# Higher Prevalence of Obsessive-Compulsive Symptoms in Patients With Blepharospasm Than in Patients With Hemifacial Spasm

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Objective: The prevalences of obsessive-compulsive symptoms in patients suffering from blepharospasm and in those with hemifacial spasm were determined. The two conditions have similar symptoms, but only blepharospasm is etiologically linked to basal ganglia dysfunction. Method: After being interviewed with the Structured Clinical Interview for DSM-III-R, 13 patients with blepharospasm and 13 with hemifacial spasm completed the SCL-90-R and the Hamburg Obsession/Compulsion Inventory—Short Form. Results: Patients in the blepharospasm group had significantly more obsessive-compulsive symptoms, as indicated by higher scores on the Hamburg Obsession/Compulsion Inventory—Short Form, than the patients with hemifacial spasm. SCL-90-R scores were in the normal range for nine and eight categories, respectively (out of nine). Conclusions: The findings provide additional support for the hypothesis that obsessive-compulsive symptoms are related to basal ganglia dysfunction. (Am J Psychiatry 1998; 155:555–557)

B lepharospasm is characterized by involuntary contractions of the orbicularis oculi muscles with subsequent continuous or intermittent eyelid closures. The disorder is classified as a form of focal dystonia and has been linked to basal ganglia dysfunction (1–3). In comparison with healthy subjects, patients suffering from blepharospasm also show significantly more obsessive-compulsive symptoms (4). However, it is not clear whether these psychological disturbances are etiologically linked to the motor symptoms of blepharospasm or whether they are a result of the social and physical disability that this condition is known to produce.

This study is based on the premise that patients suffering from hemifacial spasm are an ideal comparison group for patients with blepharospasm. Hemifacial spasm is characterized by clonic-tonic contractions of the orbicularis oculi muscle and—as the disorder progresses—by involvement of other muscles of one-half of the face. Although the two conditions are similar in terms of symptoms and subjective impairment, hemifacial spasm results from peripheral facial nerve irritation and is not related to basal ganglia dysfunction (5).

Therefore, we hypothesized that blepharospasm patients would show significantly higher obsessive-compulsive scores than patients with hemifacial spasm.

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## **METHOD**

Twenty-nine sequential patients with a diagnosis of blepharospasm or hemifacial spasm from the dyskinesia outpatient clinic of the Department of Psychiatry at Göttingen University were asked if they would participate in the study. Three patients (two suffering from hemifacial spasm) refused by saying that they did not have enough time. Written informed consent was obtained from 26 patients after the procedure had been explained. All patients had been treated with botulinum A toxin at least once before inclusion in the study. None of the patients had received antidepressive or neuroleptic medication for at least 6 months before the study.

The patients were interviewed by means of the Structured Clinical Interview for DSM-III-R (SCID). Then the patients were asked to complete two rating scales: the SCL-90-R (6) and the Hamburg Obsession/Compulsion Inventory-Short Form (7). All assessments were done 30-120 minutes after treatment with botulinum A toxin. The SCL-90-R is a 90-item checklist that includes the following subscales: somatization, obsessionality, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoia, and psychoticism. The validity of the short form of the Hamburg Obsession/Compulsion Inventory has been well documented (7). It consists of 72 items that allow calculation of scores for six subcategories: checking, cleaning, arranging/hoarding, obsessional thoughts of words/pictures, counting/touching/speaking, and obsessional thoughts about doing harm to oneself or to others. A total score was calculated by addition of the scores for the six subcategories. The SCL-90-R was used to characterize the patient groups with regard to general psychopathology.

The SCL-90-R results from the two groups were compared with population-based reference values (8) by means of Student's t test for independent samples (two-tailed test). The t tests were adjusted for multiple tests by the Bonferroni-Holm method, which resulted in a corrected level of significance of p<0.0056. The scores of the two patient groups on the Hamburg Obsession/Compulsion Inventory—Short Form were compared by means of the Mann-Whitney U—Wilcoxon rank sum W test (one-tailed test) for nonparametric data.

