enced facial paresthesias of a mild, electric-shock-like nature that were also associated with the subjective sense of facial twitching. Each episode lasted approximately 5 minutes. No involuntary facial movements were ever verified by direct observation in a mirror or by observations of others. She noticed a heightened intensity of the paresthesias in the circumoral area if she smoked a cigarette after taking her medication. She could recall no associated hyperventilation, shortness of breath, coughing spells, or preceding heightened anxiety. After the third day, these symptoms never recurred despite continuation of the medication.

***Case 2.*** Ms. B, a 29-year-old Eurasian woman diagnosed by DSM-IV criteria with dysthymia and borderline personality traits, was placed on paroxetine 20 mg/day. On the second day of treatment, shortly after taking this medication, she experienced several paroxysms of electric-shock-like paresthesias isolated to the left side of her face and head. These came on repeatedly over a 3-minute period, with each "burst" lasting 5–10 seconds. Because these symptoms were so bothersome to the patient, she discontinued the medication permanently. No muscular symptoms, exacerbating or relieving factors, precipitating movements or actions, or other associated features were recalled. However, the singularity and brevity of these adverse effects may have limited such identification. The patient was subsequently placed on sertraline treatment without incident.

***Case 3.*** Ms. C, a 22-year-old Native American woman diagnosed with major depression, was placed on paroxetine 20 mg/day 2 weeks after failing a trial of sertraline 50 mg/day secondary to gastrointestinal complaints. Within minutes of taking the medication, she experienced a paroxysm of electric-shock-like paresthesias isolated to her head. They seemed unrelated to any other activity and lasted for approximately 15 seconds, remitting without residua. These symptoms recurred daily after each morning dose for the first 5 days of pharmacotherapy. Ms. C, however, was able to continue on paroxetine treatment and suffered no adverse effects thereafter.

Paroxetine has been noted to have several adverse effects related to the nervous system including somnolence, dizziness, insomnia, tremor, nervousness, anxiety, paresthesias, agitation, myoclonus, and confusion.[1] Previously, Frost and Lal[2] described three patients who experienced electric-shock-like sensations upon discontinuation of paroxetine (in two) or sertraline (in one). This was the first time paroxysmal paresthesias of an electrical character had been described associated with paroxetine. In each case, they lasted "a few seconds" or less and "traveled" or radiated from one region to another, often to the limbs. Interestingly, this description is very similar to Lhermitte's sign, "sudden 'electrical' pains occurring with neck flexion down the spine and into the upper extremities."[3] Of note, one of their patients could avoid these sensations by "keeping his head motionless." Although commonly associated with multiple sclerosis, Lhermitte's sign has been associated with over a dozen spinal cord disorders, but never with transient drug effects.[4]

By contrast, our patients represent the first cases described of paroxysmal, shock-like paresthesias associated with the initiation of paroxetine. Although each of the paroxysms of paresthesias was short-lived as in the previous cases, lasting 5 seconds to 5 minutes, all were isolated to the head or face and did not radiate or travel. All of Frost and Lal's patients had involvement of the trunk, two also had involvement of the extremities, but only one had involvement of the face or head. All

years and 27.3 years, respectively). All of our cases and two of the previous three cases had prominent anxiety features, and all patients in both groups described their paresthesias as electrical in character.

Each of our three patients reported their symptoms independently of each other over a 1.5-month period, and none knew one another. Our patients were taking no concurrent medications, their medications came from separate pharmaceutic batches, and they had no underlying medical or neurologic disorders that might have contributed to their presenting complaint. Two of the three patients had not previously received psychotropic medications. The third patient had been off sertraline therapy for 2 weeks and did not appear to be experiencing withdrawal symptoms. Although all three were nicotine dependent, only one patient reported more intensified and focused symptoms while smoking. Additionally, she was the only patient who also sensed subjective facial twitching, which may have represented fasciculations, although no objective observation occurred to confirm this.

In each of our patients, the reported paroxysmal, shock-like paresthesias had a clear proximal link to the ingestion of paroxetine, during the period of medication initiation. These cases are reported in order to alert physicians who prescribe paroxetine to the possibility of this adverse effect upon its initiation.

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army, Department of Defense, or the U.S. Government.

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## Benztropine in the Treatment of Venlafaxine-Induced Sweating

**Sir:** Sweating is a relatively common side effect seen with serotonin selective reuptake inhibitors (SSRIs) and has been reported in 7% to 11% of patients treated with SSRIs and 12% of patients treated with venlafaxine, an antidepressant with the ability to inhibit the reuptake of serotonin and norepinephrine.[1] The following case indicates that this side effect can cause severe social disruption and limit effective treatment of depression. A possible management strategy is also suggested.

***Case report.*** Ms. A, a 48-year-old school teacher with a 4-year history of depression, was diagnosed as having major depressive episode and dysthymia, but was refractory to trials of

patient noted partial improvement in her depression, but experienced "hot flashes" occurring several times a day and lasting 5

to 10 minutes. Symptoms included a feeling of heat all over her body and perspiration on the back of her neck. Follicle-stimu-

75 mg t.i.d. was accompanied by complete remission of depression, but such severe and socially embarrassing sweating that the patient felt compelled to discontinue the drug. Ms. A was encouraged to restart venlafaxine at 75 mg b.i.d. with benztropine added at 0.5 mg b.i.d. The "hot flashes" did not recur, and venlafaxine was then increased to 75 mg t.i.d. with subsequent remission of depression and no side effects.

