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ABNORMAL OPTOKINETIC AND PERCEPTUAL SPAN PARAMETERS IN CEREBELLAR-VESTIBULAR DYSFUNCTION AND RELATED ANXIETY DISORDERS[1]

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Summary.—As prior studies indicated abnormal cerebellar-vestibular-based sensorimotor mechanisms and neurological and ENG diagnostic parameters in anxiety disorders and because ocular fixation and sequential scanning are cerebellar-vestibular-modulated, it appeared reasonable to measure these and related ocular functions in matched samples of anxiety-disordered and control subjects. In this study, the optokinetically-determined fixation, sequential scanning, and perceptual span capacities obtained by means of a newly revised blurring-speed method were significantly lower or impaired in 70 anxiety-disordered patients vs 70 controls. Such data supported further the hypothesis that there may be cerebellar-vestibular predispositions to anxiety disorders and the optokinetically-based tracking method may prove useful in separating a diverse array of CV-determined or related anxiety symptoms from those of other origins. However, independent validation as well as additional studies of anxiety disorders using larger samples vs random or "normal" controls are required before conclusions are justified.

A wide and diverse range of independently performed studies have indicated a role for cerebellar-vestibular (CV) mechanisms in (1) fears/phobias and related anxiety symptoms (Benedikt, 1870; Guye, 1899; Lannois & Tournier, 1899; Fenichel, 1945; Marks & Bebbington, 1976; Blythe & McGlown, 1979; Page & Gresty, 1985; Jacob, Moller, Turner, & Wall, 1985) and (2) in optokinetic and related fixation and tracking functions (Ito, 1984; Stroud & Rauchbach, 1976; Baloh, Konrad, & Honrubia, 1975; Burde, Stroud, Roper-Hall, Wirth, & O'Leary, 1975). In prior studies CV-based sensorimotor mechanisms and related neurological and electronystagmographic (ENG) diagnostic parameters were associated with anxiety disorder (Levinson, 1980, 1984, 1986). These data together with the favorable response of anxiety-related symptoms to CV-stabilizing medications in clinical trials suggested further that anxiety disorders may be significantly CV-determined or predisposed (Frank & Levinson, 1976-1977, 1977; Levinson, 1980, 1984, 1986). As the ocular fixation and sequential scanning mechanisms are CV-modulated according to Sir John Eccles (personal correspondence, 1987), Nobel Laureate in cerebellar neurophysiology,

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and as these and related perceptual span functions and symptoms improved via CV-modulated oculomotor exercises and medications (Halliwell & Solan, 1972; Pierce, 1977; Flax, Mozlin, & Solan, 1984; Levinson, 1980, 1984), it seemed reasonable to expect that these tracking mechanisms may be impaired in CV-related anxiety disorders (Levinson, 1989b, 1989c). The aim of this study was to compare the optokinetic and perceptual span parameters in a sample of anxiety-disordered patients and matched controls.

Method

Subjects

Using a revised blurring-speed method (Levinson, 1989a), optokinetically-determined fixation, tracking, and perceptual span parameters were obtained from 70 volunteers and 70 matched anxiety-disordered patients and compared. The controls were volunteers from schools, businesses, and neighborhoods adjacent to The Medical Dyslexic Treatment Center. The volunteers had no greater knowledge of the research procedures and aims than patients. The 70 anxiety-disordered were selected from a group of 402 similarly impaired patients previously described (Levinson, 1989c). The subsample of 70 was representative of the total 402 anxiety-disordered and matched by group to the 70 controls for mean age, age range, sex, handedness, above average IQ, and middle-class socioeconomic status. For the 70 volunteers the mean age was 27 yr. ± 12 yr.; ages ranged from 17 to 50 yr. The male/female ratio and percentages for complete-right-handed, complete-left-handed, and mixed-handed were 1/1.3, and 85.7%, 8.6%, 5.7%, respectively. As a result of matching, the anxiety-disordered subsample of 70 had a mean age of 27 yr. ± 10, and their ages ranged from 17 to 50 yr.; the male/female ratio and percentages of handedness were closely comparable to those in the volunteer sample.

