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A CEREBELLAR-VESTIBULAR EXPLANATION FOR FEARS/PHOBIAS: HYPOTHESIS AND STUDY [1, 2]

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Summary.—To clarify and test the cerebellar-vestibular (CV) basis of fears/phobias, responses of 4000 learning disabled children, adolescents, and adults with neurological and electronystagmographic (ENG) evidence of CV-dysfunction were analyzed for anxiety-related symptoms. Of this sample, 64.6% indicated fears/phobias; females were significantly more predisposed; mixed-handedness was significantly related to fears of heights and reduced vestibular response or asymmetric vestibular functioning. Also, adults had a higher incidence of the specific fears/phobias characterizing agoraphobia than children and adolescents. Analysis of factors reported as triggering the fears/phobias led to (1) a classification and theory of fears/phobias, obsessions/compulsions, and related anxiety symptoms based on realistic or traumatic, neurotic, and CV- or other CNS-based mechanisms rather than on DSM-III—R surface descriptions; (2) an understanding of the relationships between mitral valve prolapse, agoraphobia and panic episodes, as well as depression; and (3) new insights into differential diagnosis and selective treatment.

A relationship between cerebellar-vestibular (CV) dysfunction and fears/phobias and anxiety was first apparent to the author as a result of a chance clinical observation. Learning disabled or dyslexic individuals with CV-dysfunction unexpectedly reported improvements in a wide array of seemingly unrelated fears/phobias and anxiety states following experimental treatment with CV-stabilizing antimotion sickness medications intended to improve only their academic symptoms (Frank & Levinson, 1977; Levinson, 1980). This apparent relationship led to an hypothesis that CV-dysfunction was the basis for observed phobias when (1) the sensory-motor mechanisms found triggering these fears/phobias were noted to be similar, if not identical, to the CV-determined mechanisms shown to be responsible for shaping the academic and related symptoms in learning disabilities (Levinson, 1980, 1984, 1986) and (2) phobic patients in psychotherapy for 1 to 2 yr. were belatedly found to have histories and presence of learning disabilities and CV-dysfunction (Levinson, 1980). (3) The antimotion-sickness medications and all the existing categories of antipanic and antiphobic medications had a

[1]This paper, originally entitled The Cerebellar-Vestibular Basis of Fears/Phobias, was presented in part at the American Psychiatric Association meeting in Chicago, Illinois, May 1987 and at the First International Conference of Neurological Dysfunction, Chester, England, October 1987. The author acknowledges the staff of The Medical Dyslexic Treatment Center for their help during the preparation of this manuscript. Several members deserve special thanks for their assistance in the research and data analysis, i.e., John Lim, Ming Tang, Linda Gerry, Kenny Moss, and Rebecca Hsu.

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common CV-stabilizing function (Levinson, 1986; McClure, Lycett, & Baskerville, 1982; Bastecky, Boleloucky, & Skovronsky, 1981). (4) Caloric and rotation stimulation during ENG testing as well as a host of infections, traumatic, toxic, and allergic situations impairing CV or "inner ear" functioning occasionally triggered panic attacks and phobias in a manner analogous to the response of individuals predisposed to sodium lactate infusion (Levinson, 1986; Pitts & McClure, 1967).

Despite the dominance of psychoanalytic, conditioning, and biochemical research and corresponding theories of anxiety disorders (Klein, 1981), a review of the literature yielded a wide range of independently derived clinical evidence consistent with a cerebellar-vestibular basis for these very same symptoms. For example, Benedikt (1870) as early as 1870 suggested that agoraphobia may be of vestibular origin. Guye as well as Lannois and Tournier published similar findings in 1899. Years later Marks and Bebbington (1976) described space phobias as possible agoraphobic variants associated with vestibular dysfunction; and Blythe and McGlown (1979) highlighted vestibular mechanisms in agoraphobia and claustrophobia. Recently, Page and Gresty (1985) described motorists' vestibular disorientation and resulting driving phobias; and Jacob, Moller, Turner, and Wall (1985) noted otoneurological dysfunctioning in patients with panic disorder and agoraphobia with panic episodes.

In addition, Fenichel (1945) clearly described a clinical association of anxiety neuroses, height and vehicle phobias with such vestibular symptoms as vertigo, imbalance, and motion sickness. Albeit Fenichel believed these anxiety-related symptoms were due primarily to early childhood conflicts which secondarily involved erotic vestibular stimulation, an alternative neurophysiological hypothesis appeared more likely. Might not a primary CV-dysfunction resulting in vertigo, imbalance, and motion sickness secondarily trigger corresponding anxieties about losing control, fears of height or losing one's balance, and fears of motion-related activities or moving vehicles? Might not the analysis of other fears/phobias, especially those characterizing a CV-dysfunctioning population, highlight the existence of previously unsuspected CV-determining phobic vectors?

To clarify the presence of an hypothesized CV/phobic relationship, a series of analyses were undertaken. In this study, the fears/phobias of 4000 learning disabled individuals with significant neurological and ENG evidence of CV-dysfunction were (1) quantitatively assessed to estimate the prevalence and frequency in this highly selected CV-dysfunctioning population and their possible associations with sex, handedness, and age groups, namely, children, adolescents, and adults, (2) qualitatively analyzed to explore underlying CV- and nonCV-triggering, determining, or predisposing mechanisms, and (3) reclassified according to CV- and nonCV-functional

determinants. Follow-up studies are needed to analyze corresponding data obtained from matched random controls and CV-normal samples with fears/phobias. Comparative data from these control groups are considered essential for further elucidation and validation of the proposed CV-basis of fears/phobias.

