Case report: A 44-year-old man of Polish descent first presented to the psychiatric services in 1982 with recurrent unipolar depressions. His subsequent treatment was complicated by his suffering significant side-effects on most first-line antidepressants. Those that he was able to tolerate (e.g. low-dose clomipramine) tended to induce hypomania after a few weeks of treatment. It was in some desperation, therefore, that I put him on L-tryptophan in 1985. Such was his apparent sensitivity to psychotropic medication that he became hypomanic on 4.5 g of L-tryptophan per day. During the next few months he became very stable on 1·5 g of L-tryptophan per day and he was discharged from follow-up in July 1985.

He was referred back by his general practitioner this August. It seems that he had remained entirely well over the five years previous to this, apart from a bout of depression with biological features every three months or so. At these points he took about 1·5 g of L-tryptophan over two days and found that this quickly restored his well being. When he was referred back to me he had been constantly depressed for three months with anergia, early-morning wakening, loss of appetite and anhedonia.

Another patient (a medical practitioner) who had suffered sleep disturbance on withdrawing from the L-tryptophan on which she had been maintained, had researched the topic and established that pumpkin seeds were the most cost-effective means of administering natural L-tryptophan: a typical 250 g bag contains 1300 mg and costs about £1.00.

Within 24 hours of ingesting about 200 grams of pumpkin seeds (i.e. about 1 gram of L-tryptophan), the patient felt quite transformed. He was no longer anergic or depressed and happily returned to work the following day.

Clearly this patient is atypical in that his mood state appears to be remarkably sensitive to L-tryptophan. However, for patients who were successfully maintained on L-tryptophan or for whom one wishes to instigate L-tryptophan, pumpkin seeds may represent the best alternative at present. Perhaps, as in this case, the biggest hurdle is overcoming the scepticism of one's patient!

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Down's syndrome, dementia and myoclonic jerks

SIR: The recent publicity surrounding bovine spongiform encephalopathy (BSE) has intensified the search for cases of the human concomitant, Creuzfeldt Jakob disease. The combination of a dementing process and myoclonic jerks in a person under the age of 70 years raises suspicions of such a diagnosis. I urge for caution before reaching this conclusion.

There are two accounts in the literature of a combination of Down's syndrome, dementia and myoclonus (Blumbergs *et al*, 1981; Good & Howard, 1982). Both were originally presumed to be Creuzfeldt Jakob disease but post-mortem findings indicated Alzheimer's disease which commonly occurs prematurely in people with Down's syndrome. Faden & Townsend (1976) draw attention to the confusion arising when myoclonus occurs in association with dementia, and assert that this should not be considered pathognomonic of Creuzfeldt Jakob disease and that a diagnosis of Alzheimer's disease should be considered. This difficult diagnostic problem is illustrated by the following case.

Case report: A is a 48-year-old single lady with Down's syndrome. She developed *grand mal* epilepsy three years ago and an electroencephalogram (EEG) at that time showed no evidence of a space-occupying lesion. She was successfully treated with phenytoin and seizures are now infrequent.

For the last 18 months there has been a steady deterioration in her memory and she is now unable to recognise close family members. Her speech has deteriorated and she spends much of her time muttering to herself. Her self-care has deteriorated and she has become doubly incontinent, necessitating full nursing care. Her motivation is poor and her mood is labile. One year ago she developed myoclonic jerks. These occur only 1–2 hours after waking and affect her head, arms, shoulders, body and legs. They are abrupt, brief, irregular, asymmetrical and can be so severe as to catapult her out of her wheelchair.

A repeat EEG shows generalised irregular 3–7 Hz slow activity, with no change from her EEG of 1987. In particular, there is no evidence of periodic biphasic or triphasic complexes characteristic of Creuzfeldt Jakob disease.

In the light of the experience of other authors, the time course of the clinical findings and the lack of specific abnormalities on the EEG of this patient, the diagnosis should be Alzheimer's disease until proven otherwise.

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How informed and binding is informed consent?

SIR: We report a case in which the patient's consent for an interview with him to be broadcast, given while he was well, had an affect on his mental state.

Case report: AB, a 37-year-old male homosexual teacher was diagnosed as having AIDS following pneumocystis carinii pneumonia. Initially, he accepted the diagnosis well but following a move to his new flat he became depressed. He withdrew from his friends and family, wished he were dead, but did not make any suicidal gestures. He was admitted to an acute psychiatric ward with the diagnosis of depressive illness, where he revealed that he was preoccupied with the idea that the interview he had recorded for a gay programme on television would reveal his sexual orientation and illness to the families of children he had taught previously. He feared that these families would harm him and the popular press would scapegoat him. He regretted having made the interview and had approached the television production team involved repeatedly, asking for his material to be withdrawn. He was informed that they had gained his written consent for the interview to be broadcast and intended to proceed with it. Following admission and noting his mental state, the medical team contacted the programme director and requested that material involving the patient be edited. The director consented after receiving the letter. This certainly helped alleviate the patient's worries, and following antidepressant medication and psychological treatment the patient was discharged.

As doctors, we are often critical of the way in which the broadcast and newspaper media collect and report information about patients. We present an example of the media behaving in the responsible manner, in cooperation with the medical staff, and the beneficial effect of such an intervention upon the patient's view of life and mood. While we are heartened by one experience in this case, the issue of informed consent for the broadcast of material containing medical as well as psychiatric patients, seems to be an area in which the best interests of the patients are often compromised by the desire of the journalists to collect visual images to accompany their stories about ward closures, living in the community and advances in medical technology. There seems to be an urgent need to review the issues involved in protecting patients from the media (especially the ones who may change their minds and withdraw their consent) in a society where a free press often comes close to compromising a patient's right to anonymity and confidentiality.

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CORRIGENDUM

Journal, October 1990, **157,** 626. The full reference should read *Journal of Mental Science,* 1890, **36,** 150–151. "Morton Hospital" should read "Horton Hospital".

A HUNDRED YEARS AGO

"Traumatic neuroses"

This is a convenient term, but, like other terms which are convenient, it is apt to include far too much. In this country the most important traumatic neurosis which is met with is no doubt the so-called "railway spine", and it may be that cases placed in this category are as varied as those which Hoffman (Berlin. Klin. Woch., No. 29) found among a series of twenty-four cases of "traumatic neurosis". Of those twenty-four, ten were found to have undoubted signs of organic mischief; in six the symptoms were partly the result of exaggeration and partly of simulation; in eight there was malingering, proved to be so after careful observation for several weeks. The author protests against the use of the term for such varied conditions, pointing out that in a so-called traumatic neurosis we may have to deal with organic nerve injury, with hysteria, the result of injury, with shock to the cerebrospinal system, with neurasthenia, or even with a true psychosis.

Reference

The Lancet, 17 January 1891.

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