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The sub-clinical see-saw nystagmus embedded in infantile nystagmus ☆

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Abstract

A transient, decompensated vertical phoria in an individual with infantile nystagmus syndrome (INS) resulted in two images that oscillated vertically—a diplopic oscillopsia. Ocular motor studies during the vertical oscillopsia recreated by vertical prisms, led to the identification of a sub-clinical see-saw nystagmus (SSN), present under the prism-induced diplopic condition. Retrospective analysis of ocular motor recordings made prior to the above episode of vertical diplopia revealed the presence of that same sub-clinical SSN. The SSN had not been detected previously despite extensive observations and recordings of this subject's pendular IN over a period of forty years. Three- dimensional search-coil data from fourteen additional INS subjects (with pendular and jerk waveforms) confirmed the existence of sub-clinical SSN embedded within the clinically detectable horizontal-torsional IN in seven of the fifteen and a sub-clinical, conjugate, vertical component in the remaining eight. Unlike the clinically visible SSN found in achiasma, the cause of this sub-clinical SSN is hypothesized to be due to a failure of the forces of the oblique muscles (responsible for the torsional component of the IN) to balance out the associated forces of the vertical recti; the net result is a small, sub-clinical SSN. Thus, so-called "horizontal" IN is actually a horizontal-torsional oscillation with a secondary, sub-clinical SSN or conjugate vertical component. The suppression of oscillopsia by efference copy in INS appears to be accomplished for each eye individually, even in a binocular individual. However, failure to fuse the two images results in oscillopsia of one of them.
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Keywords: Infantile ("congenital") nystagmus; See-saw nystagmus; Oscillopsia; Diplopia

1. Introduction

The infantile nystagmus syndrome (INS) (CEMAS Working Group, 2001) contains pathognomonic waveforms that usually include a horizontal-torsional oscillation of the eyes (Averbuch-Heller, Dell'Osso, Leigh, Jacobs, & Stahl, 2002) that, in its purest form: is not associated with an afferent deficit or strabismus; is not accompanied by oscillopsia; and does not disturb binocular single-vision. The horizontal and torsional components usually have the same waveform and are phase locked. Diplopia is the appearance of two *static* images of the visual scene, usually caused by the misalignment of the eyes in one or more planes. This study of the relationship between see-saw nystagmus (SSN) and IN was prompted by a *unique* observation made by an observer with INS (S1) during a spontaneous, transient episode of a decompensated vertical phoria—a transient period of hypertropia (Dell'Osso, Daroff, & Tomsak, 2001).

The above observations of disjunctive vertical oscillopsia under both spontaneous (described in more detail below) and induced vertical tropias suggested several hypothetical mechanisms that might be responsible. The following were considered: (1) there was a singular occurrence of

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a vertical component in the IN of the deviated eye that was *uncorrected* by efference copy; (2) there was either a rivalry or suppression mechanism (modulated by the IN oscillation) between the two disparate images; and (3) there was a transient uniocular vertical nystagmus that was responsible for, or coincided with, the sudden loss of vertical fusion and the vertical oscillopsia of the diplopic image.

To investigate these possibilities, we studied the eye-movement data of S1, taken during fixation of a stationary target under different conditions of induced vertical diplopia. Our startling findings prompted us to reexamine eye-movement data from this subject taken prior to the episode of diplopia (e.g., in both 1996 and 1984) during fixation but without induced vertical diplopia. Finally, to investigate the generality of those findings (between subjects and waveforms), we retrospectively studied fixation data from fourteen additional subjects with clinically detectable horizontal-torsional IN (i.e., no vertical components were visible).

2. Case report

The initial subject of this study (S1) was a 60 year-old male with hereditary IN, best-corrected visual acuity of 20/25 OU, 60 arc seconds of stereopsis, a small vertical phoria, and no afferent deficits. Prior to the events in the above report, he had never experienced a migraine headache, visual aura, or diplopia. An MRI scan of the head, performed at that time, was normal. Quantitative oculographic recordings made over a 40-year period, using infrared, search-coil, and video recording systems, had identified the subject's IN as horizontal-torsional; no vertical component was noted.

2.1. Observation

"A sudden onset of vertical diplopia occurred while the subject rapidly descended a flight of stairs. The lower, diplopic image of the visual field, coming from the right eye, appeared to be oscillating vertically over a static image of the visual field from the left eye. Thus, the subject went from his normal stationary, binocular perception of the world to one with diplopia and uniocular vertical oscillopsia. At the bottom of the stairs, he looked at a blank wall with a room number sign. The 2 in. high by 6 in. wide sign appeared as two signs, vertically displaced by ~4 in., leaving a 2-in. separation between them, with the lower one oscillating vertically at ~2–3 Hz with an estimated peak-to-peak amplitude of <5° (based on the distances involved). During the oscillation, the lower image did not overlap the upper. Attempts to fuse the images brought them closer by raising the lower image but did not achieve single-vision. Pushing the right eye slightly downward by applying pressure on the upper eyelid also lessened the vertical diplopia. Occlusion of either eye eliminated both diplopia and oscillopsia. The spontaneous, vertical, diplopic oscillopsia lasted ~3 min and has not reoccurred" (Dell'Osso et al., 2001).

