster convergence and two having faster divergence for the extreme vergence movements studied there. The implication is clear, therefore, that the convergence and divergence dynamics are controlled by distinct brainstem mechanisms, either of which can show abnormalities while the other is within the normal range (in the sense that they form a large cluster with low variability of the convergence dynamics Figure 2). The fact that for the majority of the participants, the convergence mechanism is within a range defined as "normal" implies that it is less

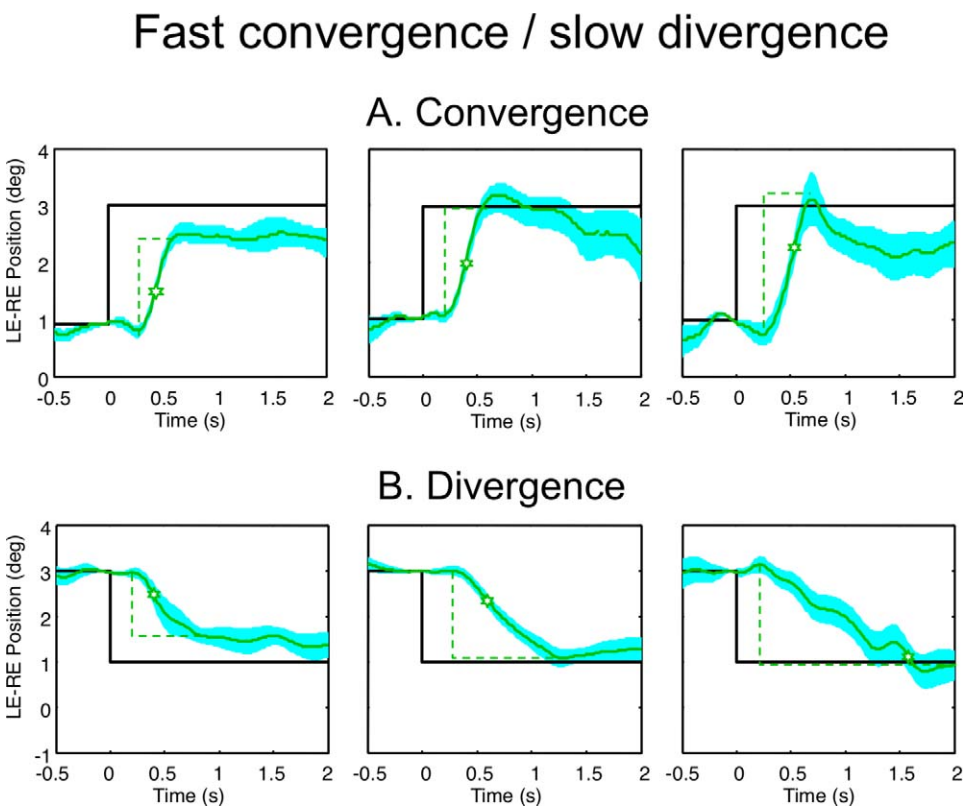

Figure 9. Examples of vergence movements from three individuals with typical convergence responses but slow divergence movements, plotted with the same conventions as Figure 3. The vergence movements are again repeatable to within a standard error of relatively few arcmin.

susceptible to neurological disruption than the divergence mechanism (whether from developmental or idiopathic etiologies). The two vergence directions both appear to be capable of similar dynamics (i.e., when both are in the typical range), but differences in dynamics in the nontypical cases suggest that they have independent control mechanisms that are differentially susceptible to prevailing forms of disruption.

The high temporal asymmetry indices for the slow divergence movements suggest that they conform to the closed-loop feedback control (in contrast to the close-to-zero values for the typical divergence movements, implying open-loop control). Thus, the presence of two reliable forms of temporal asymmetry may be regarded as confirmation of the presence of a distinct mechanism underlying the slow disparity vergence movement behavior. The asymmetric variety may represent the operation of the closed-loop feedback mechanism in cases where the open-loop mechanism fails to operate, for either ontogenetic or adventitious postdevelopmental reasons.

Curiously, the convergence responses in the Slow Divergence group exhibit the unexpected behavior of a negative asymmetry, implying a gradual onset of the velocity profile rather than the gradual offset predicted by the closed-loop hypothesis. Similar behavior is seen below for the Anomalous Compound Divergence group and can be interpreted as a form of nonlinear neural recruitment, generating progressive acceleration of the movement toward the end of the response.

