Shader, 1977). The few cases that have appeared in the literature in the past 30 years, including those occurring in children (Holder, 1988), have had similar features. An atypical case is presented of a man with an affective psychosis in clear consciousness.

Case report. A 33-year-old entertainer with no family or personal history of psychiatric illness was brought to hospital in an agitated state. Five weeks earlier he had been prescribed proguanil (200 mg/day) and chloroquine (300 mg/week) as antimalarial prophylaxis before a holiday to Africa. After each dose of chloroquine he had noticed a brief period of arousal. On return from holiday he took a dose of 600 mg of chloroquine in error, after which he was mildly irritable and overactive. He continued to take both drugs in the prescribed dosage and became increasingly disturbed over the following week. On admission to hospital he was overactive, irritable, and talkative, experiencing racing thoughts and expressing delusions of reference and grandeur. He was fully alert and orientated and there were no features suggestive of a confusional state. Physical examination, and haematological and biochemical investigations were all normal. A diagnosis of hypomania was made and he received a single dose of 5 mg haloperidol. His mental state returned to normal within three days and the antimalarial prophylaxis was reinstated. The day after receiving a further 300 mg of chloroquine he again developed features of a hypomanic episode without confusion. Recovery was complete after three days and he was discharged. Three weeks later he developed a brief episode of agitation without psychosis and without taking further medication. He has remained well in the year of follow-up.

I believe this is the first reported case of a true manic episode without confusion in response to chloroquine. This drug has also been reported to cause involuntary movements of extrapyramidal type (Umez-Eronini & Eronini, 1977), and it is possible that these two rare adverse effects may be mediated by a common dopaminergic pathway. Doctors prescribing antimalarial medication routinely should be aware of the neuropsychiatric complications, and psychiatrists should be as alert to the importance of a history of recent travel as their physician colleagues.

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Catatonia and NMS

SIR: I read with interest the article entitled "Catatonia: harbinger of the neuroleptic malignant syndrome" (*Journal*, March 1991, **158**, 419–421) by Drs White & Robins. I would like to describe a case of neuroleptic malignant syndrome (NMS) consistent with their findings.

Case report. A 53-year-old single man, with a 30-year history of recurrent presentations of schizophrenia with catatonic symptoms, presented on this occasion with onset of catatonic symptoms (mutism, negativism, odd posturing, and waxy flexibility) over a few days. This was his usual mode of presentation. Physical examination on admission revealed a tachycardia of 130 and a temperature of 38.5°C. In view of the pyrexia, autonomic symptoms and catatonia, further investigation for NMS was undertaken. A rising level of 5000 Iv/l of creatine phosphokinase (CPK) was noted. He was transferred to a medical ward and received symptomatic and supportive treatment with full recovery. His catatonic symptoms also improved.

In this case, the only differences from his usual mode of presentation over many years were the presence of autonomic symptoms and hyperpyrexia with raised CPK. The hypothesis advanced by Drs White & Robins could explain the emergence of NMS in this case. Their hypothesis is based upon reports of a central dopamine deficiency in NMS (Fricchione, 1985; Horn *et al*, 1988) and the use of reduced diencephalic dopaminergic transmission to explain hyperthermia and catatonic signs in lethal catatonia (Mann *et al*, 1986). They suggest that already deficient dopaminergic activity in the brain of catatonics would be aggravated by the dopamine blockade produced by neuroleptics.

I would like to suggest that the possibility of NMS should be considered in anyone presenting with catatonic features. The reasons for this include: catatonic features may be a presentation of NMS, even in patients with previous presentation of catatonia; catatonia may predispose to NMS as hinted by Drs White & Robins; and, in the presence of catatonia, unless specifically looked for, other signs of NMS may not be obviously apparent. Perhaps, every patient with catatonia should have serum CPK levels checked.

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