Mixed-handedness was judged present when an individual was naturally able to perform one or more functions, such as eating, catching, throwing, writing, etc., better or as well with the nondominant hand as the dominant one. The remainder were either completely right-handed or completely left-handed. All subjects had either normal or corrected 20/20 visual acuity as tested independently and by means of a Snellen chart.

Diagnostic Information

Anxiety disorder.—In the previously mentioned neurological and ENG analysis (Levinson, 1989c), 402 consecutively referred subjects with severe phobias and related anxiety symptoms had been diagnosed as anxiety-disordered using DSM-III—R criteria (American Psychiatric Assn, 1987). In this study, a representative subsample of 70 anxiety-disordered were selected from the 402 by a computer programmed to match the volunteer controls.

Of the 70, 28.5% were pharmacologically treated by other physicians and were taking minimal amounts of antidepressant and antipanic medications during testing. The optokinetic and perceptual span parameters for the medicated and nonmedicated subgroups were compared.

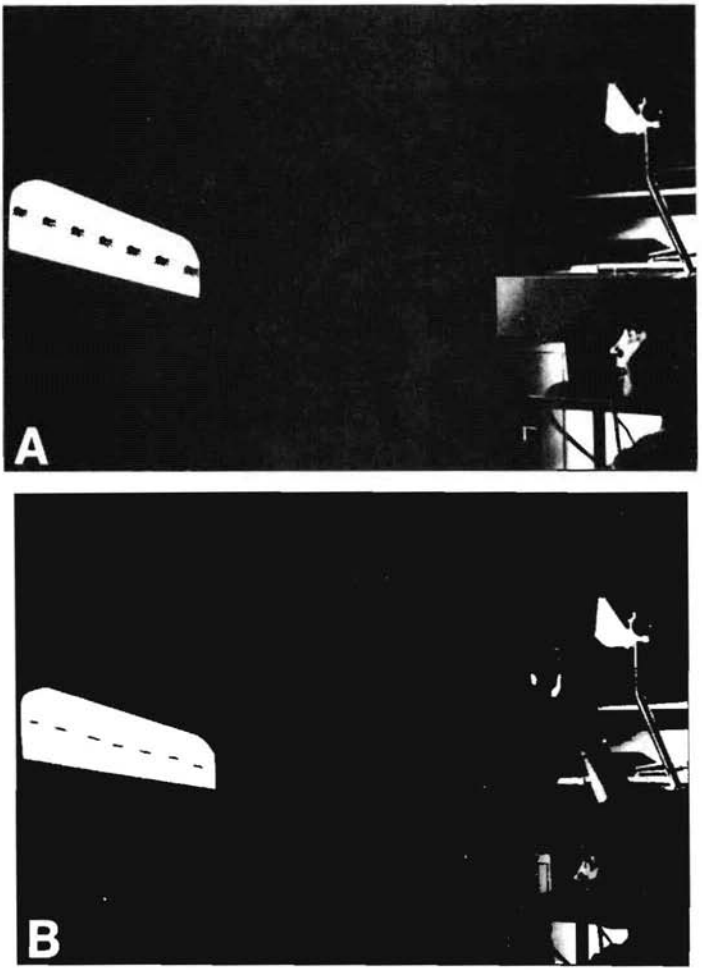

FIG. 1. (A) A subject observing the elephant Mode I gestalt during optokinetic testing with the 3-D optical scanner and (B) reporting (by raising hand) when the elephant sequence initially appeared blurry (Reprinted from Levinson, 1989a, p. 41)

CV-dysfunction.—The CV-determined neurological and ENG parameters characterizing the 70 anxiety-disordered patients were representative of the sample of 402 from whom they were selected. The neurological and ENG techniques have been described elsewhere (Levinson, 1980, 1989c;

Brookler & Pulec, 1970; Brookler, 1971). To ensure diagnostic reliability, CV-dysfunction is reported using two or more abnormal neurological and/or ENG parameters per diagnostic category per subject. Accordingly, of these 70 anxiety-disordered patients, 91% evidenced CV-neurological or ENG dysfunction, 83% neurological dysfunction, and 57% ENG dysfunction. [The corresponding parameters characterizing the larger sample of 402 anxiety-disordered patients were 94.0%, 81.8%, and 61.6%, respectively.]

