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Hyperhidrosis in social anxiety disorder

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Abstract

Purpose: Excessive sweating (hyperhidrosis) is an overlooked and potentially disabling symptom, which is often seen in social anxiety disorder (SAD). We conducted a retrospective review of data acquired in patients with SAD who had participated in placebo-controlled clinical trials of fluoxetine, cognitive behavior therapy, clonazepam and gabapentin. Four specific topics were addressed: (1) overall levels of sweating; (2) characteristics of those with hyperhidrosis; (3) a comparison of active treatments relative to placebo on hyperhidrosis; and (4) an examination of baseline sweating severity as a predictor of treatment outcome. *Methods*: Using the Brief Social Phobia Scale (BSPS) and Social Phobia Inventory (SPIN), we examined the above questions. *Results*: Hyperhidrosis was found in 24.8–32.3% of 375 subjects assessed, depending upon the scale used. Hyperhidrosis was associated with higher levels of disability, fear, avoidance, and other physiologic symptoms. While treatment in general was associated with a reduction in the rate of hyperhidrosis from 23.7% to 9.7% (BSPS), and 34.0% to 15.5% (SPIN), only fluoxetine differed significantly from placebo in respect of change in sweating score from baseline to endpoint. In an ANCOVA, gabapentin differed from placebo on the SPIN. *Conclusion*: We conclude that hyperhidrosis is frequently seen in patients with SAD, and that its response to treatment is variable. Further attention should be paid to the possible importance of this symptom in social anxiety.

Keywords: Excessive sweating; Hyperhidrosis; Social anxiety; Treatment

1. Introduction

Heightened physiological arousal is often seen in social anxiety disorder (SAD), and may be the principal cause for a person's desire to seek treatment (van Vliet et al., 1994). Of these symptoms, blushing, trembling, and sweating are particularly distinctive. Excessive sweating, or hyperhidrosis, is well recognized in dermatologic and neurological settings (Malone et al., 1986; Bell et al., 2000; Heckmann et al., 1999) but is quite neglected in psychiatry, despite being a prominent feature of SAD (Norton et al., 2001). It can be difficult to treat, although one report suggests possible benefit of clonidine (Goldstein, 1987). While the physiological symptoms in SAD as a whole are responsive to treatment, e.g. to SSRI and gabapentin (Stein et al., 1999; van Ameringen et al., 2001; Pande et al., 1999), the individual symptoms have not been specifically addressed.

Hyperhidrosis can be associated with significantly impaired quality of life, even interfering with the ability to carry out professional responsibilities (Amir et al., 2000). Moran and Brady (1991), for example, reported that it can have "devastating consequences for work and social activities." Indeed, hyperhidrosis can be so distressing that sufferers go to considerable lengths in order to receive surgical treatment, often traveling internationally to undergo endoscopic transthoracic cervical sympathectomy (ETS). Telaranta (1998) has reported that ETS can be effective for hyperhidrosis in SAD, and that the procedure is associated with a generalized improvement in other physiologic symptoms (e.g. blushing, tremor), as well as in anxiety as a whole, at 4-month follow up.

Given the neglect of hyperhidrosis in SAD, and its potential importance, the authors decided to review their accumulated data over 10 years of clinical trials in SAD, wherein we measured: (1) the severity of reported sweating along with other physiological symptoms (blushing, tremor,

Abbreviations: BSPS, Brief Social Phobia Scale; SAD, Social Anxiety Disorder; SPIN, Social Phobia Inventory; ETS, Endoscopic Thoracic Sympathectomy; SDS, Sheehan Disability Scale; LSAS, Liebowitz Social Anxiety Scale; SVS, Sheehan Stress Vulnerability; IRB, Institutional Review Board.

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and palpitations) by means of an interview-rated scale, the Brief Social Phobia Scale (BSPS) (Davidson et al., 1997), and (ii) subjective distress from sweating by the more recently developed self-rated Social Phobia Inventory (SPIN) (Connor et al., 2000). The objectives of this paper were the following: (a) to report on severity and distress of sweating; (b) to describe the features of individuals with hyperhidrosis; and (c) to describe the response of hyperhidrosis to a variety of treatments, all of which were compared against placebo.