Anomalous compound divergence response

In the present sample, a substantial subpopulation of about 18% of the subjects (with no strabismus on a cover test or reported history of TBI) showed an inability to make prompt divergence movements. Instead, the divergence stimulus evoked the compound response of an initial *convergence* movement comparable to the response to the convergence stimulus, followed by a larger divergence movement bringing the eyes approximately to the intended target disparity despite having initially moved in the wrong direction (Figures 10 and 11). We have not been able to identify any previous reports of this type of oculomotor anomaly. For example, nothing of this kind is mentioned in the National Eye Institute's Classification of Eye Movement and Strabismus (CEMAS) Report (CEMAS Working Group, 2001). Even in cases of strabismus and related oculomotor disorders (Kenyon, Ciuffreda & Stark, 1980) or TBI (Thiagarajan, Ciuffreda, & Ludlam, 2011), no such behavior has been reported.

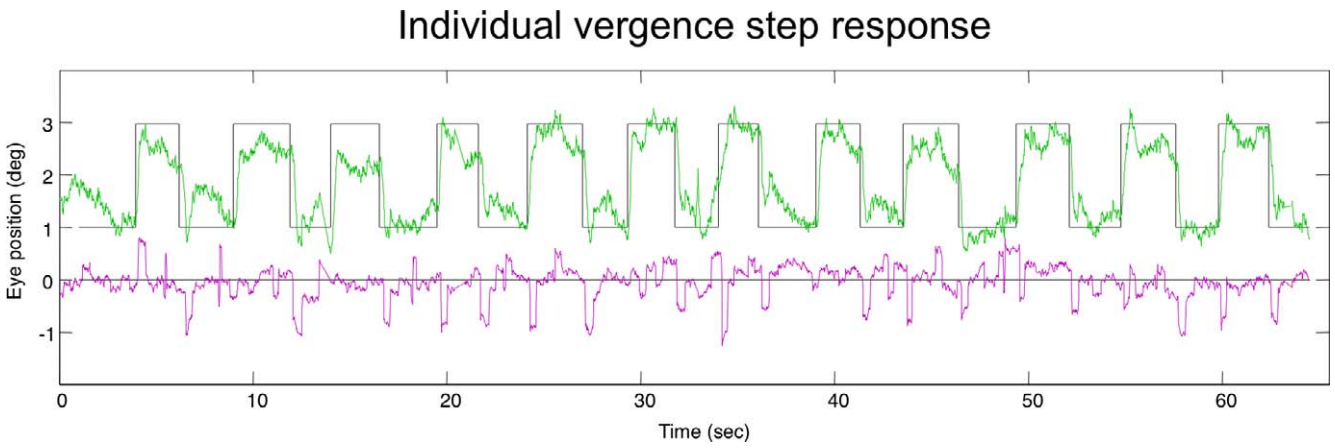

Figure 10. An individual 60 s time series of step disparity vergence movements in one case, showing repeated anomalous compound responses to the divergent disparity stimulus, with the divergence movement preceded consistently by a full convergent movement in the opposing direction. Black trace is the stimulus disparity to be tracked. Note that none of these anomalous vergence movements had a significant contribution from differential (asymmetrical) saccades, which appear as fast spikes on this time scale. The saccades on cycles 3, 7, and 10 are all bidirectional, with little net effect on the vergence angle.

It is worth considering the timing of the anomalous convergence movement in detail. Note that the anomalous initial convergence dynamics were in the typical range for convergent disparity stimuli (~300 ms in duration; see Table 1) and that the individual responses were usually similar for all individual trials as implied by the narrow error limits in Figure 11. This result makes clear that the anomalous convergence movement in the compound movement complexes was not a saccade of some kind (which should have had a duration of only about 20 ms for this amplitude), but was a genuine convergence movement.

Why should a divergence stimulus evoke an initially convergent eye movement? Two possible explanations suggest themselves: disparity-change uncertainty and an inverse priming strategy.

Disparity-change uncertainty hypothesis

(a) If disparity changes were occurring unpredictably, and if the convergence and divergence subsystems were in competition with each other but with convergence slightly predominating, it might be supposed that disparity change would tend to activate the dominant vergence direction in *both* directions before the sign of disparity change was processed, hence activating a *convergent* response in both cases. Such a response would be appropriate for the convergence stimulus but would need correcting when the stimulus was a *divergent* disparity. Thus, under this disparity-change uncertainty hypothesis, the anomalous convergence response to divergent stimuli would be a reflection of the initial uncertainty as to the direction of the disparity change. This hypothesis would also predict an appropriate direction for the convergence response, and is hence consistent with both directions of vergence behavior in these individuals.