Procedure

Optokinetic and perceptual span methods.—A revised optokinetic-tracking method was used to measure simply, rapidly, and accurately the ocular fixation, sequential scanning, and perceptual span functions assumed to be impaired in CV-determined anxiety disorders. A detailed description of this method has been reported previously (Levinson, 1980, 1989a). The various optokinetic-tracking or blurring-speed parameters and the Modes I, II and III visual testing modalities used here are summarized and illustrated. Fig. 1A shows a subject observing an (Mode I) elephant sequence and Fig. 1B indicates the subject raising her hand when the accelerating elephant sequence reaches a speed at which blurring occurs, i.e., the blurring-speed. Figs. 2, 3, and 4 indicate the Modes I, II, and III visual gestalts, respectively.

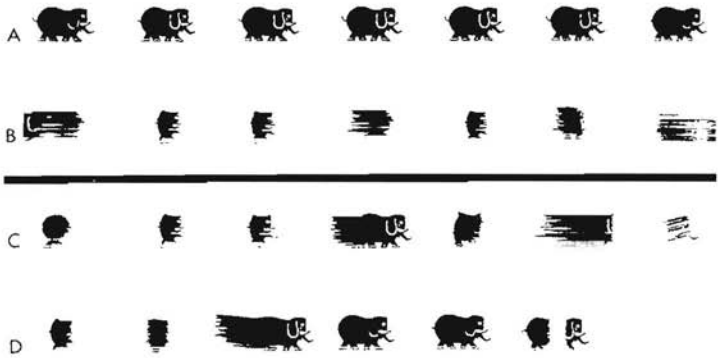

FIG. 2. (A) The elephant Mode I gestalt and (B) its blurring-speed endpoint. (C) and (D) are examples of single-targeting or tunnel vision. Mode I testing was used here to obtain an estimate of the visual span during the perception of the stationary elephant sequence (Reprinted from Levinson, 1989a, p. 43).

Mode I testing.—The Mode I gestalt consisted of a visual span of seven black elephants set against a blank, white surround (Fig. 2). In this study, the Mode I gestalt was only used to test the visual span during the perception of stationary events. Here, subjects were asked to concentrate and fixate on the center elephant of a stationary seven-elephant sequence and report the total number of elephants they could clearly recognize without moving their eyes.

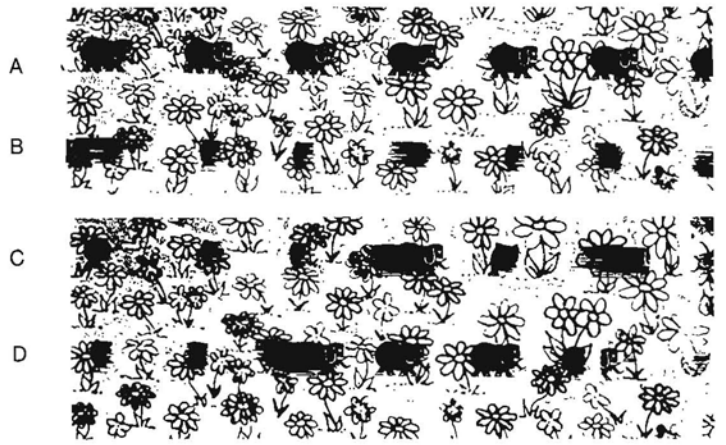

FIG. 3. (A) The elephant Mode II gestalt and (B) its blurring-speed endpoint. (C) and (D) are examples of single-targeting or tunnel vision. Mode II testing was used here to estimate the blurring-speed endpoint as well as the visual span during optokinetic tracking of the moving elephants (Reprinted from Levinson, 1989a, p. 43).

