A Reprint from

PERCEPTUAL AND MOTOR SKILLS

February 1989

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Perceptual and Motor Skills, 1989, 68, 323-338.

THE CEREBELLAR-VESTIBULAR PREDISPOSITION TO ANXIETY DISORDERS [1]

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Summary.—To test for a cerebellar-vestibular (CV) predisposition to anxiety disorder, 402 consecutively referred subjects with varying anxiety symptoms were separated into eight DSM-III—R diagnostic categories and evaluated for CV dysfunction, using neurological and electronystagmographic (ENG) examinations. Of the total sample, 94% evidenced CV-dysfunction on the basis of two or more abnormal neurological or ENG parameters per subject. All DSM-III—R diagnostic anxiety-disorder categories contained a high percentage of abnormal neurological and ENG parameters, regardless of the size of the subsample. Moreover, each DSM-III—R subsample of anxiety disorders contained additional coexisting symptoms of anxiety sufficient to overlap with and form the basis for diagnosis of most other DSM-III—R anxiety-disorder categories. Such findings suggested that anxiety disorders, regardless of surface descriptions and DSM-III—R category, have a common denominator with varying symptom-shaping mechanisms and that this denominator is significantly CV-based. Although the above findings do not justify cause and effect convictions, they have provided crucial insights leading to (1) a proposed functional classification based on underlying determining mechanisms rather than on descriptions of symptoms, (2) a possible relationship between anxiety and learning disorders, and (3) a new method of treating these disorders by means of CV-stabilizing medications in conjunction with traditional approaches. Needless to say, independent and controlled studies, including comparisons with "normal" persons, are required for both validation and elucidation of those specific determining vs compensatory mechanisms and related diagnostic parameters crucial for symptom formation.

A wide range of independently derived clinical evidence supporting a cerebellar-vestibular (CV) predisposition or "somatic compliance" (Freud, 1958, p. 248) for anxiety disorders remained isolated from the scientific mainstream as a result of the overwhelming dominance of psychoanalytic, conditioning, and biochemical data and explanations of these symptoms (Klein, 1981). For example, Benedikt in 1870 suggested that agoraphobia may be of vestibular origin; and Guye as well as Lannois and Tournier published similar findings in 1899. Approximately 80 years later, Marks and Bebbington (1976) described space phobias as possible agoraphobic variants associated with vestibular dysfunction, and Blythe and McGlown (1979) pointed to vestibular mechanisms as determining agoraphobic and claustrophobic symptoms. Recently, Page and Gresty (1985) described motorists'

[1]This paper was presented at the First International Conference of Neurological Dysfunction, Chester, England, October 1987. Special thanks are extended to my staff and that of the journal for dedicated help in preparing this paper for publication. Address requests for reprints to H. N. Levinson, M.D., Medical Dyslexic Treatment Center, 600 Northern Boulevard, Great Neck, NY 11021.

vestibular disorientation and resulting driving phobias. Also, Jacob, Moller, Turner, and Wall (1985) noted a high incidence of otoneurological dysfunctioning in patients with panic disorder and agoraphobia with panic episodes.

In addition, Fenichel (1945) clearly described clinical association of anxiety neuroses, height and vehicle phobias with such vestibular symptoms as vertigo, imbalance, and motion sickness. Although Fenichel believed these anxiety-related symptoms were based in early childhood conflicts secondarily involving erotic vestibular stimulation, an alternative neurophysiological hypothesis seemed more likely. In other words, it appeared simpler to assume (and test the assumption) that a primary CV-dysfunction resulting in vertigo, imbalance, and motion sickness may secondarily trigger corresponding anxieties about losing control, fears of height or losing one's balance, and fears of motion-related activities or moving vehicles.

A relationship between cerebellar-vestibular (CV) dysfunction and fears/phobias as well as associated anxiety states first seemed possible to the author in the early 1970s as a result of a chance series of clinical observations. CV-dysfunctioning dyslexic children and adults experimentally treated with CV-stabilizing antimotion sickness and related medications unexpectedly reported significant improvements in their fears/phobias, anxiety and frustration levels, mood and self-esteem, and occasionally even obsessive-compulsive symptoms (Frank & Levinson, 1976-77, 1977; Levinson, 1980, 1984). Although these improvements were most often associated with favorable academic responses, at times only academic or only balance and coordination, or only anxiety benefits were reported, suggesting that responses to the medication were often function- and symptom-specific and so of a primary rather than secondary nature. Paradoxically, phobics in psychoanalytic psychotherapy were less fortunate. Despite experiencing significant intrapsychic and interpersonal improvements, their fears/phobias and anxiety or panic symptoms most often remained as impervious to treatment as they were to a meaningful explanation, regardless of effort and desire (Levinson, 1980). Unfortunately, although behavior modification and pharmacotherapy were eventually recognized to offer phobics statistically a greater chance for relief than psychotherapy (Sheehan, 1983), all theories corresponding to these dominant treatment modalities remained equally unsuccessful in explaining the shape, form, combinations, and clinical variations characterizing a majority of anxiety symptoms, including those defining the DSM-III—R categories. Obviously, something crucial appeared missing from the traditional understanding of anxiety disorders.