However, although the *timing* of each stimulus event was jittered over a 1 s range in our paradigm, the *sign* (and amplitude) of each disparity change (i.e., convergent or divergent) was perfectly predictable throughout the sequence. Each convergence stimulus was followed by a divergence stimulus, and vice versa. Thus, on a cognitive level at least, the subject had every opportunity to gear up for a divergence response to each divergence stimulus. Of course, this does not necessarily imply that the directional information was available to the burst neurons generating the initial burst of activation to drive the fast onset of the divergence movement, but the system had the ~300 ms latency period before the movement took place to compute the sign of the disparity (which is about the length of the perceptual integration time for disparity processing; Tyler & Julesz, 1980; Tyler, 1983), so there is little reason to suppose that the disparity information would not have been available. The disparity-change uncertainty explanation thus seems relatively implausible.

Inverse priming strategy hypothesis

(b) The other hypothesis to account for these anomalous compound divergence movements is an "inverse priming" strategy for divergence initiation. This hypothesis is based on the concept that there is some impediment to initiating a divergence movement and that making a convergence movement primes the system to a state of readiness to be able to implement the required divergence movement. This priming mechanism could be similar to the priming of secondary express saccades (Fischer & Ramsperger, 1984; Takagi, Frohman, & Zee, 1985; Fischer, Weber &