Mode II testing.—The Mode II gestalt consisted of a visual span of seven black elephants set against a colored, floral background (Fig. 3). To measure *maximum optokinetic tracking capacity*, the elephant foreground was slowly accelerated against the stationary floral background until the onset of blurring of the elephant-sequence was reported by the observer. The speed of the elephant-sequence triggering blurring or the *blurring-speed* was assumed to be a measure of the maximum oculomotor tracking capacity. To measure the *perceptual span* in this mode, observers were asked to report the number of elephants they could clearly recognize just before the blurring-speed was reached.

Mode III testing.—The Mode III gestalt (Fig. 4) consisted of an optokinetic foreground (black stripes resembling a picket fence) projected against a stationary background consisting of a visual span of seven black elephants set against a blank, white surround (Mode I gestalt). Observers were asked to concentrate on the moving optokinetic foreground and report whether the elephant-sequence was experienced as blurred and to describe what they saw. The presence of *background-blurring* was assumed to be an indicator of impaired capacity for foreground/background fixation and refixation. Subjects were also routinely evaluated for perceptual span and the presence or absence of movement illusions during Mode III testing. *Movement illusions* represented foreground/background movement reversals, i.e., subjects experienced themselves or the stationary elephants in motion. Both background-blurring and movement illusions were considered probable indicators of perceptual instability.

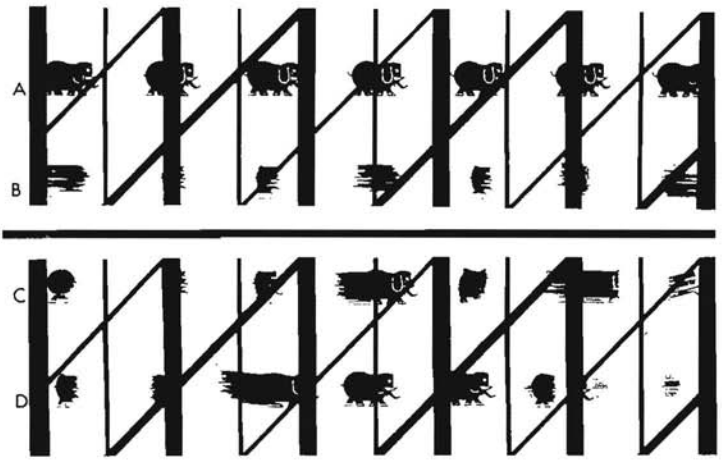

FIG. 4. (A) The elephant Mode III gestalt and (B) its Mode III blurring endpoint. (C) and (D) are examples of single-targeting or tunnel vision. Mode III testing here was used to ascertain the presence of background-blurring, movement illusions, and the visual span during movement of the optokinetic grid (Reprinted from Levinson, 1989a, p. 44).

The 3D Optical Scanner (Levinson, 1980) used to project the Modes I, II, and III images was calibrated daily to ensure blurring-speed accuracy. All optokinetic data, blurring-speed aside, were counted when two successive trials gave identical results. When two successive blurring-speeds were ≤ 0.2 ft/sec. apart, the averaged blurring-speed was used. Accordingly, all subjects were tested at least three times, the first test being a trial run.

RESULTS

A direct comparison, parameter by parameter, for the anxiety-disordered group and the volunteer controls was made using a two-tail t test. The analysis also estimated possible relations among the various optokinetic and perceptual or visual-span parameters as well as their possible association with age, sex, and handedness. Table 1 gives means and *SD*s or percentages for each measure for each of the two groups. Table 2 shows the values for these same parameters for the anxiety-disordered groups as a function of sex. The blurring-speed velocity is expressed in ft/sec., the perceptual span is given in the number of elephants clearly seen and recognized, and background-blurring and movement illusions are reported as percentages of the total sample experiencing these illusions.