The above-mentioned relation of cerebellar-vestibular dysfunctioning to phobic or anxiety disorders was eventually restated as an hypothesis when

the very same CV-related sensorimotor mechanisms determining learning disabilities or dyslexic symptoms (de Quiros, 1976; Ayers, 1972; Kohen-Raz, 1988; Levinson, 1980, 1988, 1989a) were found shaping, codetermining, and readily explaining a wide array of anxiety symptoms (Levinson, 1986, 1989b, 1989c). For example, (1) *Imbalance mechanisms* often led to fears of heights, bridges, steps, falling, tripping, escalators, walking across wide-open spaces or intersections as well as fears of dizziness and losing control. (2) *Dyscoordination mechanisms* triggered fears of driving, sports, swimming, water, running, writing, and speaking (especially in public or social circumstances) as well as fears of walking or navigating across a busy intersection with no one or nothing to hold on to while having to turn one's head and judge distance, speed, and direction of criss-crossing cars.

(3) *Disturbances of muscle tone* such as "jelly legs" often triggered or reinforced anxieties of walking alone for fear of falling and losing control. Even the head bobbing sometimes reported among those anxiety-disordered appeared to result from unstable tone within the head and neck support muscles. (4) *Impaired motion-processing* resulted in either "motion-sickness" and/or motion-anxiety responses to specific motion vectors and so readily explained fears of moving elevators, escalators, trains, planes, buses, and even the motion characterizing walking, and/or the fears triggered by such movement-confining situations as standing, sitting or lying still, as well as confining and restricting crowds. (5) *Impaired proprioception* underlay and explained a series of body-image illusions which were often unconsciously displaced outward and experienced as the floor tipping, rocking, being soft like "mush," and feeling or anticipating "imaginary holes" in floors, sidewalks, bridges, buildings, planes.

(6) *Disturbances in orientation (compass functions)* may result in disorientation, spatial-temporal confusion, and a corresponding fear of new places, new situations, getting lost, and traveling alone. (7) *Sensory overloading*, real or relative, may result in photophobic or acoustic "crowd" phobias triggered by such visual signals as fluorescent light, sun, flickering, specific colors (occasionally relieved by tinted glasses), and such noise signals as screeching brakes, thunder, and "loud" social gatherings. Specific tactile or steady (constant) emotional stimuli may result in contact and "relationship or commitment phobias" and claustrophobic-like anxiety. Even olfactory, barometric, and related sensory stimuli were noted to be phobic triggers. (8) *Sensory deprivation*, real or relative, triggered by shielding environments such as rooms without windows, underwater, darkness, tunnels and subways, steel elevators, and crowds, may result in "claustrophobic" anxiety.

(9) *Perseveration mechanisms* may result in the repetitive thoughts and actions characterizing obsessions and compulsions as well as the inability to inhibit, erase, or forget traumatic experiences, including those triggered by

anxiety or panic attacks. (10) *Disturbances in the regulation of anxiety* were analogous to (and often overlapping with) the mechanisms modulating "typical" responses of motion sickness and tended to vary from exaggerated (panic) to absent (psychopathy), and repetitive or perseverative to singular episodes. (11) *Secondary destabilization* of the autonomic nervous system, dysautonomia (sweating, palpitations, temperature changes, etc.), and dysregulation of the reflex (swallowing and breathing) centers in the medulla oblongata (Carr & Sheehan, 1984) and the nucleus coeruleus (Redmond, 1977; Redmond & Huang, 1979) as well as the anticipatory anxiety triggered by cerebral cortical sensitization readily explained a host of secondary or associated anxiety-related symptoms. Although these CV-based mechanisms of anxiety disorders were carefully dissected and described in "pure" form (Levinson, 1980, 1986, 1989c), all such symptoms and samples were often variable, overdetermined mixtures and combinations or resultants of three major functional determinants, Type I: realistic, traumatic or conditioned determinants, Type II: neurotic determinants, and Type III: CV-based determinants. Type IV or primary nonCV-neurochemical and neurophysiological-determined or triggered fears/phobias/anxiety states exist; however, Type IV mechanisms were not significantly or obviously manifest in this and prior samples studied by the author (Levinson, 1986, 1989c). Accordingly, the discussion of Type IV fears/phobias was deferred pending further investigation.