TABLE 1. Psychopathological Symptom Scores of Patients With Blepharospasm and Hemifacial Spasm in Comparison With Population-Based Reference Values

	Score of Reference Group (N=1,006) (8)		Blepharospasm (N=13)				Hemifacial Spasm (N=13)			
				Comparison With Reference Group <sup>a</sup>					Comparison With Reference Group <sup>a</sup>	
			Score		+		Score		+	
SCL-90-R Category	Mean	SD	Mean	SD	(df=1017)	p	Mean	SD	(df=1017)	p
Somatization	0.35	0.30	0.51	0.33	1.91	0.05	0.65	0.51	3.54	0.001
Obsessionality	0.47	0.38	0.64	0.61	1.59	0.10	0.18	0.20	2.75	0.01
Interpersonal sensitivity	0.41	0.38	0.39	0.44	0.19	0.90	0.12	0.21	2.75	0.01
Depression	0.40	0.38	0.44	0.43	0.38	0.70	0.16	0.21	2.27	0.02
Anxiety	0.29	0.32	0.42	0.40	1.45	0.15	0.24	0.29	0.56	0.60
Hostility	0.31	0.34	0.13	0.20	1.90	0.05	0.14	0.21	1.80	0.05
Phobic anxiety	0.14	0.22	0.19	0.27	0.81	0.40	0.06	0.10	1.31	0.20
Paranoia	0.35	0.37	0.29	0.29	0.58	0.60	0.17	0.30	1.75	0.10
Psychoticism	0.18	0.24	0.17	0.20	0.15	0.90	0.10	0.23	1.19	0.25

<sup>&</sup>lt;sup>a</sup>Two-tailed t test. Bonferroni-corrected significance was set at p<0.0056.

#### RESULTS

The 13 patients with blepharospasm and 13 patients with hemifacial spasm did not differ in terms of age (blepharospasm: mean=67.3 years, SD=7.2; hemifacial spasm: mean=61.6 years, SD=11.2), sex ratio (blepharospasm: seven men, six women; hemifacial spasm: six men, seven women), or duration of the disease (blepharospasm: mean=8.2 years, SD=6.4; hemifacial spasm: mean=7.9 years, SD=4.9). According to the SCID, none of the patients fulfilled the diagnostic criteria for obsessive-compulsive disorder (OCD). Four patients in each group had a lifetime diagnosis of major depression but were not acutely depressed at the time of the interview. In the blepharospasm group, three patients suffered from simple phobias, one patient had a social phobia, one patient had a history of alcohol abuse, and one patient had a history of analgesics abuse. In the hemifacial spasm group, two patients fulfilled the diagnostic criteria for panic disorder with or without agoraphobia.

Table 1 shows that the patients in the blepharospasm and the hemifacial spasm groups did not differ significantly from population-based reference values for eight out of nine categories of psychopathology (SCL-90-R). Patients in the hemifacial spasm group showed significantly higher somatization scores than the reference group. The total scores on the Hamburg Obsession/Compulsion Inventory—Short Form were significantly higher for the blepharospasm group (median=18.0, range=5-34) than for the patients suffering from hemifacial spasm (median=12.0, range=2-32) (p=0.03). The compulsive behavior was mainly related to control, cleaning, and ordering.

# DISCUSSION

In the present study patients with blepharospasm had significantly more obsessive-compulsive symptoms than did patients suffering from hemifacial spasm.

These results are in agreement with those of former studies, in which patients suffering from blepharospasm or spasmodic torticollis had worse obsessivecompulsive and depressive symptoms than did healthy comparison subjects (4, 9). A limitation of these former studies was that the psychopathological findings could have been a reflection of social and physical disabilities associated with these conditions, which are not present in healthy subjects. However, both of our patient groups had SCL-90-R scores that were normal for eight out of nine categories. A similar result was obtained in another recent study of patients suffering from blepharospasm and hemifacial spasm (10); that study focused on illness-related psychosocial changes, which were significantly greater in the blepharospasm group, and no specific scales for the detection of obsessivecompulsive symptoms were used.

There is some phenomenological similarity between focal dystonias and OCD regarding the persistent, perseverative, repetitive, and involuntary nature of the symptoms. The occurrence of obsessive-compulsive symptoms as part of the postencephalitis parkinsonian syndrome (11) stimulated the search for a biological link between basal ganglia dysfunction and obsessive-compulsive symptoms. Compulsive behavior and obsessional thinking have been observed after basal ganglia lesions (12) and in patients with Gilles de la Tourette's syndrome (13). Accordingly, symptomatic dystonias can be associated with structural brain lesions (14), and blepharospasm has been observed after diencephalon infarction (1). Symptomatic pharmacological treatment of blepharospasm includes lisuride, L-dopa, and anticholinergics (with response rates between 20% and 28%) and neuroleptics (with a lower response rate, 8%) (15).

During the last decade a considerable number of clinical, positron emission tomography, and other brain imaging studies have produced evidence for basal ganglia involvement in the pathogenesis of OCD (16–19). It has been postulated that a disturbed striatal filter function causes a disinhibition of cerebral activities generated in fronto-orbital and cingulate structures (20–22).