METHOD

Procedures

Learning disabled patients and/or relatives were questioned by the examining psychiatrist during routine anamnesis for past or present evidence of fears/phobias and possible triggering or determining mechanisms. Data from 4,000 children, adolescents, and adult patients were recorded and both quantitatively and qualitatively analyzed. Inasmuch as most of these individuals experienced anxieties relating to school, tests, and academic subjects, and many children appeared "normally" anxious about the dark or being alone, these specific mild or "normal" fears were not counted in the statistics unless they were of significant or incapacitating intensity. All other mild fears were counted.

The data on fears were obtained from separate clinical interviews and a questionnaire which contained the 45 most common fears or phobias given by a similar sample. Additional fears were collected and totaled 189. Although no attempt was made to grade their intensity because the aim of the present analysis was to explore the nature of underlying functional mechanisms, most ($> 90\%$) of the reported fears were of a mild, nonrestricting nature. Indeed, it was anticipated that the mechanisms responsible for triggering mild fears would be easier to dissect than the overdetermined mechanisms assumed underlying severe, complicated phobic states.

The sample was that described in a prior paper entitled, The Cerebellar-Vestibular Basis of Learning Disabilities in Children, Adolescents and Adults: Hypothesis and Study (1988). The specific criteria required to diagnose learning disability and CV-dysfunction, using neurological and ENG testing, have been described elsewhere (Levinson, 1980; Brookler & Pulec, 1970). According to the criteria used by Dr. K. Brookler in his private practice and while Chief of Otology and Neurotology at Lenox Hill Hospital, New York City, any one abnormal CV-determined ENG or neurological (or optokinetic) parameter is consistent with CV-dysfunctioning. Because this sample was consistent in the quality of their academic-related symptoms and apparent determining mechanisms and 99.5% evidenced ≥ 1 (and 94.1% evidenced ≥ 2) abnormal ENG or other neurological parameters, it seemed reasonable to assume that the entire sample showed CV-dysfunction. The distribution of abnormal CV-determined neurological and

ENG parameters characterizing this sample are displayed in Tables 1 and 2 below, respectively.

TABLE 1

NEUROLOGICAL PARAMETERS AS A FUNCTION OF AGE IN CHILDREN, ADOLESCENTS, AND ADULTS

Neurological Parameter	Total		Children		Adolescents	
	N	%	7 to 12 yr.		13 to 18 yr.	
			n	%	*n*	%
Sample Size	4000		1465		1156	
1 or more signs	3582	96.3	1437	98.1	1092	94.4
2 or more signs	3265	81.6	1324	90.4	876	75.8
Ocular Dysmetria	3164	79.1	1201	82.0	858	74.2
Romberg-Monopedal	2366	59.2	917	62.6	601	52.0
Dysdiadochokinesis	987	24.7	549	37.5	225	19.5
Finger-nose	876	21.9	339	23.1	176	15.2
Finger-finger	2886	72.2	1272	86.8	756	65.4
Tandem Dysmetria	1380	34.5	702	47.9	285	24.7
Tremor	15	0.4	5	0.3	3	0.2
Hypotonia	0	0.0	0	0.0	0	0.0

	Adults							
	Total		19 to 30 yr.		31 to 40 yr.		41 to 50 yr.	
	n	%	*n*	%	*n*	%	*n*	%
Sample size	1379		784		331		264	
1 or more signs	1323	95.9	752	96.0	316	95.4	255	96.7
2 or more signs	1065	77.2	592	75.5	266	80.4	207	78.4
Ocular Dysmetria	1104	80.1	620	79.1	263	79.4	222	84.2
Romberg-Monopedal	848	61.4	448	57.1	227	68.6	173	65.5
Dysdiadochokinesis	213	15.4	124	15.8	50	15.1	39	14.8
Finger-nose	361	26.1	202	25.8	87	26.3	72	27.3
Finger-finger	858	62.2	488	62.2	211	63.7	159	60.2
Tandem Dysmetria	393	28.4	203	25.9	112	33.8	78	29.5
Tremor	7	0.6	5	0.6	3	0.8	0	0.0
Hypotonia	0	0.0	0	0.0	0	0.0	0	0.0

The ENG, an electrical recording technique, was used in objective detection and measurement of spontaneous and position-triggered nystagmus as well as nystagmus induced by caloric vestibular stimulation. Spontaneous and position-triggered nystagmus were tested by having subjects lie down with eyes closed in the following positions: supine 0° head up, head right, head left, right lateral and left lateral positions as well as in the supine 30° position with head and neck straight ahead. Nystagmus is considered present when three consecutive beats per 10-sec. period are recorded in any given position. Its presence is inconsistent with having a normal vestibular system. Monaural (alternate binaural) and simultaneous bithermal caloric vestibular stimulation used water at 30° C and 44° C.