3. Methods

3.1. Eye movement recording

Three different methods of recording eye movements were used to record S1 over the 22 years of data recorded from this subject. The initial eye movements from S1, made during the fixation experiments using a vertical prism, were recorded using both infrared reflection and a high-speed digital video system. Some of the eye movements from the prospective analysis of S1 and all of the data from the retrospective analysis of S1–S15 were recorded using the magnetic search-coil technique in either the Daroff-Dell'Osso Ocular Motility Lab (S1–S6, horizontal, vertical, and torsional), the Laboratory of Sensorimotor Research, NIH-NEI (S7–S15, horizontal and vertical), or the Laboratory of Dr. R.M. Steinman (S1, horizontal and vertical). As has been our experience, all techniques yielded equivalent results in the planes measured.

3.2. Data analysis

All data were analyzed using the MATLAB (The MathWorks, Natick MA) computing environment and the OMtools software developed in our laboratory and available for downloading at www.omlab.org.

4. Results

4.1. Induced vertical diplopia

Vertical prisms were used to induce vertical diplopia while S1 was fixating a target resembling a vertically elongated "+" sign (see Fig. 1). Both the vertical diplopia and vertical oscillopsia described above were induced with either a base-down or base-up prism before either eye; characteristics of the oscillopsia depended on the point of fixation. During fixation at the point of intersection of the vertical line with either of the horizontal lines (real or vertically displaced) of the target, vertical oscillopsia of the fixated horizontal line was perceived. However, when fixating at a point on the vertical line lying between the two horizontal lines, a counterphase vertical oscillation of both horizontal lines was perceived. When these experiments were repeated while recording the movements of the two eyes in both planes, we discovered what we thought to be an "induced" SSN. As the infrared-reflection data in Fig. 2 show, under the conditions of vertical diplopia induced by a 4D base-down prism placed in front of the left eye, there was an "induced" SSN of approximately 0.5° peak-to-peak

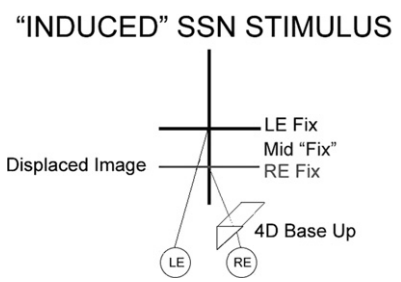

Fig. 1. The use of vertical prisms to create vertical diplopia and induced oscillopsia and see-saw nystagmus (SSN) in infantile nystagmus. Shown is the diplopic condition induced by a 4D base-up prism in front of the right eye and the three fixation conditions.