In addition, the hypothesis relating CV-dysfunction and anxiety disorder was further strengthened by a diverse series of clinically related observations. (1) All medications demonstrated to be effective in treating anxiety disorders (antipanic, antidepressants, and beta-blockers) shared a common neurophysiological denominator with the antimotion sickness and related medications. All appear clinically to *stabilize CV-functioning* (McClure, Lycett, & Baskerville, 1982; Bastecky, Boleloucky, & Skovronsky, 1981; Levinson, 1986). (2) Any of these same therapeutic medications may trigger or exacerbate phobic and anxiety (or CV-related) symptoms in predisposed individuals, suggesting the medications are all targeting primary neurophysiological centers and mechanisms vital for modulating anxiety disorders. (3) Vertigo, disorientation, "fogginess," imbalance, and proprioceptive disturbances tend to characterize panic disorder, agoraphobia, and CV-dysfunction. (4) All of the above symptoms were occasionally triggered by caloric and rotation stimulation as well as optokinetic examination—diagnostic modalities known to stress and test the level of CV-functioning or dysfunctioning (Levinson, 1980, 1986). (5) Anxiety disorders are occasionally triggered by ear and sinus infections, allergies, TMJ, abscessed molars and/or their removal, barometric pressure and related changes, mononucleosis associated with vertigo, surgical procedures and peri-

lymph fistulas, perilymphatic hypotension and/or Ménière's syndrome affecting the middle- and inner-ear systems as well as trauma to the head, neck, and ear (Levinson, 1986). (6) Anxiety disorders may spontaneously improve or remit following treatment of such apparently unrelated disorders as sinus, ear, and throat infections, allergies, vertigo, etc. (Levinson, 1986).

Although anxiety disorders are classified into distinct and separate categories in the DSM-III—R (American Psychiatric Assn, 1987) diagnostic criteria, a significant number of patients with anxiety disorders were clinically noted to share symptoms and diagnostic criteria that fitted multiple DSM-III—R categories, often rendering a singular diagnosis difficult or impossible. In other words, simple phobias may also be associated with social phobias with and without panic disorder. Agoraphobics frequently show both simple and social phobias with and without panic disorder; and all DSM-III—R anxiety categories may evidence clinically some obsessive/compulsive symptomatology. This DSM-III—R diagnostic overlap suggested one of two explanatory possibilities: (1) that all or most of the anxiety disorders share common denominators and that the corresponding anxiety-related symptoms and DSM-III—R categories characterizing a given individual and sample are determined by varying combinations and intensities of related, overlapping Types I, II, and III (and/or IV) mechanisms or (2) that each and every symptom of anxiety disorder and DSM-III—R category characterizing a given individual represents a unique and separate disorder.

As previously described, only CV-related mechanisms appeared capable of explaining the diverse shapes and forms of a majority of the symptoms characterizing the various DSM-III—R diagnostic categories for anxiety disorder and so also their combinations and diagnostic overlap per patient and sample. In view of the unlikely chance that agoraphobics with and without panic disorder who also experience simple and social phobias as well as obsessions/compulsions and generalized anxiety, have multiple, etiologically unrelated and separate disorders responsible for each and every symptom, all anxiety-related DSM-III—R diagnostic categories were assumed to be significantly CV-based or predisposed unless proven otherwise.

Current Research Aims

In this study, a specific attempt was made to test the assumption and hypothesis (1) that CV-dysfunction, as documented by neurological and ENG testing, characterizes anxiety-disordered samples, regardless of DSM-III—R diagnostic category; (2) that any given DSM-III—R diagnosed anxiety-disordered sample of significant size contains coexisting anxiety-related symptoms sufficient to overlap with and diagnose most other DSM-III—R categories. It was anticipated that a significant diagnostic overlap among the various DSM-III—R categories of anxiety disorder would strongly support the presence of a common denominator of anxiety disor-

ders, and a high incidence of both CV-dysfunction and learning disabilities (previously correlated with CV-impairment) (Levinson, 1988) among the various DSM-III—R categories would strongly support the assumption that this common denominator is CV-based.

Method

Sample

Using DSM-III—R criteria, 402 consecutively referred subjects who were between the ages of 19 to 50 yr., showed severe phobias and related anxiety disorders, and completed neurological and ENG testing, were separated into eight primary diagnostic categories: panic disorder with agoraphobia (44.5%), agoraphobia without a history of panic disorder (7.5%), panic disorder without agoraphobia (3.5%), simple phobias (23.4%), social phobias (14.9%), obsessive/compulsive disorder (4.0%), generalized anxiety disorder (1.7%), and posttraumatic anxiety disorder (0.5%). Although many of the patients in this study were referred after a publication (Levinson, 1986) appeared describing the relationship between CV-dysfunction and phobias (so the possibility of biased referrals and sampling must be contemplated), the quality and phobic distribution of the anxiety-disordered sample presented were noted to be similar to that originally used in deriving the hypothesis of CV-based phobias (Levinson, 1980). All referrals prior to the availability of the 1987 DSM-III—R classification were either rediagnosed or excluded from this study. Of the total sample, 33.8% were pharmacologically treated by other physicians prior to referral and required small amounts of antipanic and antidepressant medication during the testing. This made necessary comparison of the neurological and ENG (and optokinetic) parameters in subjects with and without medications.