In summary, patients with blepharospasm differ from patients with hemifacial spasm with respect to obsessive-compulsive symptoms. However, the obsessive-compulsive symptoms of the patients in this study were not pronounced enough for a diagnosis of OCD. Our results support the hypothesis that obsessive-compulsive symptoms are related to basal ganglia dysfunction. Since the patient groups were relatively small, these findings still have to be interpreted with care. Future research should clarify whether pharmacological treatments that have proven effective in OCD are also of benefit to patients suffering from basal ganglia disorders such as idiopathic spasmodic torticollis or blepharospasm.

### REFERENCES

- Powers JM: Blepharospasm due to unilateral diencephalon infarction (letter). Neurology 1985; 35:283–284
- Jankovic J: Blepharospasm with basal ganglia lesions (letter). Arch Neurol 1986; 43:866–868
- Marsden CD: Is tardive dyskinesia a unique disorder? Psychopharmacol Suppl 1985; 2:64-71
- Bihari K, Pigott TA, Hill JL, Murphy DL: Blepharospasm and obsessive-compulsive disorder. J Nerv Ment Dis 1992; 180:130– 132
- Nielsen VK: Pathophysiology of hemifacial spasm, I: ephaptic transmission and ectopic excitation. Neurology 1984; 34:418– 426
- Derogatis LR: SCL-90-R: Administration, Scoring, and Procedures Manual, I, for the Revised Version. Baltimore, Johns Hopkins University, Clinical Psychometrics Research Unit, 1977
- Klepsch R, Zaworka W, Hand I, Lünenschloss K, Jauernig G: Derivation and validation of the Hamburg Obsession/Compulsion Inventory—Short Form (HOCI-S): first results. Psychol Assess 1991; 1:196–201
- Franke G: The Symptom-Checklist SCL-90-R of Derogatis— German version. Göttingen, Germany, Beltz Test, 1995
- 9. Bihari K, Hill JL, Murphy DL: Obsessive-compulsive character-

- istics in patients with idiopathic spasmodic torticollis. Psychiatry Res 1992; 42:267-272
- Scheidt CE, Schuller B, Rayki O, Kommerell G, Deuschl G: Relative absence of psychopathology in benign essential ble-pharospasm and hemifacial spasm. Neurology 1996; 47:43–45
- Schilder P: The organic background of obsessions and compulsions. Am J Psychiatry 1938; 94:1397–1416
- Swedo SE, Rapoport JL, Cheslow DL, Leonard HL, Ayoub EM, Hosier DM, Wald ER: High prevalence of obsessive-compulsive symptoms in patients with Sydenham's chorea. Am J Psychiatry 1989; 146:246–249
- Pauls DL, Leckman JF, Towbin KE, Zahner GE, Cohen DJ: A possible genetic relationship exists between Tourette's syndrome and obsessive-compulsive disorder. Psychopharmacol Bull 1986; 22:730–733
- Kostic VS, Stojanovic-Svetel M, Kacar A: Symptomatic dystonias associated with structural brain lesions. Can J Neurol Sci 1996; 23:53–56
- Grandas F, Elston J, Quinn N, Marsden CD: Blepharospasm: a review of 264 patients. J Neurol Neurosurg Psychiatry 1988; 51: 767–772
- Benkelfat C, Nordahl TE, Semple WE, King AC, Murphy DL, Cohen RM: Local cerebral glucose metabolic rates in obsessivecompulsive disorder: patients treated with clomipramine. Arch Gen Psychiatry 1990; 47:840–848
- Baxter LR Jr, Phelps ME, Mazziotta JC, Guze BH, Schwartz JM, Selin CE: Local cerebral glucose metabolic rates in obsessivecompulsive disorder: a comparison with rates in unipolar depression and in normal controls. Arch Gen Psychiatry 1987; 44:211– 218
- Baxter LR Jr: Neuroimaging studies of obsessive compulsive disorder. Psychiatr Clin North Am 1992; 15:871–884
- Luxenberg JS, Swedo SE, Flament MF, Friedland RP, Rapoport J, Rapoport SI: Neuroanatomical abnormalities in obsessivecompulsive disorder detected with quantitative X-ray computed tomography. Am J Psychiatry 1988; 145:1089–1093
- Rapoport JL: Recent advances in obsessive-compulsive disorder. Neuropsychopharmacology 1991; 5:1–10
- Insel TR: Neurobiology of obsessive compulsive disorder: a review. Int Clin Psychopharmacol 1992; 7(suppl 1):31–33
- Insel TR: Toward a neuroanatomy of obsessive-compulsive disorder. Arch Gen Psychiatry 1992; 49:739–744