2. Methods

Subjects met DSM-IIIR (American Psychiatric Association, 1987) or DSM-IV (American Psychiatric Association, 1994) criteria for SAD, which in almost all cases was of the generalized subtype. The sample comprised 375 subjects (216 men and 159 women), who were ethnically grouped into 322 Caucasian and 49 non-Caucasian, with four unaccounted. Mean (S.D.) age was 37.9 (9.5) years. Data in up to 375 subjects were available with the BSPS, depending upon which particular comparisons were performed, whereas the maximum number of available SPIN comparisons was 272. All studies had received Institutional Review Board (IRB) approval, and subjects provided written informed consent.

On the BSPS subjects are asked: "When you are in a situation involving contact with other people, or when you are thinking about such a situation, do you experience sweating?" The symptom is rated over the last week on a scale from 0 (*none*) to 4 (*extreme*). For the purposes of this report, we grouped those with scores of 0 or 1 (*none* or *mild*) and compared them to individuals with scores of 3 (*severe*) or 4 (*extreme*). On the self-rated SPIN, subjects respond to the question "Sweating in front of people causes me distress," and similarly rated on a scale of 0–4 using the same anchors.

In this sample, we report on treatment effects for fluoxetine vs. placebo ($n=32$, $n=38$) and cognitive behavioral therapy (CBT) vs. placebo ($n=38$ each). Both of these comparisons are drawn from a nearly completed study of SAD in which fluoxetine and CBT were included as treatment arms vs. placebo (Davidson and Foa, unpublished data). A second trial compared clonazepam vs. placebo ($n=23$ each) (Davidson et al., 1993) and the third study compared gabapentin vs. placebo ($n=33$, $n=29$) (Pande et al., 1999). Both the BSPS and the SPIN were used in all studies except the comparison of clonazepam and placebo. Data at baseline were included from a fourth open-label study with clonazepam, which was followed by double-blind discontinuation (Connor et al., 1998). While this study does not inform us as to the comparative effects of treatment vs. placebo on sweating, it provides baseline information on characteristics and intensity of sweating ($n=51$).

Rates of sweating severity were assessed at baseline. As noted above, hyperhidrosis (or high sweating) was defined as a score of 3 or 4 on each scale. Comparisons of high vs. low sweating groups were made at baseline with respect to gender, age, age at onset, and symptom severity. Chi-square, Fisher's, and *t* tests were used as appropriate. For non-normally distributed data, Wilcoxon and Kruskal–Wallis tests were used. Median and 25th and 75th percentile (or first and third quartile) scores are given.

Comparisons of each treatment against placebo were undertaken at baseline, endpoint and for the baseline–endpoint difference. Nonparametric Wilcoxon and Kruskal–Wallis tests were used. In some trials, other measures were utilized, specifically the Liebowitz Social Anxiety Scale (LSAS) (Liebowitz, 1996), the Sheehan Disability Scale (SDS) (Sheehan et al., 1996), and the Sheehan Vulnerability Stress Scale (SVS) (Sheehan et al., 1990). These were used to characterize the high and low sweating groups. For assessing treatment vs. placebo differences, the Wilcoxon and Kruskal–Wallis tests were supplemented by ANCOVA, which was conducted to adjust for baseline differences. In some instances, data were missing on the SPIN, such that sample sizes were smaller than for the BSPS, and the placebo-controlled study of clonazepam was conducted before the SPIN had been developed.

3. Results

3.1. Severity of sweating and rates of hyperhidrosis

3.1.1. Brief Social Phobia Scale

The mean (S.D.) and median (1st, 3rd Q) baseline sweating scores ($n=375$) were 1.6 (1.2) and 2 (1, 2). 24.8% ($n=93$) reported hyperhidrosis, while 30.9% ($n=116$) reported moderate sweating and 44.3% ($n=166$) reported either no or mild sweating.

3.1.2. Social Phobia Inventory

Mean (S.D.) and median scores ($n=272$), were 1.8 (1.3) and 2 (1, 3), respectively. 32.3% ($n=88$) reported hyperhidrosis, 22.8% ($n=62$) reported moderate sweating, and 44.8% ($n=122$) reported no or minimal sweating.

These results indicate that on average, subjects entering clinical trials exhibited mild to moderate levels of sweating. A breakdown according to individual scores is shown in Table 1.

Table 1
Rates of hyperhidrosis (%)[a] according to severity and measurement

Item score	BSPS ($n=375$)	SPIN ($n=272$)
0 (*none*)	23.5	21.3
1 (*mild*)	20.8	23.5
2 (*moderate*)	30.9	22.8
3 (*severe*)	18.4	22.4
4 (*extreme*)	6.4	9.9

[a] Hyperhidrosis defined as a sweating score of 3 or 4.