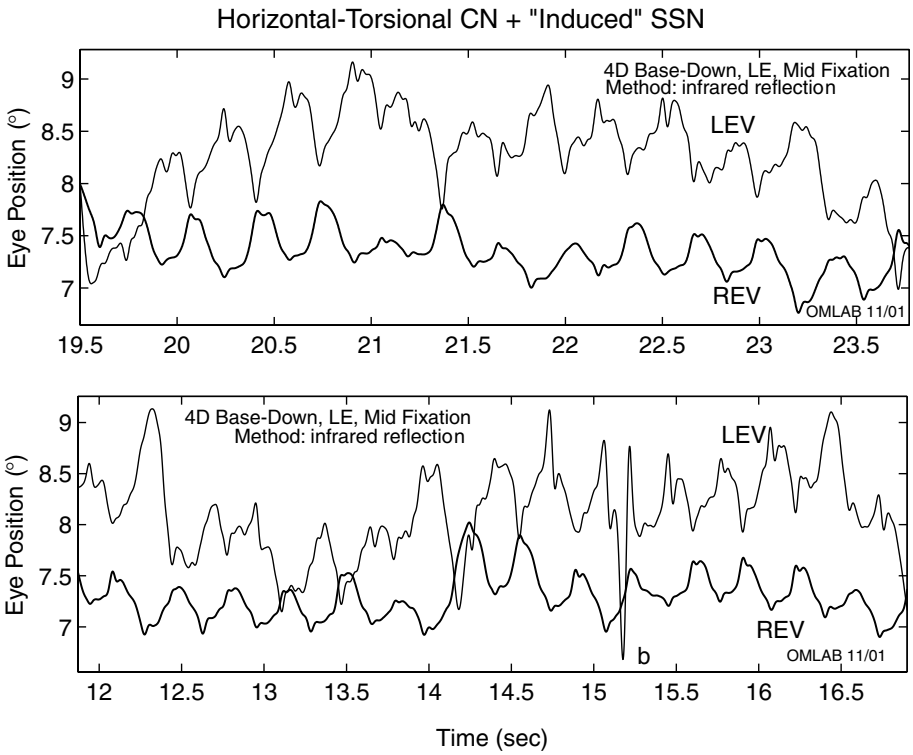

Fig. 2. Right and left vertical eye movements of S1 during two intervals of induced diplopia using a 4D base-down prism in front of the left eye while fixating midway between the two horizontal lines. The data in both panels were taken using infrared reflection. The "induced" see-saw nystagmus (SSN) was phase locked with and had the same pseudopendular with rightward foveating saccades waveform as the horizontal component (not shown) of the IN. In this and subsequent figures, REV, right eye vertical; LEV, left eye vertical. In this and the following figures, upward movement of the trace is right, up, or clockwise from the point of view of the subject and the heavier traces in each plane are from the right eye.

amplitude. In Fig. 3, where a 4D base-down prism placed in front of the right eye was used, an equivalent "induced" SSN also was measured. In the top panel, the digital-video data show the SSN was phase locked to the horizontal component of the IN; in the bottom panel, the magnetic search-coil data also show the SSN was phase locked to the torsional component of the IN. That allowed both foveating saccades and periods of extended foveation in all planes of this pseudopendular (with rightward and clockwise foveating saccades) waveform to coincide, thereby providing for the highest visual acuity possible.

4.2. Fixation without diplopia

Our documentation of an "induced" SSN when vertical prisms prevented fusion led us to reexamine ocular motor data of S1, taken during normal fixation of a stationary target (i.e., without diplopia). The digital-video data shown in Fig. 4 (top panel) were taken in January of 2001, several months after the episode of spontaneous diplopia and vertical oscillopsia. A somewhat smaller SSN was present during normal fixation and it was phase locked to the horizontal and torsional components of the pseudopendular with rightward and clockwise foveating saccades waveforms. Search-coil data taken five years earlier (in 1996) also contained a low-amplitude SSN that was phase locked to the horizontal and torsional components of the same pseudopendular waveform. Finally, examination of the earliest search-coil data from S1 (taken in 1984) revealed the same low-amplitude, sub-clinical SSN during fixation of a stationary target (see Fig. 5). As in the more recent data, the SSN was phase locked to the horizontal component of the pseudopendular with rightward foveating saccades waveform.

4.3. Data from additional subjects

Our finding that S1's sub-clinical SSN was neither a result of his transient episode of decompensated vertical phoria nor induced by prisms led us to look for the presence of SSN in the ocular motor data previously taken from fourteen additional subjects. Subjects S2–S10 had either idiopathic or hereditary INS and S11–S15 had INS plus albinism. Fig. 6 contains typical search-coil data from two of the eighteen additional subjects (S7 and S4) who had jerk waveforms. As the top panel shows, S7's jerk left with extended foveation IN waveform had a sub-clinical jerk SSN phase locked to the horizontal component of the IN. In the bottom panel, S4's sub-clinical SSN was also phase locked with the jerk left with extended foveation horizontal component of this IN waveform. Table 1 summarizes the ocular motor characteristics of all 15 subjects. Seven had sub-clinical SSN embedded within their horizontal-torsional IN (column 6, shown bold); in most cases, the SSN