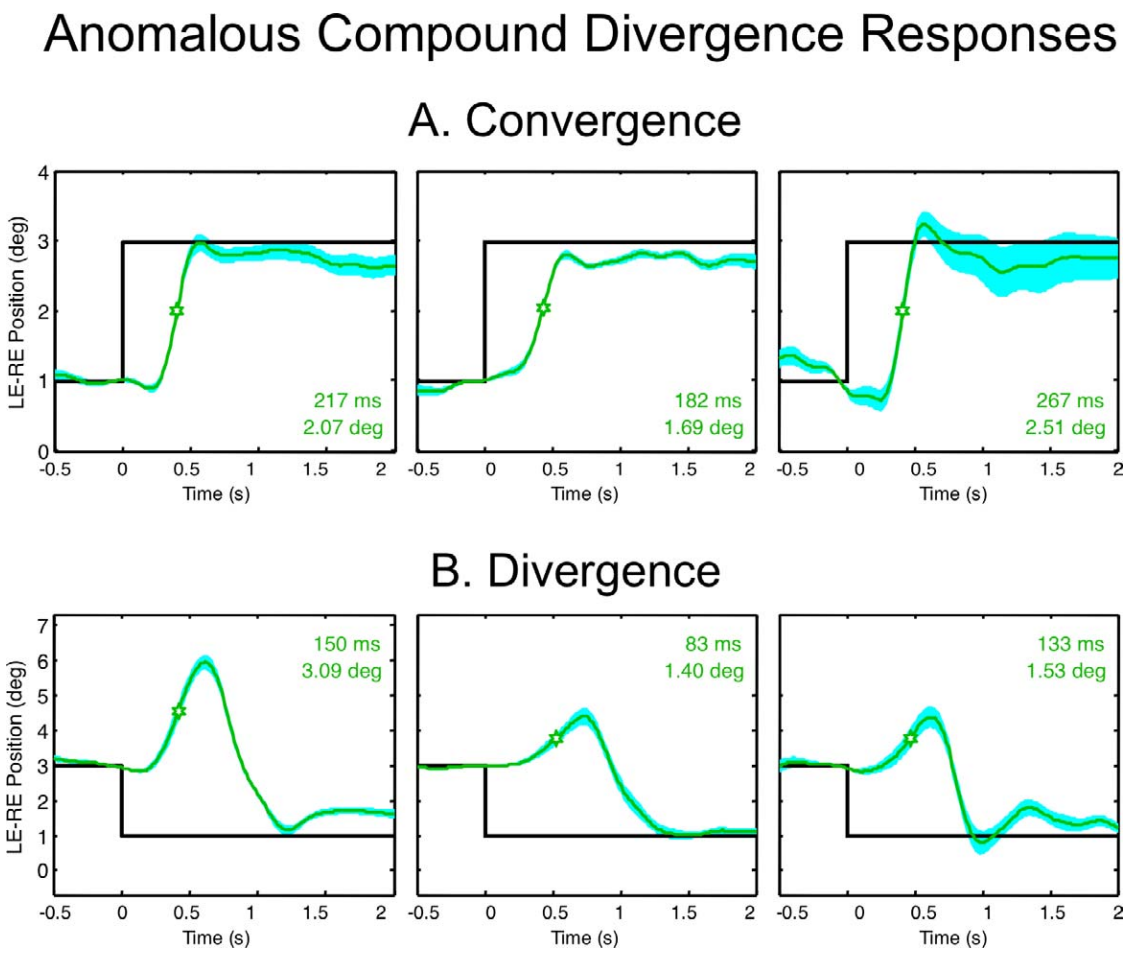

Figure 11. Averaged convergence and divergence movements (green curves) from three individuals with anomalous compound divergence behavior (plotted with the same conventions as Figure 3). (A) Examples of typical average convergence responses. (B) Average divergence movements for the same three individuals. Note the low error across the repeated examples of the divergence movements, implying a nonadapting stereotypical behavior.

Biscaldi, 1993; Coubard, Daunys, & Kapoula, 2004; Van Koningsbruggen & Rafal, 2009). On this hypothesis, these Anomalous Compound Divergence movements are manifesting some deficiency in the divergence system that derives in the ability to *initiate* divergence movements. Making the anomalous convergence movement would release the system from this initiation defect and allow the divergence mechanism to operate normally from that point onwards.

It should be stressed that the variability of the compound divergence movements was of the order of 0.1° or less, implying that the vergence behavior was highly consistent across the 12 stimulus repeats despite the recurring knowledge that the initial movement was incorrect each time. Thus, on balance the repeatability of the anomalous compound behavior seems to favor the inverse priming hypothesis, but the idea that the disparity sign signal is not reaching the brainstem control nuclei in these individuals cannot be excluded.

Once primed, the divergence movement typically has sufficient amplitude to overcome the convergence error and to reach the appropriate divergence angle for the target. However, the temporal asymmetries for these compound divergence movements were large, close to the exponential prediction, implying that the divergence systems the open-loop burst mechanism in these individuals was lacking or weak. The convergence responses in this group, however, had the opposite character with a strong *negative* asymmetry, implying gradual build up of the velocity with a more rapid response offset. This form of asymmetry is consistent with a progressive recruitment of activation following the response onset, perhaps by positive feedback (as opposed to the progressive decay of the activation implied by negative feedback).

Saccades and vergence/saccadic interactions

The saccade measures of Figure 6 are included to emphasize the profound difference in time scale

between vergence and saccadic step responses. Although it was not the main topic of this paper, we note in passing that the 20° saccades analyzed for the "typical" subpopulation had a highly stereotypical behavior, with each subject showing some stable degree of undershoot of the initial saccade, followed by a stereotypical corrective saccade to reach the repetitively presented target (Figure 6). Despite performing 24 back-and-forth movements between the same two targets, there was no tendency to correct the initial saccadic amplitude to reach the target in one shot. This result is consistent with the initial reports of corrective saccades (Becker & Fuchs, 1969; Becker, 1972), and may reflect a habitual tendency to perform combined eye and head movements for large changes in stimulus position, even though head movements were eliminated by our chin and forehead restraints.

With respect to saccadic/vergence interactions, we found no evidence of a consistent saccadic component in the averaged vergence responses (see Figure 7). Although there were often refixations within the range of the 1.25° fixation target, they did not occur at a systematic timing relative to the vergence onset, and therefore could not be analyzed in a consistent fashion. The symmetric disparity vergence behavior for the present population is best characterized as being fully controlled by the vergence system with no significant interaction with the saccadic system.

Conclusion

This survey of vergence dynamics in a nonacademic population sample showed a remarkable variety of vergence behaviors. The population was subdivided into nine main groups according to whether the convergence/divergence dynamics were typical, moderately slowed, or severely slowed: typical vergence dynamics, atypically slow dynamics of either convergence and divergence or both, moderately slowed dynamics of either convergence and divergence (with severe slowing of the other) or both, anomalous compound divergence behavior, and mixed or inconsistent vergence responses.

