are responsible for inducing dystonia as explained.

There are many reports that show that blepharoclonus/Meige syndrome responds to clonazepam,[11] clozapine,[12] trihexyphenidyl, perphenazine, fluphenazine[10] haloperidol, L-dopa with deprenyl, botulinum toxin A,[13] quetiapine.[14] This patient responded to haloperidol but did not improve with anticholinergics and atypical neuroleptics. This could be affected by a different receptor profile of typical and atypical neuroleptics. Conventional antipsychotics, like haloperidol, bind to D2 more efficiently than atypical drugs and 5HT2A binding of atypical drugs reverses the D2 blockade in nigrostriatal pathway. In addition, GABA concentration in the pallidum is also regulated by direct effect of D2 binding drugs in the pallidum where they can either decrease[15] or increase extracellular GABA,[16] thereby influencing thalamic activity.

In conclusion, to date we are not able to explain the pathophysiology of dystonia completely and further research is required to understand the differential effects of drugs.

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## Using Pramipexole in Neuropsychiatry: A Cautionary Note

*SIR*: Recent reports have recommended using the D2/D3 agonist pramipexole as a treatment for various psychiatric disorders such as depression in Parkinson's disease, treatment-resistant depression and bipolar depression.[1–3] These reports have not adequately emphasized the psychiatric toxicity of pramipexole. While hallucinations have been described as side effects of pramipexole,[4] there are few reports of other psychotic symptoms such as delusions. A recent case report described delusional jealousy arising in the context of mania.[5] We report a case of acute nonaffective psychosis with delusional jealousy developing on pramipexole treatment of Holmes' tremor, remitting on stopping the drug, relapsing on rechallenge with a lower dose and gradually remitting with complete stoppage.

**Case Report**

"Mrs. A," a 37-year old Caucasian woman, had a subarachnoid hemorrhage 7 years ago from a basilar tip aneurysm following which she was left with a spastic hemiparesis in the right limbs, partial third nerve palsy, mild dysarthria and right-sided Holmes' tremor. She had been taking daily doses of frusemide, 20mg, mirtazapine, 45mg, tizanidine, 12mg, and amantadine, 300mg, for several years. Three months prior to the current admission, a daily regimen of pramipexole, 2.1mg, was added and the dose increased to 2.8mg daily after 2 months. Three to 4 weeks after the increase in dose, she started hearing a buzzing sound that she associated with a mobile phone and muffled sounds which she identified as the

voices of her husband and his brother. Later she developed the belief that her husband had bought a secret mobile phone because he was having an affair with another woman and was leaving the house to meet this woman as soon she fell asleep. On some occasions she acted on her belief, searching and confronting her husband repeatedly in an attempt to find the mobile phone and was verbally and physically aggressive at times. Later, she also experienced visual hallucinations, seeing her husband through her bedroom window. There were no symptoms of depression or mania. There was no alteration in consciousness and she was fully aware of time, place, and person.

There was no history of misuse of illicit drugs or alcohol. Physical examination did not reveal new findings. All laboratory tests were within normal limits. A urine screen for drug abuse was negative. As an initial measure, the regimen of pramipexole was stopped on admission. From the second day of admission, an improvement was observed. The frequency of the auditory hallucination decreased significantly with only two episodes, each lasting less than 1 minute, reported. No visual hallucinations, delusions or other psychotic symptoms were elicited. Pramipexole was reintroduced at 1.05mg daily and within a week, the buzzing sound and the delusion of infidelity recommenced. This led to further aggressive behavior, and therefore it was decided to stop pramipexole completely. In a few weeks the symptoms started to resolve and they finally disappeared in 2 months, although this was accompanied by a moderate worsening of the tremor.

**Comment**

As this patient was receiving another dopaminergic drug, amantadine, and mirtazapine, also reported to precipitate psychosis, at the time of onset of the psychosis, it is impossible to be certain that pramipexole caused the psychosis. But the temporal correlation with increase in dosage of the drug and the time course of improvement on discontinuation point to the involvement of pramipexole in the onset of psychosis. Psychiatrists need to be vigilant for the emergence of psychosis, particularly when using pramipexole in patients at risk for psychosis.

Iatrogenic psychiatric presentations may also serve as models for understanding the development of delusions. The content of psychopathology, the theme of jealousy in a married woman with severe and long-term physical disability, may be explained in psychosocial terms though not the form of the delusion itself. An overarching formulation to explain both form and content of psychopathology would be along the model proposed by Kapur, linking hyperdopaminergic states and aberrant salience attached to external objects and internal representations.[6] The study of drug-induced delusions may also be helpful in advancing our understanding of the cognitive neuropsychiatry of delusion formation.[7]

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## Interictal Psychosis After Stroke With Forced Normalization

*SIR*: The World Health Organization defines a stroke as rapidly developing clinical signs of a focal (or global) disturbance of the cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin. A stroke represents a major public health problem, but relatively little work has been directed toward identifying and treating the common neuropsychiatric disorders occurring after stroke.[1] Psychosis is a rare complication of stroke, while 2% to 7% people with epilepsy have been reported to suffer from psychosis. The prevalence of psychosis in patients with temporal lobe epilepsy and/or refractory epilepsy varies from 10% to 19%, in most studies.[2] Forced normalization is often observed in psychosis of epilepsy. In 1953, Landolt[3] described a group of