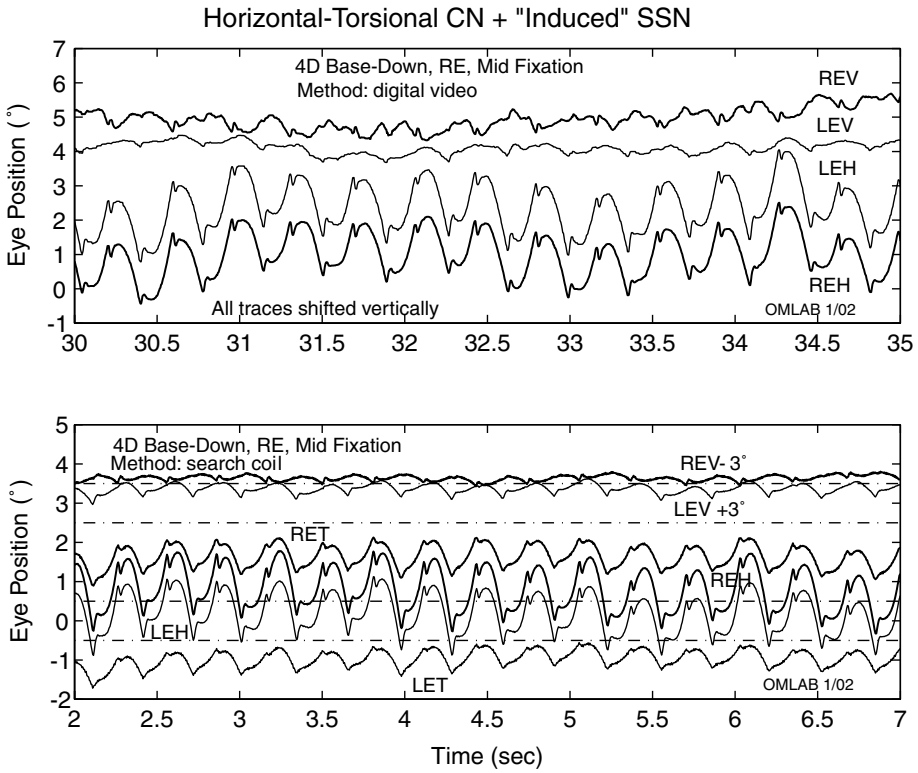

Fig. 3. Right and left horizontal, vertical, and torsional (bottom panel) eye movements of S1 during two intervals of induced diplopia using a 4D base-down prism in front of the right eye while fixating midway between the two horizontal lines. The data in the top panel were taken using digital video and that in the bottom panel, using search coils. The "induced" see-saw nystagmus (SSN) was phase locked with, and had the same pseudopendular with rightward and clockwise foveating saccades waveform as, the horizontal and torsional components of the IN. In this and subsequent figures, REH, right eye horizontal; LEH, left eye horizontal; RET, right eye torsional; Let, left eye torsional; and dot-dashed lines indicate the foveal extent. In the top panel, all traces were shifted for clarity and in the bottom panel, vertical traces were shifted as indicated.

waveform was identical to the subject's horizontal and (where measured) torsional IN waveforms (columns 3,4, and 6). For subjects who did not have asymmetric, (a)periodic alternating nystagmus (APAN), we measured the low SSN amplitudes (column 7, shown bold). In addition, we found sub-clinical, conjugate, vertical IN components in 11 subjects (column 5). Although some of the subjects had simple combinations of waveforms, others (e.g., S8) had more complex combinations, with SSN, conjugate vertical components, or both.

5. Discussion

Previous studies have suggested that extraretinal information about the motor signals driving the eyes (efference copy) has a major role in oscillopsia suppression (Dell'Osso & Leigh, 1992; Bedell & Currie, 1993; Dell'Osso, Averbuch-Heller, & Leigh, 1997). Behavioral models of the ocular motor system with IN or other types of oscillations *require* the use of efference copy to simulate the responses exhibited by individuals with INS to a variety of input stimuli (Dell'Osso, 1968; Dell'Osso & Jacobs, 2001; Jacobs, 2001; Dell'Osso, 2002; Jacobs & Dell'Osso, 2004).

The one-time, short (3 min) period of vertical diplopia in S1 did not cause oscillopsia of either image of the visual field in the horizontal or torsional planes. The perception of each visual-field image appeared to be independently stabilized in the horizontal and torsional planes. In horizontal IN, the oscillations in the two eyes are phase locked, with equivalent waveforms and coincident foveation periods (Dell'Osso, Gauthier, Liberman, & Stark, 1972; Dell'Osso, Van der Steen, Steinman, & Collewijn, 1992) but, as in S1, the amplitudes of the two waveforms may differ (Dell'Osso, 1973). This implies that the suppression of horizontal oscillopsia in each eye occurs independently, using efference copy of the motor commands to that eye; such a monocular mechanism takes advantage of the exquisite sensitivity of each retina and fovea. The two stable perceptions are then combined into one binocular image of the world. No horizontal oscillopsia occurred during the vertical diplopia and the eye-movement data verified that the horizontal IN was unchanged. The same conclusions may be drawn about the absence of torsional oscillopsia. Individuals with oscillopsia secondary to acquired nystagmus may suppress that oscillopsia when the image motion responsible is only partially stabilized (Leigh, Rushton, Thurston, Hertle, & Yaniglos, 1988). Perhaps those with INS have a greater tolerance for retinal motion than normals but that cannot explain oscillopsia of a small SSN but not of the larger horizontal-torsional INS. Prior to our investigation of the source of the perceived *vertical* oscillopsia in the deviated eye, we entertained three possibilities.

