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Scopolamine Patch for Clozapine-Induced Sialorrhea

To the Editor: A common complication of treatment with clozapine is the development of sialorrhea. Incidence rates of hypersalivation noted in four review articles varied from zero to 80 percent, and the rate in premarket testing was 31 percent (1-4). Increased salivation may be attributable to either clozapine-induced muscarinic M4 or adrenergic agonism (1-4). Because sialorrhea is generally greater at night (3), another possible mechanism is clozapine-related alteration of the circadian rhythm.

Recommended management of sialorrhea includes dose reduction, anticholinergic agents, a clonidine patch, and ophthalmic atropine (1-5). Also, pirenzepine, a muscarinic M1 receptor antagonist, is available, and it has been occasionally used for this purpose in Europe (3,4). However, in practice most cases of sialorrhea are not adequately treated, because available treatment options have limited efficacy or unwanted side effects.

One possible alternative that, to

our knowledge, has not been previously described in the literature involves the use of a scopolamine patch. We report the case of a woman taking clozapine who had profound sialorrhea and who achieved significant relief from her symptoms with a scopolamine patch.

The patient was a 44-year-old unmarried Caucasian woman who was given a diagnosis of schizoaffective disorder at age 19 and who had been in treatment since 1997 in the department of psychiatry at the University of Connecticut Health Center. Before 1993 she had undergone multiple trials of medications, including typical and atypical antipsychotics, mood stabilizers, benzodiazepines, and clonidine, to control frequent exacerbations of symptoms, with only partial response. She continued to have residual depressive and psychotic symptoms even when her condition was stabilized. The first clozapine trial began in 1993. Her illness went into substantial remission after she began taking clozapine at a dosage of 900 mg of a day, and she avoided hospitalization for eight years.

The patient experienced sialorrhea during this time, but the condition was not socially embarrassing and did not require management. Unfortunately, clozapine treatment was complicated by the development of morbid obesity—a weight gain of 120 pounds—and hypercholesterolemia. Because of a strong family history of cardiac disease, the patient became concerned about the risk of premature cardiac-related death and decided to stop taking clozapine in August 2001. She clinically decompensated, and frequent inpatient stays were required. Neither trials of the newer atypical antipsychotics nor a course of electroconvulsive therapy significantly minimized the patient's psychotic symptoms.

The decision was made to reintroduce clozapine. Five weeks after the treatment was initiated, profound sialorrhea developed at a daily clozapine dosage of 500 mg. All attempts to decrease the dosage did not reduce hypersalivation and led to increased psy-

chopathology. Controlled-release tolterodine at a dosage of up to 4 mg a day in divided doses, a clonidine patch in dosages of up to .3 mg every 24 hours, and benztropine did not reduce the sialorrhea. The patient then started transdermal scopolamine at a dosage of 1.5 mg every 72 hours. Sialorrhea resolved entirely within hours. The benefits have persisted for many months.

The antimuscarinic scopolamine in transdermal form represents an effective option for the treatment of clozapine-induced sialorrhea. Although it can cause the same wide array of anticholinergic side effects as benztropine and other drugs of this class, the transdermal delivery allows for a highly controlled and minimal dose. As evidenced in this case, it is well tolerated and convenient. Its therapeutic value in the treatment of hypersalivation should be further investigated in a controlled prospective study.

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Trauma and Tragedy in New York City

To the Editor: Dr. Satel contends in her editorial in the December issue (1) that posttraumatic psychopathology never really materialized among

New Yorkers after the terrorist attacks of September 11, 2001.

A concerning aspect of this analysis is the unintended effect it may have if it is interpreted as a dismissal of the trauma experienced by the people of New York. Widespread subsyndromal posttraumatic stress disorder (PTSD) disrupts communities, both psychologically and economically. We know from important scholarly work led by the New York Academy of Medicine that persons who were exposed to the attacks did suffer in greater proportions than those who were not (2). In addition, Weissman (3) showed a statistically significant rise in PTSD treatment after September 11, 2001, albeit without clear causality.

Our challenge is to devise methods to reliably measure and intervene when broad social trauma strikes so that the mental health system can provide essential clinical services, and, in so doing, improve the mental health and social and economic conditions of affected communities.

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In Reply: We must not confuse human response to tragedy with pathology. To date, not a single published epidemiological study of PTSD after September 11 has been able to render diagnoses—the methods used were too limited. Common sense tells us that some small fraction of people surely met formal criteria for a mental illness as a result of the events of September 11, although the studies

are unable to tell us how many. However, Dr. McQuiston's seeming eagerness to portray normal, if painful, reactions to a catastrophe as clinical sequelae only fuels the perception that citizens are psychologically fragile in the face of terrorism.

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WHO Revises Draft of Manual on Legislation

To the Editor: In the Taking Issue column in the September 2002 issue of *Psychiatric Services* (1), I criticized a draft manual circulated by the World Health Organization (WHO) that was intended as a guide to mental health legislation around the world. The manual's provisions were similar if not identical to those of antipsychiatry ideologues and of the self-appointed legal advocates who in the 1970s denied the reality of mental illness and the efficacy of medical treatment for patients with serious mental disorders. Under the banner of reform, the legal requirements set forth in the manual would have created costly and counterproductive obstacles to psychiatric treatment.

I am delighted to report that WHO has completely revised its draft manual, taking into account the criticisms in that Taking Issue column. I believe it is now appropriate for American psychiatrists to endorse WHO's efforts and to thank those involved for their responsiveness to the detailed criticisms they received from experts from the American Psychiatric Association as well as from me. I also thank the journal's editor, John A. Talbott, for his willingness to publish the criticisms, which ruffled feathers but seem to have had a salutary effect.

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Pentagon Employees After September 11, 2001

To the Editor: In the October 2003 issue of *Psychiatric Services*, Dr. Grieger and his colleagues (1) reported the results of a study that showed a 14 percent prevalence of "probable" posttraumatic stress disorder (PTSD) among survivors of the September 11, 2001, attack on the Pentagon. The study had serious methodologic problems, quite apart from the very low survey response rate (11 percent). The authors provided insufficient detail of the scoring methods and distribution of responses for the Impact of Events Scale-Revised (IES-R) to support their conclusions about the prevalence rate. There are five possible responses to the instrument's 22 questions about symptoms: not at all, a little bit, moderately, quite a bit, and extremely (2). Dr. Grieger and his colleagues apparently scored any affirmative response as a symptom endorsement, meaning that even the response of "a little bit" was counted as positive. Thus, if participants responded in this way to one question about intrusive thoughts, three questions about avoidance symptoms, and two questions about hyperarousal symptoms, they would have screened positive for "probable PTSD."

Although it can be argued that this approach follows the basics of *DSM-IV* criteria, it also means that persons with total IES-R scores as low as 6 could be included in the "probable PTSD" category. No published studies provide support for the validity of this approach, and the approach is inconsistent with scoring methods established for the original 15-item IES or for related instruments such as the PTSD Checklist (2,3). It is also highly unlikely that a person whose responses reflected this minimal level of symptoms would meet *DSM-IV* PTSD criterion F—clinically significant distress or functional impairment.

Although the original IES and the IES-R differ in response formats and scoring, data from one of the coauthor's own studies (4) provide some