TABLE 2
ENG PARAMETERS AS A FUNCTION OF AGE IN CHILDREN, ADOLESCENTS, AND ADULTS

ENG Parameter	Total		Children		Adolescents	
	N	%	7 to 12 yr.		13 to 18 yr.	
			n	%	*n*	%
Sample Size	4000		1465		1156	
1 or more signs	3836	95.9	1416	96.6	1120	96.9
2 or more signs	2788	69.7	1080	73.7	813	70.3
Positional Dysfunction	3515	87.9	1291	88.1	1035	89.5
H. Nystagmus	1912	47.8	657	44.8	532	46.0
V. Nystagmus	3241	81.0	1203	82.1	975	84.3
Caloric Dysfunction	387	9.7	134	9.1	104	9.0
Direct. Preponderance	208	5.2	77	5.3	56	4.8
Reduced Vestib. Response	235	5.9	78	5.3	63	5.4
Simult. Cal. Dysfunction	3000	75.0	1170	79.9	869	75.2
Type 2*	1095	27.4	448	30.6	305	26.4
Type 3	412	10.3	183	12.5	104	9.0
Type 4	1493	37.3	537	36.7	459	39.7

	Adults							
	Total		19 to 30 yr.		31 to 40 yr.		41 to 50 yr.	
	n	%	*n*	%	*n*	%	*n*	%
Sample size	1379		784		331		264	
1 or more signs	1301	94.3	729	93.0	317	95.8	254	96.2
2 or more signs	895	64.9	503	64.2	218	66.0	174	65.9
Positional Dysfunction	1189	86.2	670	85.5	288	87.0	231	87.5
H. Nystagmus	723	52.4	379	48.3	189	57.1	155	58.9
V. Nystagmus	1063	77.0	598	76.3	255	77.0	210	79.4
Caloric Dysfunction	149	10.8	80	10.2	37	11.2	32	12.1
Direct. Preponderance	76	5.5	44	5.6	13	3.8	19	7.2
Reduced Vestib. Response	94	6.8	51	6.5	27	8.2	16	6.2
Simult. Cal. Dysfunction	961	69.6	535	68.2	235	71.0	191	72.2
Type 2*	341	24.7	199	25.4	85	25.6	57	21.5
Type 3	125	9.1	59	7.5	31	9.4	35	13.3
Type 4	497	36.0	278	35.5	119	36.0	100	37.8

*For definitions of Type 2, Type 3, and Type 4 caloric dysfunction, refer to p. 71.

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 ear or as a Type II response (Brookler, 1971) 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 are characterized by inconsistent vestibular responses to simultaneous binaural warm and cool water and were considered to be abnormal but of a nonlocalizing and nonspecific nature.

Subjects

The sample's ages ranged from 7 to 50 yr., with a mean age of 19 ± 10.5 yr. Of the sample, 1,465 or 36.6% were children (7 to 12 yr.) whose mean age was 9.8 ± 1.6 yr., 1,156 or 28.9% adolescents (13 to 18 yr.) whose mean age was 15.3 ± 1.6 yr., and 1,379 or 34.5% adults (19 to 50 yr.) whose mean age was 30.3 ± 9.1 yr. (Sarnoff, 1980; Sharp, 1980). Also, the adult group was divided by age into three subsamples to facilitate intergroup comparisons and intragroup analyses. The ages of these subsamples were 19 to 30 yr. with a mean age of 21.1 ± 3.1 yr., 31 to 40 yr. with a mean age of 34.5 ± 2.9 yr., and 41 to 50 yr., with a mean age of 43.9 ± 2.7 yr. The male/female ratio was 2.3/1 and the complete-righthanded/complete-lefthanded/mixedhanded ratios were 8.8/1.4/1 or 78.2%, 12.9%, 8.9%. Mixed-handedness was defined as present when an individual was able to perform one or more functions, e.g., eat, write, bat, throw, or catch, etc., as well or better with the nondominant hand. The remainder were completely righthanded and completely lefthanded individuals.

Results

Quantitative Analysis

Of the total sample of 4,000, 64.6% evidenced one or more fears/phobias. Table 3 highlights the most frequently reported 17 of about 45 fears/phobias characterizing this sample by age groups, namely, children, adolescents, and adults. Table 4 documents these fears/phobias as functions of sex and handedness. To test for a possible relation between fears/phobias and sex, handedness, as well as diagnostic neurological and ENG parameters, a statistical analysis was performed. On a two-tail t test, (1) females were significantly ($t = 6.03$, $p < .001$) more predisposed than males to fears/phobias; (2) all fears except for heights were statistically independent of handedness; mixed vs right ($t = 12.6$, $p < .001$) and lefthandedness ($t = 10.2$, $p < .001$) was significantly related to fears of heights; (3) mixed vs righthandedness ($t = 4.8$, $p < .01$) and also lefthandedness ($t = 4.4$, $p < .01$) showed significant relation to the ENG parameter of reduced vestibular response (RVR). See Table 5. All other parameters were independent of fears/phobias.

Using chi-squared, adults showed a significantly higher incidence of fears (71.8%) than children (63.9%) or adolescents (56.9%; $\chi^2 = 21.7$, $df = 2$, $p < .005$). Moreover, this higher incidence of fears among adults seemed related to the increased presence of fears characterizing agoraphobics, i.e., fears of heights ($\chi^2 = 35.9$, $df = 2$, $p < .005$) or crowds ($\chi^2 = 54.1$, $p < .005$), and feeling trapped or claustrophobic ($\chi^2 = 60.9$, $p < .005$). Also, fears of elevators were significantly higher in the 41 to 50 yr. subsample than in younger adults, adolescents, or children. Fears of planes and bridges, commonly associated with agoraphobia, seemed to be higher