Comparison of Anxiety-disordered and Control Groups

Using a two-tail t test, statistical analysis indicated that all optokinetic tracking parameters were significantly ($p \leq .01$) lower or impaired for the anxiety-disordered patients vs control subjects. No significant differences were noted between groups on the various optokinetic parameters which

TABLE 1
OPTOKINETIC TRACKING PARAMETERS: ANXIETY DISORDER VS CONTROLS

Measure	Anxiety Disorder $n = 70$		Controls $n = 70$	
	M	*SD*	*M*	*SD*
Maximum Tracking Capacity or Blurring-speed (ft/sec.)	2.0	1.1	3.2	1.3
Perceptual Span or Number of Elephants Seen				
Mode I Testing	2.6	1.5	4.3	2.3
Mode II Testing	1.9	0.9	4.5	2.1
Mode III Testing	1.8	1.1	4.1	2.2
Perceptual Instability	*n*	%	*n*	%
Impaired Fixation, Refixation, Background-blurring	39	55.7	12	17.1
Movement Illusions	33	47.1	6	8.6

characterized the medicated vs nonmedicated anxiety-disordered subsamples, so the subsamples were combined and treated as one. On two-tail t tests, no parameter met a probability level of .01.

TABLE 2
OPTOKINETIC TRACKING PARAMETERS AS FUNCTIONS OF SEX IN ANXIETY DISORDER

Measure	Female $n = 39$		Male $n = 31$	
	M	*SD*	*M*	*SD*
Maximum Tracking Capacity or Blurring-speed (ft/sec.)	2.1	1.1	1.9	0.9
Perceptual Span or Number of Elephants Seen				
Mode I Testing	2.5	1.5	2.7	1.6
Mode II Testing	1.9	0.7	1.9	0.9
Mode III Testing	1.7	0.7	2.0	1.3
Perceptual Instability	*n*	%	*n*	%
Impaired Fixation, Refixation, Background-blurring	22	56.4	17	54.8
Movement Illusions	18	46.2	15	48.4

Maximum tracking capacity (blurring-speed).—A two-tail t test indicated that the anxiety-disordered patients had significantly lower mean tracking capacities or blurring-speeds during Mode II testing (2.0 ± 1.1 ft/sec.) than the control subjects (3.2 ± 1.3 ft/sec.; $t = 5.9$, $p < .001$).

Impaired fixation and re-fixation capacity (background-blurring) and movement illusions.—During Mode III testing, 39 or 55.7% of the anxiety-disordered patients reported some background-blurring of the elephant-sequence but only 12 or 17% of the control subjects did so ($t = 4.8$, $p < .001$). These data suggest that the anxiety-disordered patients evidenced

greater impairment of fixation and refixation capacity than control subjects. Movement illusions suggestive of perceptual instability were reported by 33 or 47% of anxiety-disordered and by only 7 or 6% to 8% of control subjects ($t = 5.1$, $p < .001$).

Perceptual spans.—The anxiety-disordered patients evidenced significantly lower perceptual spans during Modes I, II, and III testing than the control subjects. During Mode I testing, the mean number of elephants clearly recognized by the anxiety-disordered patients was 2.6 ± 1.5 vs 4.3 ± 2.3 for the control subjects ($t = 5.2$, $p < .001$). During Mode II testing, the mean number of elephants clearly recognized by the anxiety-disordered patients just prior to the blurring-speed endpoint was 1.9 ± 0.9 vs 4.3 ± 2.0 for the control subjects ($t = 9.5$, $p < .001$). During Mode III testing, the mean number of elephants clearly recognized by the anxiety-disordered patients was 1.8 ($SD = 1.1$); the values of 4.1 ± 2.2 characterized the control subjects ($t = 7.8$, $p < .001$).

Relations Among Various Optokinetic Tracking Parameters

To investigate a possible relation between blurring-speeds (maximum tracking capacity) and the three perceptual-span parameters obtained during Modes I, II, and III testing, correlation matrices were computed for the anxiety-disordered patients and the control subjects separately. Positive linear relations were found among all the optokinetic parameters for the control subjects and only some of the parameters for the anxiety-disordered patients, i.e., blurring-speed and Mode III perceptual span, Mode II and Mode III perceptual spans, and Mode I and Mode II perceptual spans.