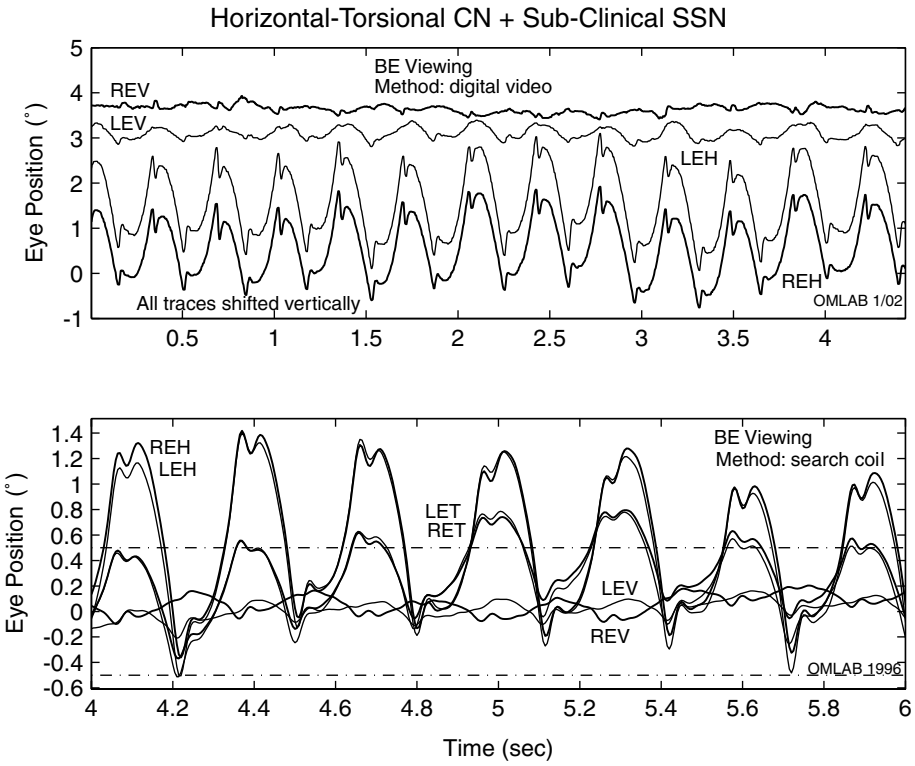

Fig. 4. Right and left horizontal, vertical, and torsional (bottom panel) eye movements of S1 during two intervals of fixation of a stationary target. The data in the top panel were taken using digital video and that in the bottom panel, using search-coils five years prior to the spontaneous incidence of diplopia. The sub-clinical see-saw nystagmus (SSN) was phase locked with, and had the same pseudopendular with rightward and clockwise foveating saccades waveform as, the horizontal and torsional components of the IN. In the top panel, all traces were shifted for clarity.

The first was that the vertical oscillopsia experienced during transient vertical diplopia resulted from a singular occurrence of a vertical component in the IN of the deviated eye that was *uncorrected* by efference copy. However, individuals with horizontal, torsional, or even vertical, components to their IN do not experience oscillopsia. That is, the oscillopsia that might result from all components of IN occurring within the efference copy feedback loop is automatically suppressed (in their respective planes) (Dell'Osso et al., 1997). It is true however, that under certain conditions, individuals with INS may experience some oscillopsia but it is not common under ordinary conditions nor does it have the debilitating effects of acquired oscillopsia (Abadi & Bjerre, 2002; Tkalcevic & Abel, 2003).

The second possibility was that either a rivalry or suppression mechanism (modulated by the IN oscillation) between the two disparate images could have been responsible for the vertical oscillopsia. At each foveation period of the horizontal IN, a change in the fixating eye would induce a vertical shift in perceived position (due to the strabismus), even though neither eye was oscillating vertically. In this way, the constant alternation of perceived vertical position might simulate the perceptual effects of a vertical oscillation. The frequency of the perceived vertical oscillation was ~2–3 Hz, similar to the horizontal IN. When this subject alternated fixation between the eyes (either using an occluder or by winking) the visual field was perceived to jump vertically downward when using the right eye and upward when using the left; this seems to support the rivalry hypothesis for the vertical oscillopsia. The vertical shift was secondary to his right hyperphoria and would become manifest when the phoria decompensated to a tropia. However, such a mechanism would be expected to produce the impression of a vertically jumping motion, due to the abrupt shift in the fixating eye; the actual perception was that of a smoothly oscillating motion.

The third possibility was that vertical oscillopsia of the diplopic image was due to a transient, uniocular vertical nystagmus associated with the sudden loss of vertical fusion. An acquired, uniocular, vertical nystagmus, presumably occurring outside of the efference-copy loop (Dell'Osso et al., 1997), could cause vertical oscillopsia despite the fact that the retinal motion from the horizontal-torsional IN does not normally induce oscillopsia. Superior oblique myokymia (SOM) can also induce uniocular vertical nystagmus and oscillopsia (Leigh, Tomsak, Seidman, & Dell'Osso, 1991). However, SOM results in a low-amplitude (~0.1°), high-frequency (up to 50 Hz) pendular nystagmus that is usually recurrent, lasts less than 10 s, and is associated with a *depression* of the affected eye.