The results could be interpreted in terms of the dual-mode analysis of vergence responses consisting of a combination of an open-loop burst response and a closed-loop feedback response in each vergence direction. The vergence dynamics for disparity jumps of a large textured field have a typical time course consistent with predominant control by the open-loop vergence-specific burst mechanism, although the subgroups showed radically different vergence behaviors. Some subjects showed markedly slow divergence responses, implying that the two vergence directions have separate control mechanisms, most likely each with separate open-loop burst and closed-loop feedback subsystems (though cases of slow convergence are rare). A further type of behavior in a distinct subgroup was a compound divergence response consisting of an initial convergence movement followed by a large corrective divergence movement, in which both components have normal time courses implying open-loop burst control.

Thus, the variety of human vergence dynamics contributes substantially to the understanding of the oculomotor control mechanisms underlying the generation of these movements.

Acknowledgments

Supported by CDMRP DM 102524.

Commercial relationships: none.
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Footnote

[1] We note that the extreme values of up to an average of about 70 °/s (35 °/s in each eye) for the maximum vergence velocities reported by Erkelens et al. (1989) were obtained for large vergence angles under full cue conditions. In terms of the "main sequence" concept, these velocities were again about a factor of 20 lower than the maximum reported saccadic velocities of 600–800 °/s and had durations of the order of a factor of 10 longer, at ~500 ms.

References

Bahill, A. T., Clark, M., & Stark, L. (1975). The main sequence, a tool for studying human eye movements. *Mathematical Biosciences*, *24*, 191–204.

Becker, W. (1972). The control of eye movements in the saccadic system. *Proceedings of the Symposium,* Freiburg im Breisgau, West Germany; Switzerland; July 20–22, 1971. pp. 233–243.

Becker, W., & Fuchs, A. F. (1969). Further properties of the human saccadic system: Eye movements and correction saccades with and without visual fixation points. *Vision Research*, *9*, 1247–1257.

CEMAS Working Group. A classification of eye

movement abnormalities and strabismus (CEMAS). In *The National Eye Institute Publications*. (www.nei.nih.gov). 2001. National Institutes of Health, National Eye Institute, Bethesda, MD.

Collewijn, H., Erkelens, C. J., & Steinman, R. M. (1995). Voluntary binocular gaze-shifts in the plane of regard: Dynamics of version and vergence. *Vision Research*, *35*, 3335–3358.

Coubard, O., Daunys, G., & Kapoula, Z. (2004). Gap effects on saccade and vergence latency. *Experimental Brain Research*, *154*, 368–381.

Coubard, O. A., & Kapoula, Z. (2008). Saccades during symmetrical vergence. *Graefes Archive for Clinical and Experimental Ophthalmology*, *246*, 521–536.

Erkelens, C. J. (2011). A dual visual-local feedback model of the vergence eye movement system. *Journal of Vision*, *11*(10):21, 1–14, http://www.journalofvision.org/content/11/10/21, doi:10.1167/11.10.21. [PubMed] [Article]

Erkelens, C. J., Van Der Steen, J., Steinman, R. M., & Collewijn, H. (1989). Ocular vergence under natural conditions. I. Continuous changes of target distance along the median plane. *Proceedings of the Royal Society of London B: Biological Science, 236*, 417–440.

Fischer, B., & Ramsperger, E. (1984). Human express saccades: Extremely short reaction times of goal directed eye movements. *Experimental Brain Research*, *57*, 191–195.

Fischer, B., Weber, H., & Biscaldi, M. (1993). The time of secondary saccades to primary targets. *Experimental Brain Research*, *97*, 356–360.

Gamlin, P. D. (1999). Subcortical neural circuits for ocular accommodation and vergence in primates. *Ophthalmic & Physiological Optics*, *19*, 81–89.

Gamlin, P. D. (2002). Neural mechanisms for the control of vergence eye movements. *Annals of the New York Academy of Sciences*, *956*, 264–272.

Gamlin, P. D., & Mays, L. E. (1992). Dynamic properties of medial rectus motoneurons during vergence eye movements. *Journal of Neurophysiology*, *67*, 64–74.

Harris, C. M., & Wolpert, D. M. (1998). Signal-dependent noise determines motor planning. *Nature*, *394*, 780–784.

Harris, C. M., & Wolpert, D. M. (2006). The main sequence of saccades optimizes speed-accuracy trade-off. *Biological Cybernetics*, *95*, 21–29.

Horng, J. L., Semmlow, J. L., Hung, G. K., & Ciuffreda, K. J. (1998). Dynamic asymmetries in disparity convergence eye movements. *Vision Research*, *38,* 2761–2768.