TABLE 3

FEARS/PHOBIAS AS A FUNCTION OF AGE IN LEARNING DISABILITIES

Fears	Total		Children		Adolescents	
	N	%	7 to 12 yr.		13 to 18 yr.	
			n	%	*n*	%
Sample size	4000		1465		1156	
Any fear	2584	64.6	936	63.9	658	56.9
1. Heights	832	20.8	249	17.0	215	18.6
2. Darkness	606	15.2	328	22.4	173	15.0
3. Elevators	221	5.5	86	5.9	49	4.2
4. Crowds	182	4.6	33	2.3	40	3.5
5. Rides	177	4.4	88	6.0	51	4.4
6. Escalators	163	4.1	71	4.8	38	3.3
7. Claustrophobia	163	4.1	20	1.4	43	3.7
8. New situations	159	4.0	68	4.6	29	2.5
9. Planes	148	3.7	47	3.2	28	2.4
10. School	128	3.2	45	3.1	36	3.1
11. Getting lost	120	3.0	32	2.2	34	2.9
12. Being alone	114	2.9	64	4.4	26	2.2
13. Bridges	113	2.8	31	2.1	27	2.3
14. Social situations	110	2.8	16	1.1	17	1.5
15. Noise	86	2.2	34	2.3	29	2.5
16. Driving	81	2.0	5	0.3	8	0.7
17. Tests	40	1.0	9	0.6	8	0.7

	Adults							
	Total		19 to 30 yr.		31 to 40 yr.		41 to 50 yr.	
	n	%	*n*	%	*n*	%	*n*	%
Sample size	1379		784		331		264	
Any fear	990	71.8	533	68.0	266	80.3	191	72.3
1. Heights	368	26.7	179	22.8	113	34.1	76	28.8
2. Darkness	105	7.6	58	6.7	28	8.5	19	7.2
3. Elevators	86	6.2	41	5.2	23	6.9	22	8.3
4. Crowds	109	7.9	59	7.7	29	8.9	21	8.0
5. Rides	38	2.8	20	2.7	10	3.0	8	3.0
6. Escalators	55	4.0	35	4.5	8	2.3	12	4.5
7. Claustrophobia	100	7.3	48	6.4	30	9.1	22	8.3
8. New situations	62	4.5	34	4.3	20	6.0	8	3.0
9. Planes	73	5.3	30	4.2	24	7.3	19	7.2
10. School	47	3.4	27	3.6	14	4.2	6	2.3
11. Getting lost	53	3.9	26	3.3	18	5.6	9	3.4
12. Being alone	24	1.7	14	1.8	8	2.3	2	0.8
13. Bridges	55	4.0	18	2.4	18	5.6	19	7.2
14. Social situations	77	5.6	36	4.0	24	7.3	17	6.4
15. Noise	23	1.7	8	0.9	9	2.7	6	2.3
16. Driving	68	4.9	32	4.3	15	4.6	21	8.0
17. Tests	24	1.7	11	1.5	7	2.1	6	2.3

TABLE 4

FEAR/PHOBIA DISTRIBUTION AS A FUNCTION OF SEX AND HANDEDNESS IN LEARNING DISABILITIES

Fears	Total		Sex			
			Male		Female	
	N	%	*n*	%	*n*	%
Sample size	4000		2771		1229	
Any fear	2584	64.6	1692	61.1	892	72.6
1. Heights	832	20.8	552	19.9	280	22.8
2. Darkness	606	15.2	411	14.9	195	15.8
3. Elevators	221	5.5	114	4.1	107	8.7
4. Crowds	182	4.6	124	4.5	58	4.7
5. Rides	177	4.4	127	4.6	50	4.1
6. Escalators	163	4.1	74	2.7	89	7.2
7. Claustrophobia	163	4.1	88	3.2	75	6.1
8. New situations	159	4.0	101	3.6	58	4.7
9. Planes	148	3.7	89	3.2	59	4.8
10. School	128	3.2	81	2.9	47	3.8
11. Getting lost	120	3.0	67	2.4	53	4.3
12. Being alone	114	2.9	74	2.7	40	3.2
13. Bridges	113	2.8	70	2.5	43	3.5
14. Social situations	111	2.8	70	2.5	41	3.3
15. Noise	86	2.2	56	2.0	30	2.4
16. Driving	81	2.0	30	1.1	51	4.2
17. Tests	39	1.0	20	0.8	19	1.5
	Handedness					
	Right		Left		Mixed	
	n	%	*n*	%	*n*	%
Sample size	3129		516		355	
Any fear	2008	64.2	332	64.3	244	68.7
1. Heights	629	20.1	104	20.2	99	27.9
2. Darkness	473	15.1	79	15.3	54	15.2
3. Elevators	173	5.5	32	6.2	16	4.4
4. Crowds	140	4.5	26	5.1	16	4.4
5. Rides	142	4.5	24	4.7	12	3.4
6. Escalators	129	4.1	20	3.9	14	3.9
7. Claustrophobia	126	4.0	20	3.9	17	4.7
8. New situations	117	3.7	23	4.5	19	5.4
9. Planes	115	3.7	20	3.9	13	3.7
10. School	96	3.1	16	3.1	16	4.4
11. Getting lost	93	3.0	19	3.7	8	2.2
12. Being alone	93	3.0	17	3.3	4	1.3
13. Bridges	87	2.8	15	2.9	11	3.1
14. Social situations	89	2.8	14	2.7	8	2.2
15. Noise	68	2.2	9	1.8	9	2.5
16. Driving	64	2.0	8	1.6	9	2.5
17. Tests	30	1.0	6	1.2	3	0.9