The results of this study suggest that *none* of the above hypotheses was correct. Although the initial data from the prism experiments supported the third hypothesis, data taken during ordinary fixation (i.e., with no vertical

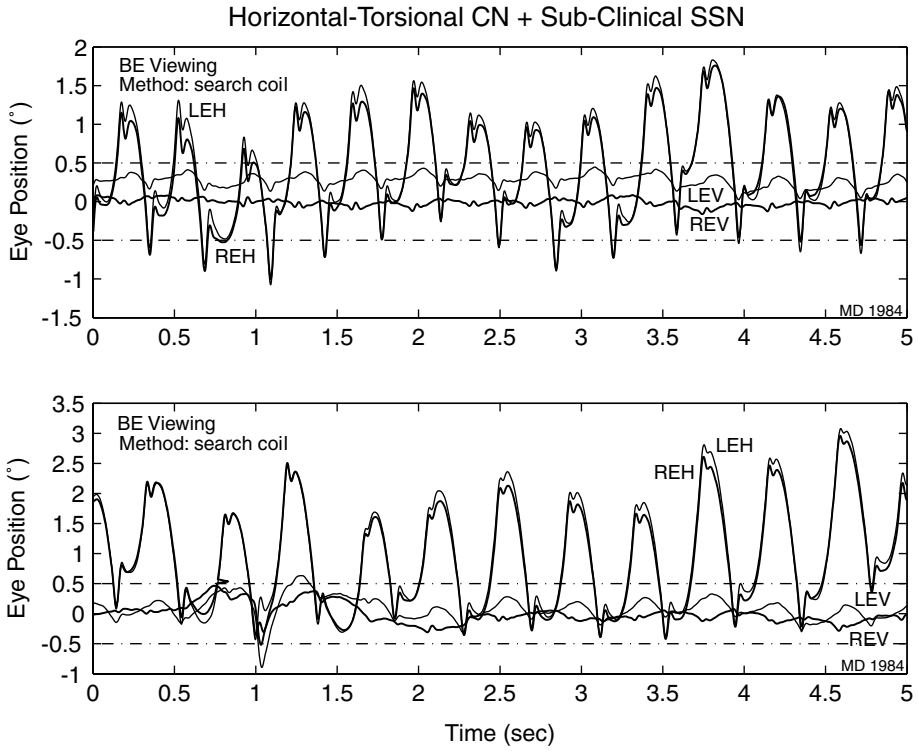

Fig. 5. Right and left horizontal and vertical eye movements of S1 during two intervals of fixation of a stationary target. The data in both panels were taken using search-coils 17 years prior to the spontaneous incidence of diplopia. The sub-clinical see-saw nystagmus (SSN) was phase locked with, and had the same pseudopendular with rightward foveating saccades waveform as, the horizontal component of the IN.

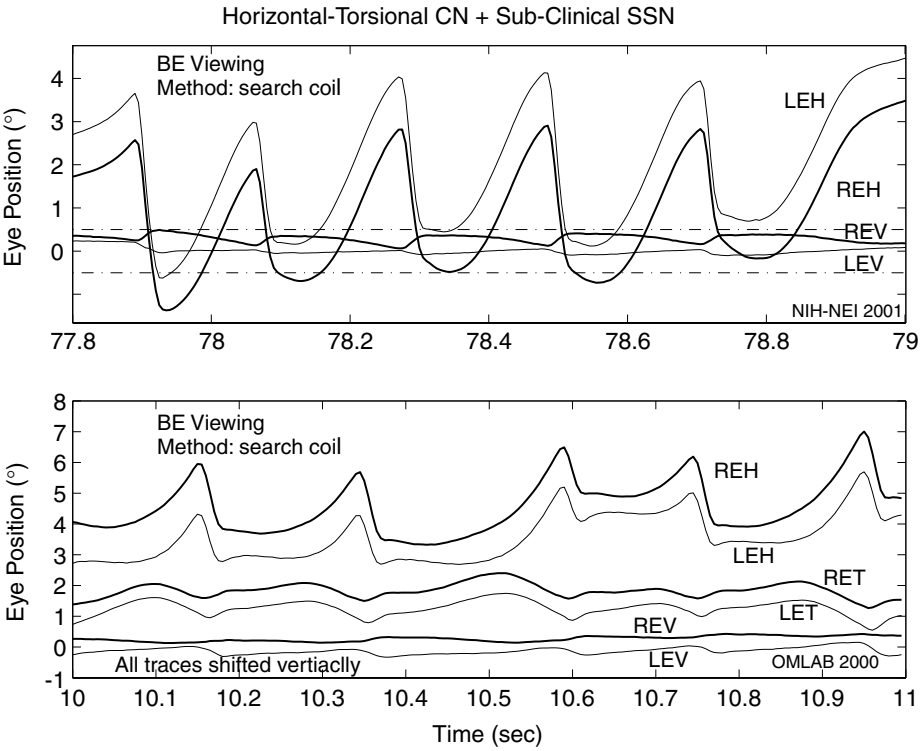

Fig. 6. Right and left horizontal and vertical eye movements of S7 (top panel) and S4 (bottom panel) during intervals of fixation of a stationary target. The data in both panels were taken using search coils. Both subjects had a jerk left with extended foveation IN waveform. The sub-clinical see-saw nystagmus (SSN) was phase locked with, and had the same jerk with extended foveation waveform as, the horizontal components of their respective IN waveforms. In the bottom panel, all traces were shifted for clarity.