The mean age of the patients was 36.5 yr. ± 11.1; ages ranged from 19 to 50 yr. The female/male ratio was 1.6/1, and the complete right-handed/complete left-handed/mixed-handed percentages and ratios were 80.4%, 13.8%, 5.8%, and 13.9/2.4/1.0, respectively. Mixed-handedness was stated to be present when a given individual self-reported performing one or more functions as well as or better than with the nondominant hand. For example, a subject who could naturally eat, write, bat, throw or catch, etc. as well or better with the nondominant as with the dominant hand was considered mixed-handed. The remainder were either completely right-handed or completely left-handed. In further studies, handedness must be qualitatively and quantitatively rated according to the criteria utilized by Briggs and Nebes (1975) so more accurate comparisons can be made with random samples and degrees of handedness can be studied. "Normal" subjects should then also be included.

Procedure

Diagnosis of CV-dysfunction.—All 402 anxiety-disordered subjects in this study were examined for CV-dysfunction using neurological examinations and ENG. Patients were also examined using an optokinetic fixation and tracking method (Levinson, 1980, 1986).

Neurological testing.—Standard neurological examinations were given to all subjects. As CV-impaired individuals most frequently employ ocular fixation and concentration mechanisms to compensate for impaired sensorimotor functions (Levinson, 1980), all subjects were tested in a way which minimized compensatory techniques and maximized the emergence of abnormal CV signs. Patients were examined for dysdiadochokinesis and finger-to-nose and finger-to-thumb sequencing with eyes closed and upon distraction. In addition, eyes-closed Romberg testing was intensified when patients were instructed to balance themselves on one foot or in the monopedal stance (Levinson, 1980).

ENG testing.—The ENG technique and evaluation are based on that used by Kenneth Brookler in his private practice and while he was Chief of Otology and Neurotology at Lenox Hill Hospital in New York City and is also consistent with that used by Noel Cohen, Chairman of Otolaryngology at New York University Medical Center. The examination consisted of positional testing for horizontal and vertical spontaneous and position-triggered nystagmus as well as monaural (alternate binaural) and simultaneous bithermal caloric stimulation using water at 30°C and 44°C.

Positional testing was performed on all individuals with the eyes closed using the supine 0° head up, head right, head left, right lateral and left lateral positions as well as the supine 30° position with head and neck straight ahead. (Head hanging and right and left Hallpike positions were not tested.) Nystagmus was considered inconsistent with a normal vestibular system when three consecutive beats per 10-sec. period were recorded in any given position. The monaural or alternate bithermal and simultaneous bithermal caloric responses were measured for unilateral weakness and directional preponderance. Unilateral vestibular weakness or reduced vestibular response (RVR) was defined as a difference of 30% or more in slow phase velocity on stimulation of the right versus left ears or as a "Type II" response on simultaneous caloric stimulation. Directional preponderance (DP) was defined as a difference of at least 30% in right- versus left-beating nystagmus, corresponding to a "Type III" response. "Type IV" responses (characterized by inconsistent vestibular responses to simultaneous binaural warm and cool water) were considered to be abnormal but of nonlocalizing and nonspecific nature. The exact details of this ENG technique are available elsewhere (Jongkees, Mass, & Philipszoon, 1962; Brookler, 1971; Levinson, 1980).

The incidence of ENG-detected CV-dysfunction in this sample would have been significantly higher had posturography been performed and rotation stimulation data counted. Romberg positional nystagmus is a relatively newly researched parameter (Levinson, 1980) and so is scored separately and not counted in the present statistical analysis. According to Brookler (in a personal correspondence), the presence of any one neurological or ENG parameter is consistent with CV-dysfunction. Using more conservative diagnostic criteria, however, CV-dysfunction was also defined and reported on the basis of two or more abnormal neurological or ENG parameters per subject.

DSM-III—R "Diagnostic Overlap" in anxiety disorder.—To demonstrate the presence of a DSM-III—R "Diagnostic Overlap," each of the 402 subjects was psychiatrically interviewed to ascertain all of their current anxiety-related symptoms as well as the corresponding ages of onset. Before the first visit, each subject was required to write or dictate a complete history of the onset and clinical course of their presenting anxiety disorder as well as the existence of past anxiety-related symptoms and those still present. Whenever possible, they were encouraged to state what they thought were important triggers and determining mechanisms. The most severe and bothersome of presenting symptoms were used for a primary diagnosis according to DSM-III—R criteria. At times, however, this decision was difficult for there appeared to be two or more "primary" DSM-III—R diagnostic anxiety categories affected. All primary and remaining or coexisting (nonprimary) anxiety symptoms were recorded for each subject. In addition, each subject was required to complete a questionnaire containing 45 of the most common anxiety symptoms previously found to characterize more than 4,000 CV-dysfunctional and learning disabled individuals of varying ages (Levinson, 1989c).[2] The clinical, written, and form data were then integrated and read into a computer. The primary diagnostic DSM-III—R anxiety symptoms aside, all remaining or coexisting anxiety-related symptoms characterizing a given subject and sample were used for secondary, tertiary or nonprimary overlapping DSM-III—R diagnoses. As a result, the extent to which any sample with DSM-III—R anxiety disorder overlapped with other DSM-III—R categories could readily be determined. Needless to say, considerable overlap would strongly suggest that anxiety disorders share common predisposing or determining denominators. Moreover, the DSM-III—R diagnostic criteria for anxiety disorder were reviewed and the acknowledged overlap was recorded.