TABLE 5
ENG PARAMETERS AS A FUNCTION OF SEX AND HANDEDNESS

Sex	Total		Handedness							
			Total		Right		Left		Mixed	
	N	%	*n*	%	*n*	%	*n*	%	*n*	%
			Male							
Sample size	4000		2771	69.3	2156	53.9	343	8.6	272	6.8
1 or more signs	3836	95.9	2664	96.1	2077	96.4	332	96.8	255	93.6
2 or more signs	2788	69.7	1929	69.6	1537	71.3	224	65.4	168	61.8
Positional Dysf.	3515	87.9	2435	87.9	1905	88.4	301	87.8	229	84.3
H. Nystagmus	1912	47.8	1293	46.7	995	46.1	161	46.8	138	50.8
V. Nystagmus	3241	81.0	2239	80.8	1752	81.3	282	82.1	206	75.6
Caloric Dysf.	387	9.7	258	9.3	209	9.7	24	6.9	26	9.7
D.P.*	208	5.2	133	4.8	111	5.1	11	3.2	11	4.0
R.V.R.*	235	5.9	155	5.6	118	5.4	16	4.6	22	8.3
Simult. Cal. Dysf.	3000	75.0	2085	75.3	1650	76.5	249	72.7	186	68.5
Type 2*	1095	27.4	734	26.5	598	27.7	78	22.6	59	21.7
Type 3	412	10.3	287	10.4	220	10.2	36	10.4	32	11.7
Type 4	1493	37.3	1063	38.4	831	38.6	137	39.9	95	35.0
			Handedness							
			Total		Right		Left		Mixed	
			n	%	*n*	%	*n*	%	*n*	%
			Female							
Sample size			1229	30.7	973	24.3	173	4.3	83	2.1
1 or more signs			1172	95.4	932	95.8	161	93.3	79	94.9
2 or more signs			859	69.9	693	71.2	104	59.9	62	74.3
Positional Dysf.			1080	87.8	861	88.5	143	82.5	76	91.5
H. Nystagmus			618	50.3	497	51.1	79	45.6	42	50.7
V. Nystagmus			1002	81.5	797	82.0	131	75.7	73	88.4
Caloric Dysf.			129	10.5	109	11.2	12	6.8	8	9.5
D.P.*			75	6.1	61	6.3	8	4.6	6	7.2
R.V.R.*			79	6.4	63	6.5	9	5.3	7	8.0
Simult. Cal. Dysf.			915	74.4	732	75.3	121	69.8	62	74.3
Type 2*			360	29.3	285	29.3	53	30.4	22	26.9
Type 3			125	10.1	101	10.3	15	8.4	9	11.2
Type 4			430	35.0	347	35.6	53	30.9	30	36.1

*D.P.—Directional preponderance. R.V.R.—Reduced vestibular response. For definitions of Type 2, Type 3, and Type 4 caloric dysfunction, refer to p. 71.

among adults than other age groups but their respective frequencies were too low for accurate statistical evaluation. Although fears of darkness were reported more often by children and adolescents than by adults, it appears likely that this difference probably reflected an inability of adults to recall having had a severe form of this fear when younger.

Qualitative Analysis

Inasmuch as the reported mechanisms characterizing the fears/phobias

for this sample yielded unexpected insights as the study progressed, an inadvertent but recognized bias was introduced as a function of the number of individuals questioned and fears analyzed. Only the qualitative data concerning these mechanisms are presented here although general impressions as to frequency are noted. The various factors reported to underlie similar named fears/phobias and related anxiety symptoms were the same, regardless of the subjects' ages. The most common fears characterizing the whole sample are discussed in descending order of their frequency. The percent of occurrence of each specific fear is indicated.

A qualitative analysis of the various mechanisms stated to be associated with, underlying, or responsible for the various fears/phobias of this sample showed (1) fears of heights (20.8%) were invariably related to fears of falling, losing one's balance, being pulled toward the visually sighted target below; only rarely did a fear of heights result from a fall. (2) Fears of darkness (15.2%) were frequently related to a variety of intensified fantasies at bedtime although a small percentage of these fears were triggered by the vertigo and disorientation resulting when the eyes were closed and/or the position of the head and neck changed prior to falling asleep. (3) Elevators (5.5%) commonly triggered fears of falling, being caught between the closing doors, and being trapped, as well as motion-related vertigo, imbalance, disorientation and anxiety—all intensified by the visual or sensory-deprivation experienced upon the doors closing and the loss of needed reference points. (4) Crowds (4.6%) appeared to represent visual and acoustic overloading or flooding and triggered vertigo, imbalance, disorientation, dyscoordination and anxiety, which resulted in fears of getting lost, falling, "jelly legs," losing control, being stuck or trapped. Occasionally crowds also provoked or intensified preexisting feelings of inadequacy and embarrassment. (5) Fears associated with amusement rides (4.4%) were often due to motion sickness and related anxiety as well as fears of heights and fears of being trapped and out of control. (6) Escalators (4.1%) often triggered fears of misstepping while getting on and/or off, getting one's foot caught, being trapped, as well as vertigo, imbalance and anxiety provoked when looking down from a height or at the grid-like pattern of moving steps. (7) Claustrophobic (4.1%) patients frequently did not identify the responsible triggering mechanisms although some reported vertigo, imbalance, disorientation and anxiety when in dark, tight, enclosed spaces, and all felt relief when they were able to see or get out. (8) New situations and places (4.0%) triggered disorientation and anxiety in those who easily lost their orientation and reference points. (9) Planes (3.7%) not infrequently triggered realistic fears. Many reported experiences of motion sickness and/or anxiety during acceleration, deceleration, circular, or up and down movement; others feared falling through the floor and crashing; and some only feared drowning while