On a two-tail t test, the mean blurring-speeds ($\pm SD$) corresponding to the anxiety-disordered subsamples reporting either background-blurring (1.5 ± 0.7) or movement illusions (1.6 ± 0.9) during Mode III fixation and refixation testing were significantly lower than the respective subsamples reporting *no*-background-blurring (2.3 ± 0.8; $t = 3.6$, $p < .001$) and *no*-movement illusions (2.2 ± 1.2; $t = 2.6$, $p < .01$). A similar relation was found for background-blurring and low blurring-speed within controls. Movement illusions were too infrequently reported among controls for meaningful statistical evaluation.

All of the above optokinetic-tracking parameters were statistically independent of sex in the anxiety-disordered and control group. Using a two-tail t test, no parameter met a probability level less than 0.1. Unfortunately, the size of the sample for the handedness data was insufficient for statistical evaluation. A regression analysis was performed on blurring-speed and perceptual span parameters vs age for both groups. There was no significant variation in blurring-speed or perceptual span characterizing the anxiety-disordered and control subjects of 17 to 50 yr. of age.

Discussion

Based on a revised optokinetically-based tracking method, ocular fixation, sequential scanning, and perceptual span were described as impaired in anxiety-disordered patients relative to a control group. As the optokinetic response as well as the ocular fixation and sequential tracking mechanisms are CV-modulated (Ito, 1984; Eccles, personal correspondence, 1987), and as anxiety-disordered patients showed a significant presence of CV-dysfunctioning neurological and ENG parameters (Levinson, 1980, 1989b, 1989c), the optokinetic data supported further the hypothesis that anxiety disorders are CV-predisposed.

Even the narrowed perceptual span or tunnel vision in anxiety-disordered relative to control subjects is consistent with the known CV-dysfunctioning mechanisms called *decomposition of movement* (Dow & Moruzzi, 1958). As the vestibular system plays a vital role in the reciprocal coordination of peripheral and central vision (Guedry, Lentz, & Jell, 1979; Lovegrove, Heddle, & Slaghus, 1980; Dichgans, 1977), it appeared reasonable to expect that a CV-based decomposition or simplification of this oculomotor visual process into its basic unitary components would yield either central or peripheral vision and so a restricted perceptual field. Indeed, whereas most CV-dysfunctioning, anxiety-disordered subjects reported tunnel vision during optokinetic testing, a few appeared to see elephants better in the periphery than in the central field, and occasionally only every second or third elephant was seen along the seven-elephant sequence.

Also, a wide range of compensatory mechanisms were suggested by patients' attempts to reintegrate or 'recompose' central and peripheral visual mechanisms in regaining and maintaining functional or perceptual integrity within this adaptively vital visual system. Central vision was functionally stabilized by compensatory foreground concentration and peripheral (background) inhibition or tunnel vision in many CV-dysfunctioning patients. Where fixation mechanisms were significantly impaired, central vision appeared to be suppressed in favor of peripheral vision (Levinson, 1980). In retrospect, reciprocally active, coordinated and integrated inhibitory vs facilitating central and peripheral or foreground/background visual mechanisms were normally but silently present. However, in the presence of CV-dysfunctioning the integrity of such mechanisms is impaired, "decomposed," or simplified unless compensation occurs.

Despite the fact that most CV-dysfunctioning, anxiety-disordered subjects evidenced reduced fixation, tracking, and perceptual span capacities, some did not and a few exhibited overcompensatory functioning. Moreover, although many CV-dysfunctioning patients showed equally reduced perceptual spans in all three testing modes, some persons showed significant intertest variations. Upon exploration, each testing mode triggered a unique

combination of dysfunctioning and compensatory mechanisms for subjects and groups.