[2]This fear/phobia questionnaire is on file with Microfiche Publications in Document NAPS-04640. Remit $12.25 for photocopy or $4.00 for fiche to Microfiche Publications, POB 3513, Grand Central Station, New York, NY 10017.

Coexistence of Learning Disabilities

A past and present history of symptoms of learning disability was obtained from each subject so that the approximate incidence (and symptomatic distribution) of learning disabilities could be obtained for the total anxiety-disordered sample and for each of the diagnostic categories. Learning disability is characterized by symptoms significantly affecting reading, writing, spelling, mathematics, memory, speech, sense of direction and time, simple grammar, concentration, and activity level. As noted earlier, past studies have shown strong associations of learning disability with CV-dysfunctioning sensorimotor mechanisms and related abnormal neurological and ENG (and optokinetic) parameters (Frank & Levinson, 1973; Levinson, 1980, 1988, 1989a).

Results

CV-dysfunction and Anxiety Disorder

The distribution of DSM-III—R anxiety disorders characterizing 402 consecutively referred subjects who completed the diagnostic testing as well as the corresponding abnormal CV-determined neurological and ENG parameters is detailed in Tables 1 and 2. As noted, CV-dysfunction was arbitrarily and conservatively defined on the basis of ≥ 2 abnormal parameters per neurological or ENG diagnostic category per subject. Accordingly, 94% evidenced CV-dysfunctioning on the basis of neurological or ENG examinations, 81.8% neurological dysfunctioning, and 61.6% ENG dysfunctioning. On the basis of one or more abnormal neurological or ENG parameter per subject, 99.5% of the sample evidenced CV-dysfunction. In a follow-up study (Levinson, 1989b), anxiety-disordered patients were tested using an optokinetically-based tracking methodology shown to be effective in differentiating CV-dysfunctioning individuals with learning disabilities from controls (Levinson, 1989a). By adding this CV-determined optokinetic diagnostic method to the neurological and ENG testing, the incidence of diagnosable CV-dysfunctioning parameters was significantly increased.

CV-dysfunction and DSM-III—R Categories of Anxiety Disorder

As noted in Tables 1 and 2, all DSM-III—R categories of anxiety disorder showed high percentages of abnormal neurological and ENG parameters, regardless of the number of subjects. Although the data in DSM-III—R categories with only a few subjects (panic disorder, generalized anxiety disorder, posttraumatic anxiety disorder) could not be evaluated statistically, the presence of CV-dysfunctioning appears clinically significant and most certainly justifies further studies of larger samples. Using chi-squared, all neurological and ENG parameters were independent of DSM-III—R categories (when of sufficient size), sex, and handedness. There were

TABLE 1
CEREBELLAR-VESTIBULAR NEUROLOGICAL PARAMETERS IN ANXIETY DISORDER

Signs	Total		PA, Agor		Agor, no PA		Social		Simple	
	N	%	*n*	%	*n*	%	*n*	%	*n*	%
Total Sample	402		179		30		60		94	
1 or More Signs	382	95.0	170	95.0	28	93.3	57	95.0	89	94.7
2 or More Signs	329	81.8	147	82.1	26	86.7	47	78.3	75	79.8
Ocular-Dysmetria	321	79.9	143	79.9	22	73.3	50	83.3	72	76.6
Romberg-Monopedal	279	69.4	124	69.3	25	83.3	36	60.0	67	71.3
Dysdiadochokinesis	68	16.9	34	19.0	6	20.0	9	15.0	12	12.8
Finger to Nose	143	35.6	58	32.4	15	50.0	22	36.7	31	33.0
Finger to Finger	222	55.2	100	55.9	17	56.7	34	56.7	50	53.2
Tandem Dysmetria	144	35.8	65	36.3	11	36.7	20	33.3	30	31.9
Tremor	8	2.0	3	1.7	1	3.3	1	1.7	2	2.1
Hypotonia	5	1.2	2	1.1	1	3.3	1	1.7	1	1.1