Table 2
Baseline comparison of subjects with high and low levels of sweating according to the BSPS [Mean (S.D.)]

	BSPS		
	High[a] ($n=93$)	Low[b] ($n=166$)	
Age (years)	36.5 (9.9)	37.5 (9.2)	NS
% Male	60.2	54.8	NS
Age at onset (years) ($n=218$)	21.2 (27.0)	21.2 (27.0)	NS
BSPS[c]—Fear	18.8 (4.0)	16.2 (4.3)	$F=23.28$, $df=1,257$, $P<.0001$
BSPS—Avoidance	18.1 (4.8)	15.6 (5.0)	$F=15.09$, $df=1,257$, $P=.0001$
BSPS—Other physiological symptoms	6.9 (2.7)	4.3 (2.4)	$F=62.89$, $df=1,257$, $P<.0001$
LSAS[d] ($n=237$)	84.2 (23.0)	78.3 (22.4)	$F=3.75$, $df=1,236$, $P=.005$
SDS[e] ($n=188$)	17.7 (6.5)	15.6 (5.7)	$F=5.73$, $df=1,186$, $P=.01$
SVS[f] (n=63)	5.7 (2.6)	4.6 (2.5)	$F=2.93$, $df=1,63$, $P=.09$

[a] Score of 3 or 4.
[b] Score of 0 or 1.
[c] BSPS = Brief Social Phobia Scale (Davidson et al., 1997).
[d] LSAS = Liebowitz Social Anxiety Scale (Liebowitz, 1996).
[e] SDS = Sheehan Disability Scale (Sheehan et al., 1996).
[f] SVS = Sheehan Stress Vulnerability Scale (Sheehan et al., 1990).

3.2. Differences between groups at baseline

3.2.1. Brief Social Phobia Scale

When comparing the characteristics of those with hyperhidrosis ($n=93$) to those with minimal sweating ($n=166$) (Table 2), those with hyperhidrosis ($n=93$) had higher ratings on all social anxiety measures and on the SDS; the groups did not differ on demographic measures or on stress vulnerability. Further, the hyperhidrosis group was significantly more distressed by other physiological symptoms (blushing, tremor, and palpitations) [mean (S.D.): 6.94 (2.72) vs. 4.34 (2.42); $P<.0001$].

3.2.2. Social Phobia Inventory

Those with hyperhidrosis ($n=88$) were distinguishable from those with minimal sweating ($n=122$) by having higher rates of disability on the SDS and higher rates of social anxiety on the LSAS and on all BSPS subscales (all $P<.05$) (Table 3). Significantly higher levels of general physiological distress were also associated with hyperhidrosis [6.28 (2.71)] vs. minimal sweating subjects [4.32 (2.69); $P<.0001$]. No differences were noted for age, gender, or age at onset of social phobia.

Self-rated distress from sweating correlated significantly with observer-based rating of reported sweating at baseline ($r=.54$, $P<.0001$, $n=272$) and at endpoint ($r=.57$, $P<.0001$, $n=192$).

3.3. Effects of treatment

3.3.1. Brief Social Phobia Scale

Posttreatment data were available for 338 subjects. While 23.7% ($n=80$) of the sample were noted to qualify for hyperhidrosis at pretreatment, only 9.7% ($n=35$) reported hyperhidrosis posttreatment (Table 4). Actual mean (S.D.) pre- and posttreatment scores ($n=338$) for the entire group were 1.61 (1.20) and 0.89 (1.06), respectively. For all active treatments ($n=250$) vs. placebo ($n=88$), pretreatment scores were 1.62 (0.78) and 1.60 (1.19), respectively, while posttreatment scores were 0.78 (1.01) and 1.19 (1.16), respectively, with significantly greater improvement observed with active treatment ($P=0.001$). The effect of individual treatments on sweating was generally disappointing, however, with a significant difference noted only in favor of fluoxetine over placebo ($P=.008$) (Table 5). This effect was upheld after analysis of covariance, given the

Table 3
Baseline comparison of subjects with high and low levels of sweating according to the SPIN [Mean (S.D.)]