Table 1
Nystagmus types and primary-position INS and SSN waveforms

Subject	INS: DIO, ERED, or +ALB	Waveforms				|SSN| (°p-p)
		IN [conjugate directions]			SSNVrt	
		Hor	Tor	Vrt		
S1	Hereditary	PP_{fs}[R-L]	PP_{fs}[CW-CCW]	—	**PP_{fs}**	**0.1–0.5**
S2	Idiopathic	(J, J_{ef})[L]	(J, J_{ef})[CCW]	(J, J_{ef})[U]	—	—
S3	Idiopathic	J_{ef}[R]	J_{ef}[CW]	—	**J_{ef}**	**0.1–0.2**
S4	Hereditary	J_{ef}[R, L]	J_{ef}[CCW, CW]	—	**J_{ef}**	**0.2–0.4**
S5	Idiopathic	J_{ef}[L]	J_{ef}[CCW]	J_{ef}[U]	—	—
S6	Idiopathic	J_{ef}[R, L]	J_{ef}[CW, CCW]	J_{ef}[U]	—	—
S7	Idiopathic	J_{ef}[L]	DNR	—	**J_{ef}**	**0.4**
S8	Idiopathic	(DJ_{ef}, J)[L]		—	**J**	**APAN**
		P[R-L]		—	**P**	**APAN**
		P[R-L]	DNR	P[U-D]	—	—
		PC[R]		J_{ef}[U]	—	—
S9	Idiopathic	(J, PC)[R]		J[U]	—	—
		(J_{ef}, PC)[L]	DNR	PC[D]	—	
		(J_{ef}, PC)[L]		J[U]	—	—
S10	Idiopathic	J_{ef}[L]		PC[D]	—	—
		(J,J_{ef})[R]	DNR	(J, J_{ef})[U]	—	—
		PC[L]		J[U]	—	—
S11	+ALB	PP_{fs}[R-L]		—	**J_{ef}, J**	**0.2–0.8**
		PP_{fs}[R-L]	DNR	J[U]	—	—
S12	+ALB	(PC, J_{ef})[L]		(J_{ef})[U]	—	—
		PJ[R]	DNR	J[U]	—	—
S13	+ALB	J[R]		—	**J**	**APAN**
		J[R]	DNR	J [D]	—	—
		(J, J_{ef})[L]		J [D]	—	—
S14	+ALB	(PP_{fs}, P, P_{fs})[R-L]		(PP_{fs}, P, P_{fs})[U-D]	—	—
		(PC, J_{ef})[L]	DNR	J_{ef}[D]	—	—
S15	+ALB	(PC, J, DJ)[L]		(J, DJ)[D]	—	**APAN**
		(PC, J, DJ)[R]	DNR	(J, DJ)[U]	—	—
		P[R-L]		P[U-D]	—	—

IN(S), infantile nystagmus (syndrome); SSN, see-saw nystagmus (sub-clinical); Hor, horizontal; Tor, torsional; Vrt, vertical; IDIO, idiopathic; HERED, hereditary; +ALB, plus albinism; J_{ef}, jerk with extended foveation; PP_{fs}, pseudopendular with foveating saccades; J, jerk; PC, pseudocycloid; °p-p, degrees peak-to-peak; R, right; L, left; CW, clockwise; CCW, counterclockwise; U, up; D, down; APAN, asymmetric: (a)periodic alternating nystagmus with variable amplitudes; DNR, did not record. Example: S8 had DJ_{ef} and J [both beating left], P [diagonal—right up and left down], P [horizontal] and PC [beating right and up], waveforms. During the diagonal PC and P waveforms there were conjugate vertical J_{ef} and P components and during the horizontal jerk (DJ_{ef} and J) and P waveforms there were J and P SSN waveforms, respectively.

diplopia) also contained SSN, albeit at a lower amplitude. That left the possibility that the spontaneous diplopia event might have left a permanent, low-amplitude SSN. However, our re-evaluations of prior data taken from S1 in 1996 and as far back as 1984 showed that the "induced" SSN was not caused by the loss of vertical fusion but was an exacerbation of a sub-clinical SSN that had always been present but had gone undetected. Our use of data taken with three different methods in several laboratories satisfied us that the sub-clinical SSN was not an artifact of our IR system (i.e., cross talk). Furthermore, the data taken from S2–S15 confirmed that sub-clinical SSN is common in other subjects with horizontal-torsional IN who have no conjugate vertical component and is independent of the IN waveform, occurring in both pendular and jerk waveforms. For the 15 subjects studied, we found SSN in 7 subjects (two of whom had APAN) and a sub-clinical, conjugate, vertical IN component in 11. The latter finding suggests that some individuals with IN have a sub-clinical, conjugate, vertical component. As the Table shows, although vertical components are phase locked to horizontal-torsional components, they may not always have the same waveforms (e.g., S8–S15). Also, a horizontal waveform may be combined with both a conjugate vertical component and SSN (e.g., S8, S11, and S13) or, different SSN waveforms (e.g., S11).