	OBS/ COMP		PA, no Agor		Gen. Anxiety		Post-traumatic	
	n	%	*n*	%	*n*	%	*n*	%
Total Sample	16		14		7		2	
1 or More Signs	15	93.8	14	100.0	7	100.0	2	100.0
2 or More Signs	13	81.3	13	92.9	6	85.7	2	100.0
Ocular-Dysmetria	13	81.9	12	85.7	7	100.0	2	100.0
Romberg-Monopedal	9	56.3	10	71.4	6	85.7	2	100.0
Dysdiadochokinesis	2	12.5	4	28.6	1	14.3	0	0.0
Finger to Nose	5	31.3	8	57.1	3	42.9	1	50.0
Finger to Finger	7	43.8	7	50.0	5	71.4	2	100.0
Tandem Dysmetria	6	37.5	7	50.0	3	42.9	2	100.0
Tremor	0	0.0	0	0.0	1	14.3	0	0.0
Hypotonia	0	0.0	0	0.0	0	0.0	0	0.0

Note.—PA, Agor is Panic Disorder with Agoraphobia; Agor, no PA is Agoraphobia without a history of panic disorder; PA, no Agor is Panic Disorder without Agoraphobia; Gen. Anxiety is Generalized Anxiety Disorder.

no significant differences noted in the neurological and ENG (and optokinetic) parameters for medicated vs nonmedicated anxiety-disordered patients. On a two-tailed *t* test, no parameter met a probability level of .01. As a result, the medicated and nonmedicated groups were combined for statistical analysis.

Learning Disabilities and Anxiety Disorders

Ninety-two percent of the anxiety-disordered patients had a past or present history of learning disabilities consistent with the definition utilized in Public Law 94-142 (The Education for All the Handicapped Children Act). Deficits were experienced in one or more functions affecting reading, writing, spelling, mathematics, memory, speech, simple grammar, concentration, activity level, time and direction as well as associated motor difficulties

TABLE 2
CEREBELLAR-VESTIBULAR ENG PARAMETERS IN ANXIETY DISORDER

Signs	Total		PA, Agor		Agor, no PA		Social		Simple	
	N	%	*n*	%	*n*	%	*n*	%	*n*	%
Total Sample	402		179		30		60		94	
1 or More Signs	374	93.0	163	91.1	28	93.3	54	90.0	91	96.8
2 or More Signs	248	61.6	100	55.9	24	80.0	35	58.3	59	62.8
Positional Nystagmus										
Total	351	87.3	153	85.5	28	93.3	51	85.0	84	89.4
H. Nystagmus	244	60.6	109	60.9	24	80.0	33	55.0	55	58.5
V. Nystagmus	307	76.3	137	76.5	20	66.7	44	73.3	75	79.8
Romberg Nystagmus	283	70.3	119	66.5	21	70.0	50	83.3	69	73.4
Romb. or Pos. Nyst.	371	92.2	162	90.5	28	93.3	56	93.3	88	93.6
Caloric Testing										
Total	30	7.46	16	8.9	4	13.3	2	3.3	5	5.3
R.V.R*	17	4.22	10	5.6	2	6.7	2	3.3	3	3.2
D.P.*	18	4.47	9	5.0	3	10.0	0	0.0	3	3.2
Simultaneous Bithermal Testing										
Total	259	64.4	103	57.5	23	76.7	38	63.3	63	67.0
Type 2	97	24.1	39	21.8	10	33.3	9	15.0	23	24.5
Type 3	36	9.0	13	7.3	3	10.0	7	11.7	9	9.6
Type 4	127	31.5	52	29.1	10	33.3	22	36.7	31	33.0

	OBS/ COMP		PA, no Agor		Gen. Anxiety		Post-traumatic	
	n	%	*n*	%	*n*	%	*n*	%
Total Sample	16		14		7		2	
1 or More Signs	15	93.8	14	100.0	7	100.0	2	100.0
2 or More Signs	15	93.8	10	71.4	4	57.1	1	50.0
Positional Nystagmus								
Total	15	93.8	12	85.7	7	100.0	1	50.0
H. Nystagmus	11	68.8	7	50.0	4	57.1	1	50.0
V. Nystagmus	11	68.8	12	85.7	7	100.0	1	50.0
Romberg Nystagmus	10	62.5	8	57.1	5	71.4	1	50.0
Romb. or Pos. Nyst.	15	93.8	13	92.9	7	100.0	2	100.0
Caloric Testing								
Total	1	6.3	1	7.1	0	0.0	1	50.0
R.V.R.*	0	0.0	0	0.0	0	0.0	0	0.0
D.P.*	1	6.3	1	7.1	0	0.0	1	50.0
Simultaneous Bithermal Testing								
Total	15	93.8	11	78.6	4	57.1	2	100.0
Type 2	9	56.3	4	28.6	2	28.6	1	50.0
Type 3	1	6.3	2	14.3	0	0.0	1	50.0
Type 4	5	31.3	5	35.7	2	28.6	0	0.0