others experienced fears of height or being trapped. (10) School phobias (3.2%) were most frequently triggered by anxieties related to academic failure, getting lost, and escalating frustration while trapped in classrooms. School-related fears were also provoked by the disorientation, vertigo, and/or imbalance secondary to such photophobic triggers as fluorescent lighting, flickering sun and shade on the way to and from school (occasionally relieved by tinted glasses), or motion sickness and anxiety provoked by the school bus, and even spontaneous panic attacks and the fear of recurrence. Rarely was this symptom triggered by separation anxiety resulting from a symbiotic relationship. Just about every school phobic child was able to play at significant distances from both home and parents. (11) Fears of getting lost (3%) were frequently related to impaired directional and orientation functioning which in young children often led to intensification of separation anxiety, rather than the reverse. (12) Being alone (2.9%) often triggered fears of getting sick, dizzy, disoriented, losing control, and occasionally being robbed or attacked with no one to help. Fears of parental injury or death were not infrequent among young children. (13) Bridges (2.8%) and/or the vertical supporting beams triggered fears of heights, falling or crashing, drowning, and being trapped. (14) Social situations (2.8%) and the resulting visual and/or noise overloading frequently impaired concentration and auditory sequencing while triggering disorientation, imbalance, vertigo, and anxiety, and often resulted in a destabilization of partially compensated or subtle communication and speech difficulties, and either triggered or intensified preexisting feelings of inferiority and being trapped. (15) Noise (2.2%) was experienced as either shrill, penetrating or overwhelming and occasionally resulted in vertigo, imbalance or tinnitus. (16) Driving (or being driven) (2%) triggered vertigo, imbalance, disorientation, inability to concentrate and coordinate, poor depth, directional and speed perception with resulting anxiety, and even claustrophobia when not in an exit lane, caught in traffic, or midway between home and destination. Obviously, the frequency of this fear was significantly higher in adults and adolescents of driving age than in children or younger adolescents. (17) Tests provoked fears in a majority of patients and panic (1.0%) in a few. Surprisingly, realistic or traumatic events were seldom reported responsible for the many fears/phobias of this sample. In contrast, CV-related mechanisms were most often stated as triggering or determining factors, regardless of age. Primary neurotic determinants appeared to play a minor role for many of the fears although they could not always be accurately detected and assessed by the methodology at hand.

Discussion

At first glance the 64.6% frequency of fears or phobias in this highly selected sample of learning disabled persons clearly suggests a CV-

determined phobic predisposition or somatic compliance when compared to the 7.7% and 20% prevalence rates reported by Agras, Sylvester, and Oliveau (1969) and by Langner and Michael (1963), respectively. However, a greater number of milder fears may have been obtained and counted in this survey. Accordingly, follow-up studies using an upgraded fear/phobia questionnaire should be undertaken to analyze corresponding data obtained from matched random and CV-normal controls who report fears/phobias. Comparative data from such control groups are considered essential for clarifying and validating the hypothesized cerebellar-vestibular basis of fears/phobias. In addition, these studies may highlight combinations and intensities of diagnostic parameters responsible for specific symptoms.

The unexpected statistical association found between mixed-handedness, fears of heights, and ENG-measured reduced vestibular response (RVR) suggests that asymmetric vestibular dysfunction or reduced vestibular response (RVR) may lead to imbalance-related fears of heights as well as compensatory shifts in cortical and/or lateral dominance, i.e., mixed-handedness. Perhaps studies of footedness will be found more related to imbalance-related fears. Moreover, the fact that females manifested more fears than males in most studies (Agras, *et al.*, 1969; Langner & Michael, 1963) raises the possibility that the CV-based mechanisms and triggers underlying the sex-related fears may be sensitized and destabilized by hormonal fluctuations in a manner analogous to the release of vertigo in menstrual-related states. Also, the higher incidence among the adults of fears characterizing agoraphobics (i.e., fears of heights, crowds, enclosed spaces or claustrophobia, driving or being driven, elevators, planes, bridges) appears consistent with the reported onset of agoraphobia in the very late teens and throughout adulthood (American Psychiatric Assn, 1987).

Although 65% of this CV-dysfunctional sample reported fears/phobias, 35% did not. This finding suggests that CV-dysfunction may be a predisposing rather than a determining factor or that only specific CV-based mechanisms lead to the development of fears. Moreover, fears/phobias were sometimes triggered by minimal rather than severe CV-impairment, indicating that the intensity of CV-dysfunction is often insufficient to predict a phobic response. In retrospect, two mechanisms were required for phobic development, (1) a mechanism responsible for determining the shape of the fears/phobias and (2) a mechanism triggering or releasing and sustaining a corresponding anxiety response. Based on clinical observations, these two mechanisms were assumed to be related but individually overdetermined. Obviously only a pathologically functioning anxiety mechanism is required for "pure" panic attacks and panic disorder.

The qualitative analysis of the many reported mechanisms found to characterize the fears/phobias of this sample led to identifying three distinct

primary phobic origins or types: Realistic or Traumatic fears/phobias—Type I, Neurotic fears/phobias—Type II, and CV-determined or based fears/phobias—Type III. These primary phobic types or mechanisms were capable of explaining most, if not all, of the fears characterizing this sample. Although nonCV neurochemical and neurophysiological CNS-determined or triggered fears/phobias (Type IV) exist, none were apparent in this sample. As described, any specific fear/phobia might be related to a number of different underlying mechanisms, and any given mechanism might be associated with several different phobias. In addition, combinations of fears/phobias per patient were observed to result from overdetermined combinations of determining mechanisms. If nothing else, these observations clearly highlighted the urgent diagnostic/therapeutic need to modify and replace the current descriptive classification of phobias and related anxiety disorders in DSM-III—R (American Psychiatric Assn, 1987) with one emphasizing mechanisms of action and functional determinism. Obviously treatment modalities must ultimately be geared towards reversing and compensating for the mixed and divergent groupings of overdetermined mechanisms rather than directed at the specified phobic outcome.