The qualitative analyses of these seemingly inconsistent and paradoxical quantitative differences eventually led to unique insights into the unexpected complexity of the visual tracking processes characterizing the optokinetic method, CV-dysfunctioning, and the multidimensional vectors serving to regain and maintain functional or perceptual capacity and integrity (Levinson, 1980, 1989a, 1989b, 1989c). For example, the perceptual spans of some CV-dysfunctioning patients appeared paradoxically widened by the more complex moving vectors triggered during Mode II or Mode III testing and narrowed by the seemingly simple efforts required to concentrate on and visualize a stationary central or foreground target during Mode I testing. Upon analysis, movement vectors occasionally facilitated peripheral perception (perhaps via disinhibitory mechanisms), whereas stationary central fixation and concentration resulted in compensatory peripheral inhibition. Needless to say, the number of observations needed for new clinical insights appeared exponentially higher than those needed for their statistical validation. Considering the complexity of dysfunctioning and compensatory variables in play, it appeared remarkable that linear correlations were observed among many of the optokinetic tracking parameters. These data suggested the following: (1) All the optokinetic parameters are most probably measuring functional derivatives of the same cerebellar-vestibular common denominator. (2) Compensatory factors were sufficiently minimized during the testing to allow some of these associations to appear.

These very same CV-determined optokinetic-tracking and perceptual span (as well as neurological and ENG) parameters were reported to be similarly impaired and compensated for in learning disabilities or dyslexia (Frank & Levinson, 1973, 1975-6, 1976; Levinson, 1980, 1988, 1989a). Moreover, a wide and diverse array of studies have independently supported this CV-determined and oculomotor role in learning disabilities or dyslexia (Ayers, 1972; deQuiros, 1976; Blythe & McGlown, 1979; Kohen-Raz, 1970; Pavlidis, 1981, 1985; Black, Collins, DeRouach, & Zubrick, 1984). As CV-based sensorimotor mechanisms, neurological, ENG, and optokinetic diagnostic parameters characterized patients with learning disabilities and those with anxiety disorders, and as symptoms of both disorders significantly overlap with one another and respond favorably to a wide variety of CV-stabilizing medications including the antipanic, antidepressant, and beta-blocking agents (Levinson, 1980, 1986; McClure, Lycett, & Baskerville, 1982; Bastecky, Bolelоucky, & Skovronsky, 1981), it appeared likely that both disorders are determined by similar underlying CV denominators.

Improvements in the symptoms resulting from impaired ocular fixation, sequential scanning, perceptual span, and disruptions within related central

and peripheral inhibition and facilitation mechanisms were reported by a majority of CV-dysfunctioning patients (anxiety-disordered and dyslexic or learning disabled) treated with CV-stabilizing medications in clinical trials (Levinson, 1980, 1984, 1986). For example, the favorable responses of oculomotor symptoms (blurring, scrambling, oscillopsia, reversals, losing one's place, tunnel vision, etc.) to the CV-stabilizing medications suggested that the CV-system plays a key role in both their creation and compensation. These observations and assumptions are supported by the studies of Robinson (1976), whose data clearly indicated that the cerebellum detects and repairs oculomotor reflexes.

Favorable responses to the CV-stabilizing medications were also noted clinically in a variety of interconnected CV-based photophobic or visual "overloading" symptoms triggered by fluorescent lighting or sunlight, alternating sun and shade strobe-like effects, glare, specific colors, and patterns of "crowds"-animate or inanimate and moving or stationary. These favorable responses suggested: (1) The CV-stabilizing medications facilitate CV-modulated visual inhibition, shielding, or "internal filtering" in a manner analogous to their well-recognized, similar role in compensating for motion overloading (real or relative), related sensorimotor symptoms, and avoidance or anxiety responses. (2) Visual disinhibition or impaired filtering may result from CV-dysfunctioning and may secondarily destabilize other interconnected CV functions just as motion overloading destabilizes interconnected sensory, balance, coordination, and associated CV-functioning (Levinson, 1980, 1986, 1989c).