Note.—PA, Agor is Panic Disorder with Agoraphobia; Agor, no PA is Agoraphobia without a history of panic disorder; PA, no Agor is Panic Disorder without Agoraphobia; Gen. Anxiety is Generalized Anxiety Disorder. The Romberg Positional parameter is a developing research parameter. It is included in the table but not included in the over-all statistical analysis.
*R.V.R.—Reduced vestibular response; D.P.—Directional preponderance.

in balance, coordination, and rhythm. Ten percent of the symptoms of learning disability occurred *only after* the onset of anxiety disorders, clinically suggesting a relationship. This association was reinforced when a significantly greater percentage of subjects noted an intensification or extension of prior symptoms of learning disability with the onset of the anxiety disorder. However, it was sometimes difficult to decide clinically whether this intensification or extension was of a primary or secondary origin. Follow-up studies will attempt to distinguish patterns of cerebellar-vestibular diagnostic parameters crucial for anxiety disorders vs learning disabilities.

Diagnostic Overlap

Each DSM-III—R diagnostic category with a reasonably large sample contained a variety of coexisting anxiety-related symptoms sufficient to diagnose and overlap most other DSM-III—R categories for anxiety disorder. These categories included panic disorder with and without agoraphobia, agoraphobia without a history of panic disorder, simple phobias, social phobias, and obsessions/compulsions. The anxiety disorders with DSM-III—R overlap discussed so far are clearly highlighted by the frequency distribution of

TABLE 3
DSM III—R Diagnostic Overlapping in Anxiety Disorder

Primary DSM III—R Diagnoses	Total Subjects $N = 402$		Frequency of Overlapping Symptoms or Categories					
			Social		Simple		OBS/COMB	
	N_p	$(N_p/n)100$ %	n	$(n/N_p)100$	n	$(n/N_p)100$	n	$(n/N_p)100$
PA, Agor	179	44.5	28	15.6	45	25.1	56	31.4
Agor, no PA	30	7.5	5	16.7	7	23.3	8	25.6
Social	60	14.9			59	98.3	14	22.6
Simple	94	23.4	15	16.0			20	20.8
OBS/COMP	16	4.0	3	18.8	14	87.5		
PA, no Agor	14	3.5	1	7.1	11	78.6	4	29.8
Gen. Anxiety	7	1.7	0	0.0	6	85.7	2	28.6
Posttraumatic	2	0.5	0	0.0	1	50.0	2	100.0

Note.—N_p refers to the subtotal of subjects with a primary DSM III—R diagnosis; n refers to subsamples of N_p with overlapping anxiety symptoms. PA, Agor is Panic Disorder with Agoraphobia; Agor, no PA is Agoraphobia without a history of Panic Disorder; PA, no Agor is Panic Disorder without Agoraphobia; Gen. Anxiety is Generalized Anxiety Disorder.

the primary and overlapping anxiety symptoms or diagnostic categories characterizing this sample (Table 3). By DSM-III—R criteria or definition, panic with agoraphobia, agoraphobia without a history of panic disorder, and panic disorder without agoraphobia, are mutually exclusive of one another and so cannot overlap. Inasmuch as sufficient data for panic disorder without history of agoraphobia as well as posttraumatic and generalized anxiety

disorders are unavailable, these three categories cannot be discussed meaningfully.

As noted in Table 3, (1) *panic disorder with agoraphobia* significantly overlapped social phobia (15.6%), simple phobia (25.1%), and obsessive/compulsive disorder (31.4%), and (2) *agoraphobia without a history of panic disorder* significantly overlapped social phobia (16.7%), simple phobia (23.3%), and obsessive/compulsive disorder (25.6%). As agoraphobic states include a wide variety of "simple" phobias, the percent of overlapping non-agoraphobic "simple" phobias is not as high as might otherwise have been anticipated. Inasmuch as the agoraphobic and panic disorder categories are often the most severe and restricting in nature, they are invariably used clinically for a primary DSM-III—R diagnosis even when they coexist or overlap severe forms of simple and social phobias or obsessive/compulsive symptoms. Considering this, the overlap for the latter three primary DSM-III—R categories are reported only for one another: (1) *social phobias* significantly overlapped simple phobias (98.3%) and obsessive/compulsive symptoms (22.6%), (2) *simple phobias* significantly overlapped social phobias (16%) and obsessive/compulsive symptoms (20.8%), and (3) *obsessive/compulsive disorder* significantly overlapped social phobias (18.8%) and simple phobias (87.5%).

A careful review of the reported differential diagnostic DSM-III—R criteria for anxiety disorders showed significant acknowledged diagnostic overlap. For example, panic disorder may occur with and without agoraphobia and in association with social and simple phobias as well as generalized anxiety disorder. Also, simple and social phobias may coexist with agoraphobia, regardless of whether panic disorder is present. The recognizable overlap or unity characterizing anxiety disorders would be significantly greater: (1) if the phobic avoidance of specific situations (especially dirt or contamination) noted in obsessive/compulsive disorder were not by definition excluded from other simple phobias; (2) were social phobia not defined as distinct from simple phobia although both share common characteristics; and (3) if agoraphobia were viewed as a syndrome combining multiple simple phobias. This underlying unity of varying surface symptoms is further highlighted by the fact that depressive disorder is stated to be associated with or to overlap anxiety disorder and its various DSM-II—R categories. The common denominators predisposing subjects to both anxiety disorder and depression have been explored in prior work (Levinson, 1989c).

