symptoms of menopause (hot flushes and excessive perspiration) in 63% to 80% of 50 patients treated with VRD.

Despite its antidopaminergic action, VRD extrapyramidal side effects are reported as extremely uncommon.[2] However, isolated case reports described parkinsonism induced by VRD, especially when dosing recommendations had not been respected.[3] Four cases of tardive dyskinesia (TD) induced by VRD have already been described.[4–7] Here, we describe a case of severe lingual TD following long-term treatment with VRD.

**Case Report**

An 81-year-old woman was admitted to a psychiatric intensive care unit of a general hospital for acute mania. She presented excitement, mood elevation, olfactory hallucinations, and persecutory delusion. In the past, she had never been treated with any psychotropic drug. However, she acknowledged past use of VRD to treat menopausal syndrome, for at least 7 years. She had never taken any other non psychotropic drug with antidopaminergic activity. On visit, a severe lingual TD was present (Abnormal Involuntary Movement Scale Score: 4). Although her movement disorder was severe and esthetically repulsive, the patient, who wore dentures, was unaware of it. She was treated with olanzapine (up to 15 mg/day) and valproate (up to 400 mg/day), with minimal improvement of her psychotic symptoms. The severity of the lingual TD remained stable.

**Comment**

To our knowledge (MEDLINE, September 2003), this is the fifth case of TD observed after treatment of VRD. Since about 5% of old subjects present abnormal movements indistinguishable from typical TD, it is not possible to establish firmly a causal relationship between the treatment with VRD and the development of the movement disorder. Nevertheless, the risk of TD in subjects exposed to antidopaminergic drugs is at least four times greater than in subjects who had never been treated with antidopaminergic drugs. Furthermore, iatrogenic disorders resembling natural illnesses should have diagnostic priority.

The introduction of novel antipsychotics led to a significant decrease of extrapyramidal symptoms in patients affected by psychotic disorders. However, the widespread use of non psychotropic antidopaminergic drugs (e.g., VRD, metoclopramide, domperidone,) in nonpsychiatric patients, mostly prescribed by physicians with poor experience in neurological side effects, remain a serious underrecognized risk of parkinsonism and tardive syndromes.

In case of extrapyramidal signs arising in women not exposed to antipsychotic drugs, previous use of VRD should be investigated carefully. Likewise, extrapyramidal signs should be looked for in women treated with this drug.

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## Low-Dose-Trazodone-Induced Disorganized Type Psychosis

*SIR:* Trazodone, an antidepressant, rarely induces psychotic symptoms.[1] The doses used for the treatment of depression range from 150 to 600 mg/day.[1] One depressive patient has been reported to develop psychosis following trazodone administration at a dose of 300 mg/day.[2] The patient's psychotic symptoms included auditory hallucinations with threatening voices, agitation, stupor, negativism, and delusions. We herein report a case of a depressive patient who developed disorganized type psychosis shortly after taking low-dose trazodone (25 mg/day).

**Case Report**

A 28-year-old woman, who met DSM-IV criteria for major depression without any psychotic features, attempted suicide by jumping off a building and was thereafter admitted to a hospital for a pelvic fracture. She had been prescribed several antidepressants for 5 years, but drug compliance was irregular. She had a manic episode, including elated mood, agitation, irritability, disinhibition, and decreased sleep after the administration of tricyclic antidepressant. She recovered from the laparotomy for pelvic fracture but could not stay in bed due to her delirious symptoms. When she was referred to our hospital, she presented with confusion, disorienta-

tion, memory deficit, and insomnia. Her Mini-Mental State Examination (MMSE) score was 15, thus suggesting the presence of a cognitive dysfunction. An electroencephalogram (EEG) showed a focal slowing of activity in the left temporal lobe. The administration of haloperidol was initiated at a dose of 1.5 mg/day. Two weeks later, she began a rehabilitation program, which included physical therapy, to recover from delirium. For the treatment of her depressive mood and insomnia, trazodone was additionally prescribed at a dose of 25 mg/day. Three days after the addition of trazodone, she developed psychotic symptoms, including auditory and visual hallucinations, incongruous ginning, grimacing, monologue, and loosening of association. She often burst into laughter for no apparent reason. In contrast, she did not demonstrate any disturbance of consciousness, disorientation, elated mood, or agitation. At that time, her EEG findings were within the normal range, and her MMSE score was 28. She could not participate in physical therapy for her psychotic symptoms. Trazodone was discontinued, and haloperidol was increased to a dose of 6 mg /day. Approximately 2 weeks later, her psychotic symptoms abated, and she resumed participation in rehabilitation programs.

**Comment**

Trazodone has little effect on histaminergic, cholinergic, or dopaminergic transmission[3] and has a unique dual pharmacological profile since it both inhibits the synaptic serotonin reuptake and blocks of postsynaptic serotonin receptors.[3] At low doses, trazodone acts as a serotonin antagonist, whereas at high doses, as a serotonin agonist.[4] An increase in the subcortical serotonin function is postulated to be involved in the pathophysiology of psychosis.[5] In the present case, an alteration in the serotonin transmission induced by low-dose trazodone administration may contribute to the occurrence of psychosis.

The administration of trazodone is usually started at 25 mg/day.[1] Based on the above findings, it may be necessary to carefully monitor the mental status of patients after the administration of low-dose trazodone.

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## Obsessive and Compulsive Symptoms in Schizophrenia Patients—From Neuropsychology to Clinical Typology and Classification

*SIR:* Paul H. Lysaker, Ph.D., et al.[1] reported interesting findings that are in line with a previous report by the same research group.[2] In their study, patients with prominent obsessive-compulsive symptoms (OCS) had higher levels of positive symptoms, emotional discomfort, and greater deficits in executive function than schizophrenia patients without significant OCS,[2] which is consistent with literature suggesting that OCS in schizophrenia is associated with a graver clinical picture. In particular, patients with compulsions also had higher levels of negative symptoms than schizophrenia patients without significant compulsions, even when level of obsessions was controlled statistically.[2] A recent study, which examined other neurocognitive domains, revealed that the obsessive compulsive (OC) group had more severe impairments in vigilance but demonstrated superior performance on a measure of visual memory.[1]

Taken together, these findings suggest that links demonstrated in these and other studies[3–5] between OCS and clinical and neurocognitive features of schizophrenia have nonlinear and often unpredictable pattern, which raises the question of causality. Schizophrenia patients with concurrent OCS constitute an intriguing subgroup of individuals. The etiology of OC phenomena in schizophrenia remains unclear. There is significant overlap in the proposed functional circuits and dysfunction at the neurotransmitter level in OCD and schizophrenia, which may lead to co-expression of symptoms. The interactions are multiple and complex, especially in regard to the serotonin and dopamine pathways.[6]

Three main types of interrelation between OCS and psychotic disorder are described: 1) those whose OCS are independent from psychosis; 2) those whose OCS are partially related to their psychosis; and 3) those whose OCS represent a continuum of their psychosis. Clinically and anamnestically, we are able to distinguish three main subgroups of patients: 1) those who met the DSM-IV criteria for OCD before the development of schizo-