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Ejaculation associated with zuclopenthixol

SIR: We report a patient who, we believe, suffered spontaneous ejaculation as a side-effect of oral zuclopenthixol.

A 22-year-old man with a four year history of a schizophrenic illness punctuated by marked depressive and hypomanic episodes, was admitted in a state of depression with delusional ideas of guilt. He was treated with ECT ($\times 2$), lithium carbonate (800 mg/day), sulphiride (1 g/day), paroxetine (20 mg/day) and chloral hydrate (1 g/day) regularly and oral zuclopenthixol (10–20 mg) only as required for agitation.

Following the resolution of his psychotic ideation, he complained for the first time of spontaneous ejaculation in the absence of any state of sexual arousal or penile erection. This occurred only after taking oral zuclopenthixol and the ejaculations remitted once this was stopped, despite no changes in other medications.

Spontaneous ejaculation has not previously been reported in association with zuclopenthixol although it has been reported with trifluoperazine and thiothixene (Keitner & Selub, 1983). More usually, zuclopenthixol and other neuroleptics are associated with impotence and painful, reduced or absent ejaculation (Sullivan & Lukoff, 1990). Thioridazine has been particularly implicated (Kotin *et al*, 1976) and it is thought that it may act by a neuroleptic-induced blockade of smooth muscle calcium channels (Pollack *et al*, 1992). In this case, as ejaculation occurred independently of either erection or sexual arousal, it seems likely that it occurred by triggering an isolated, spontaneous contraction of vas deferens smooth muscle. Vas deferens contraction is mediated by post-synaptic alpha-1 receptors (Pollack *et al*, 1992) and although zuclopenthixol is usually known for its antagonistic activity at these receptors (Hyttel *et al*, 1985), only occasional use could perhaps cause the zuclopenthixol to have a partially agonistic effect, which would offer a possible explanation for this side-effect.

Adverse genito-urinary effects of psychotropic medication are vastly under-reported by patients, principally because of their difficulty in raising the issue. Although not previously reported this side-effect may be quite prevalent.

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Sleep disturbance in schizophrenia

SIR: In their review on electro-encephalographic sleep in schizophrenia, Keshavan *et al* (1990) concluded that the following sleep EEG findings are present in schizophrenic patients more frequently than in normal controls: decrease in total sleep time; decreased percentage of slow wave sleep; reduced REM-latency in some patients and reduced REM-compensation following REM-deprivation. However the results in relation to sleep EEG findings may reflect the variability in the studies (medicated v. non-medicated patients, chronic v. acute schizophrenics, and varying diagnostic criteria) and may be due to the heterogeneity of the schizophrenic syndrome as well.

We asked 101 clinically stable schizophrenic outpatients, all using oral neuroleptic medication, to co-operate in a short questionnaire about medication use and their health care needs. Besides other questions, four questions concerned their sleep: (1) do you have problems with your sleep? If yes, (2) do you have problems with falling asleep? (3) do you have problems with maintaining sleep? (4) do you have problems due to early awakening?

The questionnaire was administered by an independent interviewer. Demographic and psychiatric data (age, sex, diagnosis according to DSM-III-R (American Psychiatric Association, 1987), and type of medication) were collected by their therapist. Statistical analysis was performed by means of SPSS-PC.

Eight patients did not participate, so 93 patients (46 men, 47 women; mean age 47 years; range 20–75) were included in the study. No patients had acute psychotic symptoms. Thirteen different oral neuroleptics were used of which flupentixol ($n=22$),