Discussion

The hypothesis suggesting the presence of a CV-predisposition in anxiety disorders appeared significantly supported by the analysis of CV-dysfunction which indicated the following: (1) a high incidence of CV-impairment (as measured by neurological and ENG parameters) in anxiety

disorders, regardless of DSM-III—R category, (2) a significant correlation of CV-based phobic mechanisms and symptoms of learning disability with those of anxiety disorders, and (3) the onset or intensification and extension of learning disability symptoms with the onset of anxiety disorders, (4) a high symptomatic or diagnostic overlap among most DSM-III—R categories of anxiety disorders. Although the above observations do not justify convictions of cause and effect, they do provide insight into the possible mechanisms which appear to shape and determine the final symptomatic outcome and DSM-III—R categories characterizing subjects with varying anxiety disorders. Needless to say, additional controlled studies, including "normal" or asymptomatic subjects, are required for both validation and elucidation of the determining vs compensatory mechanisms and diagnostic parameters characterizing specific symptoms. However, these studies entail complex, multidimensional considerations. For example, (1) given the crucial but relatively hidden role of compensatory mechanisms, it is anticipated that a significant percentage of asymptomatic or "normal" subjects will show diagnostic evidence of cerebellar-vestibular dysfunction; (2) as CV-dysfunctioning appears to predispose individuals to a wide variety of overlapping fears/phobias and depression as well as learning disabilities and symptoms such as vertigo, motion sickness, etc., these follow-up studies will require the use and comparison of very highly screened and selected CV-determined monosymptomatic samples and CV-normal asymptomatic controls for statistical analysis to be meaningful. Indeed, these studies may show a need to develop a crucial set of additional diagnostic parameters and to apply sophisticated statistical methods to facilitate the recognition of underlying associations.

To explain simply most of the clinical and experimental data which characterize this relatively large sample of patients with anxiety disorders, a holistic theory was formulated and summarized. The anxiety-related symptoms for each diagnostic category as defined by DSM-III—R were postulated to be vector resultants of a dynamic equilibrium among groups of predisposing Types I, II, and especially Type III (and/or IV) triggers and determining mechanisms of varying intensities vs compensatory forces. One may account for both the apparent unity of anxiety disorders as a group as well as the significant surface and related variations which characterize individual subjects and samples. The apparent vulnerability of any large sample to predictable patterns and frequencies of phobic responses and triggers (Levinson, 1980, 1984, 1986) suggested the presence of predisposing built in releasing mechanisms and corresponding releasing triggers (Lorenz, 1957). Among the many and varied phobic triggers, Pitts and McClure (1967) and Levin, Liebowitz, Fyer, Gorman, and Klein (1987) reported sodium lactate infusion and CO_2 inhalation, respectively, released panic attacks in subjects

already predisposed to this disorder. Accordingly, it appeared to the present author that some phobics may intuitively or instinctively avoid crowded and/or closed-in or claustrophobic surroundings where CO_2 levels (and/or decreased O_2 levels) might trigger panic attacks and related symptoms just as others might avoid exercise or significant physical activity to minimize serum lactate levels. In retrospect, emotional conflict and stress (Type II mechanisms), realistic, traumatic and anticipatory anxiety (Type I mechanisms) as well as panic attacks of any origin may further destabilize impaired CV-functioning and trigger Type III (and/or IV) anxiety mechanisms and symptoms. As a result, behavior modification and psychotherapy as well as pharmacotherapy are helpful for some Type III (and/or Type IV) phobic persons. Each of these therapies attempts to compensate for corresponding Types I, II, and III (and/or IV) anxiety mechanisms and resulting symptoms (Levinson, 1986, 1989b, 1989c).

In conclusion, a clinically supported hypothesis of a cerebellar-vestibular basis for anxiety disorders is described. This hypothesis appears capable of explaining, encompassing, and unifying the diverse array of symptoms and data, theories, and therapies characterizing the Anxiety Disorder syndrome. Also, ensuing insights have resulted in: (1) a new classification of anxiety disorders based on determining and shaping of functional Types I, II, and III (and/or IV) mechanisms rather than on surface descriptions, i.e., the DSM-III—R classification, (2) differential dissection and selective treatment of the various interacting and overdetermining mechanisms of anxiety disorder, and (3) the use of antimotion sickness and related CV-stabilizing medications alone and in conjunction with the now traditional forms of pharmacotherapy (antidepressants, antipanic and beta-blocker medications), psychotherapy and behavior modification.

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Accepted January 10, 1989.