Hung, G., Semmlow, J. L., & Ciuffreda, K. J. (1986). A dual-mode dynamic model of the vergence eye movement system. *IEEE Transactions on Biomedical Engineering*, *33*, 1021–1028.

Hung, G. K., Ciuffreda, K. J., Semmlow, J. L., & Horng, J. L. (1994). Vergence eye movements under natural viewing conditions. *Investigative Ophthalmology & Visual Science*, *35*: 3486–3492, http://www.iovs.org/content/35/9/3486. [PubMed] [Article]

Kenyon, R. V., Ciuffreda, K. J., & Stark, L. (1980). Unequal saccades during vergence. *American Journal of Optometry & Physiological Optics*, *57*, 586–594.

Krishnan, V. V., & Stark, L. (1977). A heuristic model for the human vergence eye movement system. *IEEE Transactions on Biomedical Engineering*, *24*, 44–49.

Mays, L. E., Porter, J. D., Gamlin, P. D., & Tello, C. A. (1986). Neural control of vergence eye movements: Neurons encoding vergence velocity. *Journal of Neurophysiology*, *56*, 1007–1021.

Miller, J. M., Davison, R. C., & Gamlin, P. D. (2011). Motor nucleus activity fails to predict extraocular muscle forces in ocular convergence. *Journal of Neurophysiology*, *105*, 2863–2873.

Rashbass, C., & Westheimer, G. (1961). Disjunctive eye movements. *Journal of Physiology*, *159*, 339–360.

Robinson, D. A. (1971). Models of oculomotor neural organization. In P. Bach-y-Rita, C. C. Collins, & J. Hyde (Eds.), *The control of eye movements* (pp. 519–538). New York: Academic Press.

Semmlow, J. L., Hung, G. K., & Ciuffreda, K. J. (1986). Quantitative assessment of disparity vergence components. *Investigative Ophthalmology & Visual Science*, *27*: 558–564, http://www.iovs.org/content/27/4/558. [PubMed] [Article]

Semmlow, J. L., Hung, G. K., Horng, J. L., & Ciuffreda, K. (1993). Initial control component in disparity vergence eye movements. *Ophthalmic & Physiological Optics*, *13*, 48–55.

Semmlow, J. L., & Yuan, W. (2002). Components of disparity vergence eye movements: Application of independent component analysis. *IEEE Transactions on Biomedical Engineering*, *49*, 805–811.

Schwarz, G. E. (1978). Estimating the dimension of a model. *Annals of Statistics*, *6*, 461–464.

Strassman, A., Highstein, S. M., & McCrea, R. A. (1986). Anatomy and physiology of saccadic burst neurons in the alert squirrel monkey. II. Inhibitory

burst neurons. *Journal of Comparative Neurology*, *249*, 358–380.

Takagi, M., Frohman, E. M., & Zee, D. S. (1985). Gap-overlap effects on latencies of saccades, vergence and combined vergence-saccades in humans. *Vision Research*, *35*, 3373–3388.

Tanaka, H., Krakauer, J. W., & Qian, N. (2006). An optimization principle for determining movement duration. *Journal of Neurophysiology*, *95*, 3875–3886.

Thiagarajan, P., Ciuffreda, K. J., & Ludlam, D. P. (2011). Vergence dysfunction in mild traumatic brain injury (mTBI): A review. *Ophthalmic & Physiological Optics*, *31*, 456–468.

Tyler, C. W. (1983). Sensory processing of binocular disparity. In C. M. Schor & K. J. Ciuffreda (Eds.), *Vergence eye movements: Basic and clinical aspects* (pp.199–295). London: Butterworths.

Tyler, C. W., & Julesz, B. (1980). On the depth of the cyclopean retina. *Experimental Brain Research*, *40*, 196–202.

Van Koningsbruggen, M. G., & Rafal, R. D. (2009). Control of oculomotor reflexes: Independent effects of strategic and automatic preparation. *Experimental Brain Research*, *1192*, 761–768.

Xu-Wilson, M., Zee, D. S., & Shadmehr, R. (2009). The intrinsic value of visual information affects saccade velocities. *Experimental Brain Research*, *196*, 475–481.

Zee, D. S., Fitzgibbon, E. J., & Optican, L. M. (1992). Saccade-vergence interactions in humans. *Journal of Neurophysiology*, *68*, 1624–1641.