The eccrine sweat glands are stimulated by the sympathetic nervous system; however, the postganglionic fibers that reach the muscarinic receptors on the end organ are cholinergic. The preoptic and anterior hypothalamic nuclei are the areas of the hypothalamus that contain heat sensitive and cold sensitive neurons and are mainly responsible for the stimulation of these sweat glands.[2]

The exact mechanism of antidepressant-induced sweating is unknown. It has been reported that the use of clonidine is successful in the treatment of tricyclic-induced sweating[3] and that propranolol increases sweating.[4] These findings suggest a role for norepinephrine in the mechanism of antidepressant-induced sweating. Presumably SSRIs enhance sweating either indirectly by affecting the sympathetic system or directly by acting on the hypothalamus.

The use of the anticholinergic benztropine was effective in preventing the excess sweating in our patient and allowed her to continue treatment with venlafaxine. Benztropine most likely blocked the acetylcholine receptors on the eccrine sweat glands and thereby caused a reduction in sweating. Anticholinergic

agement of excess sweating induced by SSRIs.

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## Stereotypic Movement Disorder

**Sir:** Castellanos et al.[1] (March 1996 issue) recently reported that DSM-IV stereotypic movement disorder can be diagnosed in intellectually normal adult patients. This paper is another in a series of fascinating studies conducted by Rapoport and colleagues on the differential response of unwanted repetitive behaviors such as hair pulling[2] and nail biting[3] to clomipramine and desipramine. The idea that such behaviors fall on an obsessive-compulsive disorder (OCD) spectrum of conditions may provide a valuable heuristic in both clinical and research settings.[4]

In their paper, Castellanos et al.[1] reported the response of three patients to both clomipramine and desipramine, two of whom used limited doses. In two of the three subjects, there seemed to be a better response to clomipramine than desipramine. In only one of the three patients, however, was there a

lectually normal patients with stereotypic behaviors occasionally do present to our OCD Clinic. Previously, we have described the use of fluoxetine in patients with skin picking.[5] In addition, we have recently used serotonin specific reuptake inhibitors for other patients who would meet DSM-IV criteria for stereotypic movement disorder.

***Case 1.*** A 34-year-old man presented with repetitive rocking behaviors and repetitive face picking. The patient exhibited schizotypal and borderline personality disorder traits. He stated that the repetitive behaviors had been present since adolescence. On fluoxetine 20 mg/day, the patient experienced a decrease in anxiety, which he stated allowed him to have better control over his repetitive behaviors. These exhibited a mild to moderate improvement within a few weeks. The patient elected to continue medication for over a year.

***Case 2.*** A 48-year-old woman presented with lifelong repetitive lip biting. She met DSM-IV criteria for major depression. Previous episodes of depression had responded to fluoxetine 20 mg/day, and this regimen was again initiated. There was a marked improvement in mood within a few weeks, but only a minimal improvement in lip biting. Increasing the dose to 40 mg daily had no additional effects on this behavior. The patient continued taking fluoxetine for over a year.

***Case 3.*** A 22-year-old man presented with repetitive head banging prior to falling asleep. This behavior consumed more than an hour of time each night and had persisted since childhood. Paroxetine 20 mg/day was initiated. The patient had no improvement in his behavior on this regimen and elected to discontinue the medication after 4 weeks.

***Case 4.*** A 53-year-old woman presented with repetitive checking behaviors that met criteria for OCD. In addition, she complained of repetitive rubbing of her nose. She stated that this was simply a habit, rather than a compulsion that she did to get rid of intrusive thoughts. Both OCD and nose rubbing had been present since childhood. The patient was treated with citalopram at doses of up to 60 mg/day. There was minimal improvement in OCD symptoms over 3 months and no change in nose rubbing.

***Case 5.*** A 32-year-old woman presented with symptoms of hair pulling since age 10. She met DSM-IV criteria for trichotillomania. The patient noted that when she was unable to pull hair, for example, in public situations, she would then rock back and forth repetitively. The patient was treated with citalopram up to 40 mg/day. Within a few weeks of medication, there was improvement in both hair pulling and rocking, but over time both symptoms returned.

***Case 6.*** A 30-year-old man presented with complaints of excessive nose picking of about 6 years in duration. The patient was ashamed of this behavior, which he felt unable to control. Citalopram 20 mg/day was initiated. After 4 weeks of treatment, the patient reported minimal improvement in his behavior. However, in view of difficulty with ejaculation on the medication, he elected to discontinue treatment.

***Case 7.*** A 28-year-old man presented with contamination fears and excessive hand washing that met DSM-IV criteria for OCD. The patient also gave a history of rhythmic shaking of the right foot, which he described as leading to a reduction of tension. There was no response of symptoms to dothiepin 150 mg/day for 6 weeks or to clomipramine 150 mg/day for 8 weeks. However, on fluoxetine 60 mg/day, there was marked improvement in both OCD and foot shaking over the course of 16 weeks.