Upon carefully analyzing the mechanisms assumed to characterize the CV-based or Type III fears/phobias, a neurophysiological functional classification evolved. The array of phobic data could readily be explained by a relatively small number of known CV mechanisms modulating balance, coordination, muscle tone, motion, as well as visual, auditory, tactile, directional or compass, proprioceptive, body-image, and academic-related functions. (1) Fears of heights, steps, escalators, standing still and waiting, walking alone or unsupported in open spaces or through moving crowds, as well as the associated fears of getting dizzy, falling, fainting, losing control, and going crazy were characteristically triggered by CV-related *imbalance, perceptual,* and/or *tonal disturbances,* the latter resulting in "jelly legs." (2) Fears of driving, swimming, water, sports, even writing or speaking in distracting public situations were typically triggered by CV-determined *dyscoordination and unstable motor-related memory mechanisms.* (3) Fears of getting lost, going too far from home, new places and situations were often triggered by impaired *CV-orientation (compass) mechanisms.* (4) Fears of moving elevators, escalators, trains, planes, buses, cars, carnival rides, and even walking were triggered by impaired *CV-motion-processing mechanisms* similar to those triggering motion sickness. (6) Fears of enclosed or sensory-shielding environments such as rooms without windows, underwater, dark tunnels and subways, the dark, steel elevators, surrounding crowds, and having to remain stationary or motion-deprived were not infrequently triggered by dizziness, disorientation, imbalance, and/or "claustrophobic" anxiety secondary to *inadequate sensory reception* and/or *CV-decompensation.* (7) Fears of

failing tests, school, sounding stupid, looking ugly, feeling fat (with occasional anorexic-like responses), falling through "imaginary holes" in floors, planes, elevators, walking on tilting, shifting or cushion-like floors, as well as social or performance anxieties, most often reflected *CV-impaired academic, concentration, speech, body-image, proprioceptive*, and *coordination mechanisms*.

As suggested earlier, for CV-based or Type III fears/phobias to develop the above-described CV-related shaping mechanisms must trigger, release, and sustain a corresponding anxiety response. In prior observations, anxiety was clinically noted to be a frequent part of the motion-sickness syndrome. Accordingly, the CV system was hypothesized to trigger and modulate the anxiety response in a manner analogous to its modulation and triggering of the motion-sickness response. Just as a wide array of abnormal motion-sickness responses may be triggered by an impaired CV system, so also may a dysfunctioning CV system: (1) trigger overreactive anxiety signals (panic); (2) fail to inhibit properly and to modulate appropriate or exaggerated anxiety signals, perhaps resulting in chronic or intermittent anxiety states; (3) trigger a perseverated series of exaggerated signals (panic attacks); (4) fail to trigger normal and adaptive anxiety warning and learning signals which may perhaps explain this apparent deficiency in some "psychopaths."

In retrospect, these clinically derived neurophysiological speculations regarding the abnormal modulation of the anxiety response system were in harmony with the current biochemical views of panic attacks and panic disorder (Klein, 1981). Just as sodium lactate has been shown to trigger panic attacks in predisposed individuals (Pitts & McClure, 1967), so have a wide range of external and internal (chemical, physical, allergic, emotional or stress and fatigue-related, as well as optokinetic, rotation and caloric vestibular stimulation testing) stimuli been found clinically to destabilize CV-functioning (Levinson, 1986; Powers, 1976) and to result in phobic and/or panic symptoms (Levinson, 1986). Needless to say, abnormal anxiety responses may also be triggered by traumatic (Type I) and neurotic (Type II) determinants and even by nonCV-neurophysiological structures and chemical/metabolic changes (Type IV).

Inasmuch as the CV system is in a feedback relationship with the autonomic nervous system and *medulla oblongata* (Carr & Sheehan, 1984), the *nucleous coeruleus* (Redmond, 1977), and the anticipatory cerebral cortex, all secondary anxiety signs and symptoms such as sweating, palpitations, hyperventilization may readily be explained. Even the anticipatory triggering of anxiety in predisposed individuals is similar to the anticipatory triggering of motion sickness in those with an appropriate CV-based predisposition. As a result, therapies geared towards stabilizing the primary and/or secondary triggering systems may result in symptomatic benefit and relief.

Consideration of the determining mechanisms for each patient would suggest an appropriate therapy or combination of therapies. Traumatic or Type I phobias and mechanisms would best respond to behavior modification or desensitization, neurotic or Type II phobias and mechanisms to psychotherapy, CV or Type III (and nonCV or Type IV) phobias and mechanisms to pharmacotherapy, and mixed-types of phobias to specific combinations of therapies. As expected, apparent contradictions to this "simple" scheme were found and had to be explained. Why, for example, do many Type III phobias respond favorably to desensitization? Why are Type III phobias sometimes triggered by obvious emotional conflicts (death, divorce, illness, separation)? Why is psychotherapy beneficial?

To date, there has been no clear-cut explanation as to why anxiety-confrontation leads to reduction of anxiety for some and escalation for others. If, however, CV-dysfunction is responsible for Type III phobic anxiety, and as the CV-system can be conditioned to process more efficiently greater stimulus-confrontation via desensitization, then trigger-confrontation would result in a greater CV-processing capacity as well as a corresponding decrease in anticipatory anxiety, resulting in symptomatic relief. The techniques of desensitization are used to train astronauts to handle escalating motion stress without symptoms of motion sickness. Unfortunately, stimulus-confrontation cannot always be compensated for or conditioned and may result in CV-overloading, decompensation, and symptom intensification.