The CV system has been shown to modulate the entire sensory input and motor output (Snider & Stowell, 1944; Dow & Anderson, 1942; Adrian, 1943). Also, as CV-dysfunctioning may result in auditory, tactile, proprioceptive (and motor) as well as visual and motion disinhibition with associated symptoms, and as interconnected CV-tracking and related mechanisms are secondarily destabilized by overloading phenomena and improved by CV-stabilizing medications (Levinson, 1980, 1984, 1986), it appears reasonable to assume that CV mechanisms modulate all sensory (and motor) inhibitory mechanisms. These clinically based insights may help explain why CV-dysfunctioning, anxiety-disordered (and learning disabled or dyslexic) patients sometimes experience symptomatic relief in visually related photophobic and tracking symptoms when using colored or tinted lenses for external filtering. In other words, external lens filtering may facilitate compensation in a manner analogous to the way the CV-stabilizing medications facilitate "internal filtering." Indeed, these observations appear consistent with and help explain extensive observations by Helen Irlen, who, in a personal correspondence in 1988, reported that 50% of unselected and 75 to 80% of selected reading-disabled patients benefit from using colored lenses.

Also, prior optokinetic studies showed that the various blurring-speed parameters are gestalt-specific and so by subject and sample vary with shapes and colors (Levinson, 1980). As a result, the use of specific colored shapes and/or colored lenses may decrease visual and interconnected CV symptoms by (1) externally decreasing visual overloading and/or (2) increasing fixation and tracking capacities. The latter may result in related compensatory CV-functioning in a manner analogous to the way visual fixation and concentration are shown to decrease vestibular overreactivity, motion sickness, and the nystagmus triggered by caloric and rotation stimulation via inhibitory mechanisms (Levinson, 1980; Wilson, Maeda, & Franck, 1975). The present author has advised many a photophobic to use tinted glasses to reduce visual phobic triggers and many a dyslexic to obtain reading instructions in bold, colored, easily targeted print. The color of choice was determined clinically by trial and error or using the optokinetic-tracking method.

Moreover, the role of CV-dysfunctioning in pathological disinhibition can readily explain (1) the predisposition or sensitivity of anxiety-disordered patients to a wide variety of sensorimotor, chemical, and associated phobic triggers, and therefore (2) a wide variety of related, heretofore puzzling fears triggered by sounds, colors, smells, shapes and/or animal forms. If, for example, phylogenetically vital but currently maladaptive or useless instinctive responses and built-in release mechanisms (Lorenz, 1957a, 1957b) are either pathologically released from suppression and/or triggered by CV and related CNS disinhibitory mechanisms, then anxiety responses may suddenly arise to various "innocuous" stimuli and animal forms which are no longer adaptively inhibited, suppressed, or filtered-out. Needless to say, medications and other therapeutic techniques which facilitate selective suppression, filtering, inhibiting, or conditioning will result in symptomatic improvements (Levinson, 1980, 1984, 1986).

In conclusion, the present data tended to substantiate the hypothesis that anxiety disorders may be CV-predisposed to abnormal optokinetic-tracking and perceptual span dysfunctioning and that the optokinetic tracking method is CV-modulated and so capable of diagnosing CV-dysfunctioning. Moreover, as fears/phobias are frequently triggered by visual "overloading" patterns, "crowds" or photophobic stimuli, the complex interactions between CV-based visual and related sensorimotor disinhibitory and inhibitory mechanisms were explored. The resulting insights have added a new dimension to the understanding and treatment of phobias and related CV-determined symptoms. As dyslexia or learning disabilities and anxiety symptoms appear to be determined by CV-based sensorimotor functions, and as both disorders share overlapping symptoms as well as CV-based optokinetic, neurological, ENG, and treatment responses, it appears reasonable to assume that both disorders are caused by the same group of CV denomi-

nators, although with varying symptom-shaping mechanisms. However, independent and additional studies are required in which significantly larger numbers of patients and control subjects are employed before convictions are justified as to the CV-basis of anxiety disorders (and dyslexia or learning disabilities) and the diagnostic reliability of the optokinetic (neurological and ENG) method.

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