I report such a patient.

V.M., a male aged 82, was referred to the psychiatric department following a paracetamol overdose, but his blood levels were found to be far below the expected amounts. He gave a history of periods of severe depression and anxiety following the death of his wife (an opera singer), daughter and stepson in a car-accident in Germany, eight years previously. He described various classical symptoms such as tearfulness, early morning wakening, diurnal variation of mood, ideas of guilt, loss of interest in his usual hobbies, and auditory hallucinations of voices, accusing him and ordering him to kill himself. He admitted to one previous psychiatric admission in Germay, where he had been treated with monoamine oxidase inhibition, but he could not remember the precise address of the hospital.

On examination he appeared very depressed and agitated, and was noted to exhibit restlessness, hand-wringing and a severe tremor. No informants could be interviewed, as the patient claimed to have no friends or relatives in this country and did not want us to contact his place of work.

A diagnosis of agitated depression was made and the patient was admitted to his catchment area hospital, where he was immediately recognised by the admitting doctor. It appeared that he had a history of numerous psychiatric admissions dating back at least six years, under various names. His last admission had taken place three months earlier, and was terminated when he was arrested in a bank for attempting to draw money under yet another false name.

On this admission he improved within a few days and was discharged to the care of his protion officer a fortnight later.

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RAPID TREMOR OF THE EYELIDS AFTER OVERDOSE OF FLUPHENAZINE

DEAR SIR,

A 20 year old man with 5 admissions, in the previous 17 months, and diagnosed variously as suffering from schizo-affective or manic-depressive (manic) psychosis, was admitted after an overdose of 80 × 5 mg fluphenazine tablets and 6 × 0.5 mg benztropine tablets, 2 days before. On examination he had a rapid twitching of his upper eyelids in both the closed and open positions, synchronous with tremor of his hands and tongue. There was a superimposed slow spontaneous blinking of 8 blinks per minute (normal ± 12 blinks per minute—Carney and Hill, 1982). Ocular movements were normal. The glabellar reflex showed non-habituation, and there were other Parkinsonian features. There were no signs of tardive dyskinesia; nor of psychosis.

He was given benztropine 2 mg intravenously. Within 20 minutes the eyelid tremor had markedly decreased. He reported feeling less stiff, and there was decreased jaw stiffness. The glabellar response remained non-habituated. He was subsequently given benztropine 2 mg bd orally. One week later, the tremor of the eyelids had ceased, but the fine tremor of the hands persisted, and blinking was still slow at 10 blinks per minute. The glabellar reflex habituated after 3 taps.

Blink rate is decreased in Parkinson's disease (Hall, 1945), the latter sometimes being indistinguishable from drug-induced Parkinsonism (Baldessarini, 1980), but eye blinking is increased in schizophrenia and tardive dyskinesia (Stevens, 1978), and in Gilles de la Tourette syndrome (Cohen *et al,* 1980).

Penders and Delwaide (1971) showed a return towards normal eye movements in Parkinsonian patients treated with L-dopa or amantidine. Conversely, dopamine blockade by neuroleptics reduces the blink rates and the thought disorder in schizophrenics (Karson *et al,* 1981a). Reduction of dopamine blockade as in Stevens' patients (Stevens, 1978) who were medication-free for 1–6 months, leads to an increase in blinkrate. Also, Karson *et al* (1981b) found an increased blink rate to apomorphine after haloperidol discontinuation. The central role of dopaminergic blockade in abnormal eye movements was further illustrated by the finding (Karson *et al,* 1983) of an inverse relationship between spontaneous blink rate and platelet monoamine oxidase activity.

Here, there was a decrease in spontaneous blinking, in keeping with the picture found in drug-induced Parkinsonism i.e. a dopamine blockade. The rapid tremor of the eyelids, synchronous with the tremor of the hands and ameloriated by benztropine, is thought to be an unusual extrapyramidal neuroleptic side-effect analogous to the perioral "rabbit" tremor described by Jus *et al* (1974), (which is also relieved by anti-Parkinsonism medication (Sovner and Di Mascio, 1978)). I think that the mechanism involved in the abnormal eyelid movements was a massive dopamine

blockade, perhaps exacerbated by cholinergic overactivity during withdrawal, (Gardos *et al*, 1978) and relieved by an anti-cholinergic drug.

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SOCIAL PHOBIA

DEAR SIR,

We were interested to read the paper by Amies *et al* (*Journal*, February 1983, **142**, 174–79) that presents the distinguishing features of social phobia and agoraphobia and implies a heterogeneity within the term phobic neurosis in ICD-9 (WHO, 1978). Social phobia has a specific definition in the DSM III (APA, 1980), consisting of a fear of criticism and scrutiny in situations of public eating, speaking, writing or urination, with consequent avoidance. The definition used by Amies *et al*, however, was more general, viz. "anxiety experienced in the company of other people . . . increased with formality and the extent to which the person feels under scrutiny". Following this wider definition, they found that the situation feared mostly by their group was that of being introduced to other people.

We recently surveyed forty-six patients referred to the Maudsley Hospital, London, whose main complaint was a difficulty in initiating and maintaining conversations, especially with strangers of the opposite sex, at parties and other social gatherings. Like Amies *et al*, we found that the majority were single men, whose problem began in their second decade of life, although over 60 per cent of our group reported having no friends in childhood. Agoraphobics and social phobics defined by the DSM III were not included. Considering the very early development of the problem, we concluded that this group could be subsumed under the DSM-III term, "avoidant personality disorder" viz difficulty in relating comfortably to others, with social withdrawal, and a fear of rejection and humiliation. Unlike schizoid personality disorder, this group wishes to be able to socialize, and all our patients approached the clinic with this as their main problem.

We suspect that many patients of Amies *et al* would be better classified as having an avoidant personality disorder. The therapeutic implication of such a division may well prove to be that social phobia, like other phobias, responds to anxiety-reducing methods such as exposure (Marks, 1981), while avoidant personality disorder requires social skills training, which includes instructions and role-play of improved social performance in addition to exposure (Stravynski *et al*, 1982).

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