	SPIN		
	High ($n=88$)	Low ($n=122$)	
Age (years)	36.3 (9.7)	38.2 (9.9)	NS
% Male	55.9	52.5	NS
Age at onset (years)	19.6 (25.2)	23.4 (29.1)	NS
BSPS—Fear	18.3 (3.6)	16.1 (4.1)	$F=16.28$, $df=1,209$, $P<.0001$
BSPS—Avoidance	17.1 (4.8)	15.4 (5.0)	$F=6.44$, $df=1,209$, $P=.01$
BSPS—Physiological	6.3 (2.7)	4.3 (2.7)	$F=26.86$, $df=1,209$, $P<.0001$
LSAS ($n=197$)	86.3 (22.7)	77.0 (20.0)	$F=9.42$, $df=1,196$, $P=.002$
SDS ($n=140$)	18.0 (6.7)	15.8 (5.0)	$F=4.82$, $df=1,139$, $P=.02$

Table 4
Rates of sweating before and after treatment (%)

Severity of sweating	BSPS ($n=338$)		SPIN ($n=200$)	
	Pre	Post	Pre	Post
0 (*none*)	23.7	51.2	20.0	37.5
1 (*mild*)	21.3	19.2	24.5	29.5
2 (*moderate*)	31.4	19.8	21.5	17.5
3 (*severe*)	16.9	8.9	22.5	11.5
4 (*extreme*)	6.8	0.9	11.5	4.0

different baseline levels of hyperhidrosis in the two treatment groups [$F(1,67)=8.87$, $P=0.004$ for baseline; $F(1,67)=4.27$, P=.04 for treatment].

3.3.2. Social Phobia Inventory

Posttreatment data were available for 200 subjects. Rates of hyperhidrosis were 34.0% ($n=68$) and 15.5% ($n=31$), at pretreatment and posttreatment, respectively (Table 4). Mean pre- and posttreatment scores for $n=200$, were 1.81 (1.30) and 1.15 (1.16), respectively. For all active treatments ($n=144$) and placebo ($n=56$), pretreatment scores were 1.74 (1.30) and 1.98 (1.30), while posttreatment scores were 1.04 (1.07) and 1.42 (1.34). No difference was found between active treatment and placebo ($P=0.32$). In assessing the effects of individual treatments on sweating, a trend was noted ($P=0.07$) in favor of gabapentin over placebo.

Table 5
Effects of treatment on sweating (BSPS)

		Pre	Post	χ^{2a} (P)
Fluoxetine ($n=32$)	Median (1st, 3rd Q)	2 (1, 3)	0 (0, 2)	11.15 (.0008)
	Mean (S.D.)	1.94 (1.08)	0.72 (1.02)	
Placebo ($n=38$)	Median (1st, 3rd Q)	1 (0, 2)	1 (0, 2)	
	Mean (S.D.)	1.21 (1.04)	1.00 (1.12)	
CBT ($n=38$)	Median (1st, 3rd Q)	2 (0, 2)	1 (0, 2)	1.64 (NS)
	Mean (S.D.)	1.55 (1.20)	1.03 (1.10)	
Placebo ($n=38$)	Median (1st, 3rd Q)	1 (0, 2)	1 (0, 2)	
	Mean (S.D.)	1.21 (1.04)	1.00 (1.12)	
Clonazepam ($n=23$)	Median (1st, 3rd Q)	2 (1, 3)	0 (0, 1)	1.77 (NS)
	Mean (S.D.)	1.87 (1.36)	0.52 (0.90)	
Placebo ($n=21$)	Median (1st, 3rd Q)	2 (1, 3)	1 (0, 2)	
	Mean (S.D.)	1.90 (1.48)	1.05 (1.12)	
Gabapentin ($n=29$)	Median (1st, 3rd Q)	2 (1, 2)	1 (0, 2)	0.82 (NS)
	Mean (S.D.)	1.75 (1.09)	1.14 (1.03)	
Placebo ($n=29$)	Median (1st, 3rd Q)	2 (1, 3)	2 (0, 2)	
	Mean (S.D.)	1.89 (1.37)	1.55 (1.21)	

[a] Kruskal–Wallis test comparing active treatment vs placebo on the median differences in score between pre- and posttreatment.

