It appears that nearly half (23/50) the patients in the study were on concomitant anticholinergic medication. Quite a few items of LUNSERS which are meant to measure neuroleptic side-effects are a result of their anticholinergic properties (i.e. dry mouth, constipation, difficulty in passing water, blurred vision, restlessness, etc.). Hence, it must be very difficult to deliberate how much of these could be attributed to neuroleptics alone and how much to the additional anticholinergic medication. Perhaps a differential analysis of the scale for those who were and those who were not on anticholinergics would help increase the validity of the scale as a reliable self-measure of neuroleptic side-effects.

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Dystonia and neuroleptic medication

SIR: Psychiatrists should know more about the possible, but rare, dystonic side-effects of neuroleptic medication, with which I have had an unpleasant experience, to say the least.

Twelve years ago I suffered a severe breakdown, diagnosed as schizophrenic, and was hospitalised for 4 months. I made a good recovery but I have been on a small dose of Depixol almost constantly since then, despite numerous attempts to wean myself off it.

Nine years ago, I started developing problems with my voice. It began as an intermittent and fairly mild affliction, but degenerated over the next three years to a serious condition which affected every area of my life profoundly including my career. At times when it became so bad that I could hardly speak, I became a semi-recluse. Over the years I was seeing several different psychiatrists. They described the voice as an anxiety-related speech disorder. I saw two speech therapists who said it was a psychological problem. An ENT consultant could detect no physical problem. One of the speech therapists referred me to a psychologist, who told me that I would probably never be free of the problem.

Eventually, under pressure from my mother, I got myself referred to a third speech therapist, early last year. She suspected from the beginning a condition called spasmodic dysphonia, or laryngeal dystonia. The question of whether this could be caused by Depixol arose, on my instigation, but research by her produced no positive answer. My current psychiatrist, who was consulted, declared that he doubted that there was a connection.

On 24 January of this year I went to the National Hospital of Neurology and my voice problem was diagnosed as laryngeal dystonia. The neurologist confirmed that it must have been caused by my neuroleptic medication. I was injected in the laryngeal area with Botulinum toxin, and within 48 hours I was speaking with an ease and lack of embarrassment that I had not known for years. My self-esteem has soared and I was quickly able to reduce my medication due to lack of stress when speaking.

My psychiatrist for the last three years has since admitted that he has come across one identical and one similar case in his career, so I don't understand why he had doubted the Depixol link, or why, indeed, he had not twigged long ago. But my experience is that there is general ignorance in psychiatric circles about the various manifestations of dystonia, which, as you will know, is a well-known side-effect of neuroleptic medication.

So please do something about this ignorance in psychiatric circles. I would not wish anyone else to go through unnecessarily what I have suffered, especially as people on neuroleptic medication are by definition a mentally fragile group.

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Charles Bonnet syndrome

SIR: I read with interest the findings of Teunisse *et al* (1995) and wish to report a case of Charles Bonnet syndrome (CBS) in an elderly woman with no visual impairment.

LL, a 83-year-old woman, was well until two and a half years ago when she started having isolated visual hallucinations. She experienced these hallucinations at night just before falling asleep. On several occasions she called her neighbours and police having seen strange things around her, for instance a small white robot standing in her room, which turned to look at her and then suddenly disappeared; a large tree which stood in her doorway; a giant spider and ducks that flew in to her room through a closed window. She had full insight and described these as imaginary experiences which seemed very real. She denied history suggestive of hallucination in any other sensory modality, other disorder of thought or perception or cognitive dysfunction. She had normal eye sight and used glasses only for reading purposes. On examination

she did not have any neurological deficit. Investigations including EEG and CT scan of the head did not reveal any abnormality.

Some believe that impaired visual acuity and a disorder of brain function are both required for CBS to develop but it is also known to develop in individuals with normal vision (Podoll *et al*, 1989). There is no consensus on whether pathology in the visual system is necessary, possible or incompatible with the diagnosis of CBS (Hecaen & Albert, 1975).

In this patient, complex, isolated, persistent and recurrent visual hallucinations were hypnogogic in nature. Lesions of diencephalon and also diffuse lesions of cortex can produce hypnogogic hallucinations which are predominantly or solely visual, and the aetiology of CBS may lie in structural or functional abnormalities of this region.

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SIR: We commend Teunisse *et al* (*BJP*, February 1995, **166**, 254–257) for the first ever major case finding study using fixed criteria and a control group. However the authors' assertion that in other prevalence studies on complex visual hallucinations in visually impaired patients, the concept of Charles Bonnet syndrome (CBS) was not used is somewhat unjustified.

There is considerable confusion with the use of the term CBS by both psychiatrists and ophthalmologists. Originally defined as visual hallucinations in elderly patients without evidence of ophthalmological impairment, the term has been modified over the years to include visual hallucinatory phenomena of a pleasant or neutral nature in conjunction with a clear state of consciousness (Damas-Mora *et al*, 1982). Within this modification is the fact that it is often associated with ocular disease. There seems to be no consensus however, about the relationship between eye pathology, brain lesions and CBS. At least two definitions exist (Damas-Mora *et al*, 1982; Gold & Rabins, 1989). The authors criteria in the study are almost identical to the criteria proposed by Gold & Rabins.

Two recent prevalence studies not mentioned by the authors are worthy of mention. Norton-Willson & Munir (1987), in a retrospective study of 434 consecutive patients referred for consultation to a psychogeriatric unit over a period of 3.5 years, reported eight cases of visual perceptual disorders resembling CBS. Brown & Murphy (1992) studied 100 consecutive patients with macular choroidal neo-vascularisation in a cross-sectional fashion and reported 12 subjects with formed hallucinations (Charles Bonnet syndrome). These authors have not defined or used any criteria for CBS preferring instead to use the term visual perceptual disorders or formed visual hallucinations.

We suggest that if systematic research into this interesting phenomenon should meaningfully progress, some consensus on the criteria for CBS should be arrived at, or else we risk having to give up the use of this eponym which has so far stood the test of time.

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The cultural context of hallucinations

SIR: Al-Issa (1995) is right to emphasise the importance of the cultural context of hallucinations. But his discussion of the origins of hallucinations in terms of cultural attitudes appears incomplete.

Al-Issa distinguishes "rational" cultures, which make a rigid distinction between reality and fantasy, from the "less rational" cultures which have a more flexible distinction. In his view, a rigid distinction promotes negative attitudes towards hallucinations and makes people less introspective, less familiar with the workings of their own imagination, and so less aware of imaginings such as hallucinations.