The loss of vertical fusion in the presence of a sub-clinical SSN nystagmus appears to be the causal factor in S1's induced oscillopsia. We hypothesize that the suppression of oscillopsia in a binocular subject with INS (i.e., one with "single-vision") requires a fusible image; when two vertically infusible, oscillating images are produced, only one is perceived as stable, causing oscillopsia of the other. This explanation for the cortical mechanism required for oscillopsia suppression in a binocular individual is supported by retinal image stabilization experiments on a subject with INS (Dell'Osso et al., 1997). When only a portion of the visual field was stabilized, oscillopsia resulted in either that portion (stable on the retina) or the unstabilized portion (oscillating on the retina); both could not be simultaneously perceived as stable. The first perception is easily accepted

but the latter, where the whole room appears to be oscillating, is unsettling. Although this analogy does not rely on the same conditions as the oscillating diplopia experienced by our subject, it does expose a similar limitation on the cortical process that governs the single, stable perception of the world.

The sub-clinical SSN embedded in horizontal-torsional IN should not be confused with the larger, clinically visible SSN found in achiasma or hemichiasma (Dell'Osso & Daroff, 1998; Dell'Osso, Hogan, Jacobs, & Williams, 1999). The SSN associated with achiasma was first identified in 1992 in a family of achiasmatic Belgian sheepdogs (Dell'Osso, Williams, Jacobs, & Erchul, 1998) and subsequently in an achiasmatic human (Dell'Osso, 1996; Dell'Osso, Williams, Jacobs, & Erchul, 1996). In achiasma, SSN is due to instability in vertical-torsional control and need not have the same waveform as the horizontal component of INS. Following those initial observations identifying SSN in achiasma and the hypothesis that SSN was a sign of achiasma, other achiasmatic humans have subsequently been diagnosed and studied (Leitch et al., 1996; Hertle et al., 2002).

What could be the cause of the sub-clinical SSN embedded in horizontal-torsional IN with no conjugate vertical component? In order to produce a pure torsional movement (or oscillation) the vertical component of the forces exerted by the oblique muscles must be counter balanced by the vertical forces exerted by the vertical recti to prevent any vertical motion. However, the vertical rectus muscles are much larger and stronger than the oblique muscles. Therefore, even a slight imbalance could add a vertical component to the otherwise purely torsional movement. As Table 2 shows, the directions of these unbalanced forces coincide with those required to produce the motion described in SSN. For example, during the clockwise phase of torsional movement, the superior oblique and superior rectus muscles move the left eye while the inferior rectus and inferior oblique muscles move the right eye. If the forces in the rectus muscles exceed those in the corresponding oblique muscles, the left eye will rise and the right eye fall in addition to the conjugate clockwise torsion. Analysis of the forces and motions during the counterclockwise torsional phase predicts that the left eye will fall and the right eye will rise. The net result is, the intorting eye elevates while the extorting eye depresses-*classical SSN*. We hypothesize this to be the mechanism underlying the sub-clinical SSN embedded in horizontal-torsional IN. The similarity of the vertical and torsional waveform supports this hypothesis since the same neural signal would drive both the vertical rectus and oblique muscles.

Table 2
Muscle actions during torsional nystagmus

Torsional component		Left eye		Right eye	
Phase	Plane	Muscle	Motion	Muscle	Motion
CW	Torsion	SO& SR	CW	IO& IR	CW
	±Vertical	SR > SO	UP	IR > IO	DOWN
CCW	Torsion	IO& IR	CCW	SO& SR	CCW
	±Vertical	IR > IO	DOWN	SR > SO	UP

CW, clockwise; CCW, counterclockwise; SO, superior oblique; SR, superior rectus; IR, inferior rectus; IO, inferior oblique.

If individuals with INS have a slight vertical force imbalance causing sub-clinical SSN, does the same exist in normals? That is, if torsional optokinetic nystagmus were induced in normals, would a subtle SSN be superimposed on the torsional nystagmus? If so, that would support our hypothesis. If not, the hypothesis could still hold if individuals with INS had such a miscalibration of vertical forces, similar to the miscalibrations that cause INS.

In conclusion, a decompensated vertical phoria in a visually introspective observer with INS produced conditions under which the perception of diplopic oscillopsia could not be suppressed in the vertical plane; this provides evidence supporting the hypothesis that oscillopsia suppression is dependent on efference copy of the unaffected horizontal motion. However, in this individual, efference copy was unable to suppress the vertical oscillopsia resulting from a pre-existing, sub-clinical vertical oscillation exacerbated by diplopia. As a result of the ocular motor studies prompted by studying this subject, sub-clinical SSN was found to be embedded in the horizontal-torsional IN of 46.67% of the additional subjects we studied (i.e., all those with no conjugate vertical component). Sub-clinical SSN is hypothesized to be due to slight innervational (or force) imbalances of the vertical and oblique extraocular muscles of the eyes.

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