Table 6
Effects of treatment on sweating (SPIN)

		Pre	Post	χ^{2a} (P)
Fluoxetine ($n=23$)	Median (1st, 3rd Q)	2 (1, 3)	1 (0, 2)	0.45 (NS)
	Mean (S.D.)	2.04 (1.22)	0.96 (0.98)	
Placebo ($n=23$)	Median (1st, 3rd Q)	2 (1, 3)	1 (0, 2)	
	Mean (S.D.)	2.00 (1.20)	1.13 (1.36)	
CBT ($n=23$)	Median (1st, 3rd Q)	2 (1, 3)	1 (0, 2)	0.89 (NS)
	Mean (S.D.)	1.83 (1.37)	1.37 (1.27)	
Placebo ($n=30$)	Median (1st, 3rd Q)	2 (1, 3)	1 (0, 2)	
	Mean (S.D.)	2.00 (1.20)	1.13 (1.36)	
Gabapentin ($n=33$)	Median (1st, 3rd Q)	2 (1, 3)	1 (0, 2)	3.10 (NS)
	Mean (S.D.)	1.88 (1.22)	1.12 (1.08)	
Placebo ($n=33$)	Median (1st, 3rd Q)	2 (1, 3)	1 (1, 3)	
	Mean (S.D.)	1.97 (1.38)	1.64 (1.31)	

[a] Kruskal–Wallis test comparing active treatment vs. placebo on the median differences in score between pre- and posttreatment.

Using ANCOVA, a significant treatment effect for gabapentin emerged [$F(1,63)=4.52$, P=.03; $F(1,63)=59.70$ $P<.0001$ for baseline]. No other comparisons yielded evidence of a significant advantage for active treatment over a placebo (Table 6).

4. Discussion

Hyperhidrosis occurring in 25–33% of the sample according to measure. Although not markedly different, the self-rating scale yielded a slightly higher prevalence rate of hyperhidrosis. Differences in wording may be a factor, in that the BSPS rating is based on frequency and/or distress, whereas the SPIN relies entirely on distress. Hyperhidrosis can be the focus of substantial distress, impaired work performance. Given that, and impaired social functioning, we argue that it may justify more attention than it has so far received. Many patients, especially those who are professionally required to make presentations in the course of their daily work, find that excessive sweating may be profoundly disturbing. It is not uncommon for extensive anticipatory fear to build up around this problem. Anecdotal reports exist of hyperhidrosis rendering a person unable to perform their job, e.g. a policeman or active military person who is unable to hold on to his weapon, a secretary whose profuse sweating drenches the keyboard, or the individual who has to change his or her clothes more than three times daily. We hope that our results will draw further attention to this important aspect of SAD.

Fluoxetine was associated with statistically significant improvement in sweating with both analyses on the BSPS, and gabapentin showed superiority on the SPIN

by ANCOVA only. All active treatments together were superior to placebo, but many patients are left with residual sweating at the end of treatment, and that while they may be somewhat less distressed about having the symptom, further improvement would be desirable. Other treatment approaches thus deserve to be explored. We note as a limitation to our findings, however, that the studies were not powered to address only a single item. An additional limitation lies in the recognition that our sample of clinical trial participants may not be reflective of the larger population with social anxiety and hyperhidrosis.

Control of sweating is largely mediated through sympathetic cholinergic pathways, with adrenergic mechanisms exerting lesser influence, and being somewhat more associated with sweat odor than with volume. Some patients have tried anticholinergic treatments such as propantheline, or even topical treatments such as Drysol, with limited benefit. One potentially interesting application might be the use of microdose botulinum toxin-A by local injection. This form of treatment has proven to be highly effective in idiopathic axillary hyperhidrosis (Naumann and Lowe, 2000). Such an approach might also be an adjunctive strategy to manage hyperhidrosis with SAD. Given the benefit of fluoxetine, it may be asked whether perhaps serotonergic mechanisms may have a role in hyperhidrosis. An important and related question might be the extent to which a treatment, which abolishes a profoundly distressing peripheral symptom of social anxiety, might also have positive feedback on central (cognitive/appraisal) aspects of the disorder. The report by Telaranta (1998) suggests that this might be the case to some extent.

5. Conclusions

Hyperhidrosis was observed in over a quarter of the study sample, and was associated with more severe social anxiety symptoms and disability. It was also associated with other physiological symptoms of social anxiety. Fluoxetine was superior to placebo on the BSPS, and similarly for gabapentin on the SPIN, but other treatments known to be effective in SAD (e.g. cognitive behavior treatment, clonazepam) were of only modest benefit in treating hyperhidrosis.

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