There is no doubt that emotional conflicts may trigger Type III phobias and that these phobias may be helped by psychotherapy. To explain these observations one must assume that emotional stress leads to CV-destabilization or decompensation and to Type III phobias in somatically predisposed individuals. The relief of this stress via appropriate psychotherapy results in CV-restabilization and phobic resolution. Obviously emotional conflicts do not lead to Type III phobias when the CV system is intact. Psychotherapy then is of little help in treating Type III phobias unless emotional stress and conflict are major triggers or coexisting determinants.

This developing concept of fears/phobias may permit previous explanations and methods of treatment to be integrated. Moreover, the concept may well readily explain most of the known clinical, experimental, and statistical data characterizing this fascinating group of anxiety-related symptoms. For example, one can more readily account for the heretofore perplexing relationships between (1) mitral valve prolapse (MVP), agoraphobia and panic episodes, (2) phobias and obsessions/compulsions, (3) phobias and depressive disturbances.

If indeed CV-dysfunction may result in a "floppy" hypotonic mitral valve and loose ligaments, a dysautonomia with tachycardia and extrasystoles, agoraphobia and panic episodes (which may in turn intensify a

preexisting dysautonomia and hypotonia), as well as learning disabilities and other symptomatic and even asymptomatic states, then it is reasonable to expect that combinations of these CV-determined symptomatic and asymptomatic states coexist or may be associated with one another (Crowe, Pauls, Kerber, & Noyes, 1981; Mazza, Martin, Spacavento, Jacobsen, & Gibbs, 1986; Weissman, Shear, Kramer-Fox, & Devereux, 1987), depending on the size and quality of the sample as well as interactions between related symptoms and determining mechanisms. In addition, the observation that females with mitral valve prolapse are more susceptible to panic episodes (Kane, Woerner, Zeldis, Kramer, & Saravay, 1981) is compatible with the findings that females are more vulnerable to both motion sickness (Lentz & Collins, 1977) and phobic or panic symptoms.

The clinically observed relationship between phobias and obsessive/compulsive neuroses became readily understandable when perseveration-like mechanisms known to characterize CV-dysfunction and learning disabilities were recognized as responsible for at least some of the repetitive ideas and acts characterizing this syndrome. Moreover, a failure in the CV system's capacity for background inhibition was reasoned to result not only in a short attention span, distractibility and daydreaming, or attention deficit disorder which characterize learning disabilities (Levinson, 1980, 1984) but also in obsessions/compulsions (Levinson, 1984, 1986). For example, this background disinhibition may allow intrusion of unconscious or background thoughts and fantasies into the foreground of consciousness and result in defensive rituals which persist via perseveration-like, disinhibitory mechanisms. In other words, both phobic and obsessive/compulsive symptoms may be derived from a common or overlapping cerebellar-vestibular source but with differing determining mechanisms.

The association of mood and anxiety disorders can also be readily explained. Patients with cerebellar-vestibular-based learning disabilities who are treated with antimotion sickness medications frequently report significant improvements in both stability and reactivity of mood and anxiety, which suggests an underlying relationship. Moreover, antidepressants or mood stabilizers were useful in treating various anxiety disorders, symptoms of learning disability (hyperactivity, short attention span, distractibility, and academic disabilities) as well as such CV symptoms as vertigo, headaches, and motion sickness. If indeed CV-determined mechanisms may lead to learning disabilities, anxiety disorders, mood disturbances as well as vertigo, headaches, and motion sickness and if antidepressants help stabilize CV-functioning, then the use and efficacy of these medications in the above disorders becomes as readily understandable as the association of the aforementioned group of diverse appearing symptoms.

Inasmuch as the fears/phobias and corresponding mechanisms experi-

enced and reported by the present sample appeared similar to those characterizing psychiatric patients referred for various anxiety disorders, it seemed reasonable to assume: (1) that the various DSM-III—R anxiety disorders, agoraphobia included, reflected highly selected, severe and combined versions of the milder fears/phobias presented in this analysis, (2) that all specific DSM-III—R diagnosed anxiety disorders would be characterized by a high incidence of CV-dysfunction as assessed by neurological, ENG and optokinetic examinations, (3) that all diagnosed primary DSM-III—R anxiety disorders contained associated anxiety symptoms sufficient to overlap with and diagnose most other DSM-III—R anxiety-disorder categories, (4) that the various DSM-III—R anxiety disorder categories are derived from a common group of Types I, II, and III (and/or Type IV) mechanisms which appear qualitatively similar to those shaping the mild fears observed here. These assumptions were supported by data to appear in this journal.

To date, the psychoanalytic, behavioral and biochemical explanations of fears/phobias and related symptoms have had only limited success. Perhaps this is because each explanation has captured but a part of the total clinical reality. Here a new holistic approach is proposed to explain most preexisting concepts and therapies of anxiety disorders while adding a crucial cerebellar-vestibular neurophysiological dimension and somatic compliance. This view is derived from an analysis of the mechanisms and triggers which characterized the fears/phobias of the 4000 learning disabled individuals who showed some CV-dysfunction. This approach appears consistent with the clinical facts characterizing the diagnosis and treatment of this large and varied sample of individuals showing anxiety disorder (Levinson, 1980, 1986). Independently performed and controlled studies using randomly chosen and CV-normal subjects with fears/phobias must be undertaken to confirm and explicate the details. Such studies would allow more precise formulation of the basic relationships, refinement of definitions, and derivation of testable hypotheses.

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