the presence of prochlorperazine and an unspecified benzodiazepine in her urine; a packet of buccal prochlorperazine (Buccastem) from which 12 tablets (36 mg) had been removed was later found in her car but the source of the benzodiazepine was not identified. The abnormal eye movements resolved after twelve hours; she regained consciousness over 36 hours, declined psychiatric assistance and discharged herself from the ward.

In an unconscious patient the presence of ocular bobbing should raise the possibility of structural pathology in or around the pons. As these cases illustrate, however, other forms of spontaneous vertical eye movement disorder may be due to toxic or metabolic encephalopathies and ideally consideration should be given to such causes before embarking upon invasive investigation. Self-poisoning is by far the commonest cause of coma in patients presenting to casualty departments, but can often only be diagnosed on the basis of circumstantial evidence and subsequent toxicological screening.

Both of these patients were exposed to the unusual combination of phenothiazines and benzodiazepines. The pharmacological basis of the eye movements is not clear. It is possible that they represent a form fruste of oculogyric crisis produced by phenothiazines and modified by benzodiazepines. Typical phenothiazine-related oculogyric crises cause sustained upwards deviation of the eyes (often with a lateral component) associated with obsessional thoughts and a feeling of unease.[5] One of the present patients developed these typical episodes on regaining consciousness. Whilst unconscious, both patients had episodes of unsustained rapid conjugate upwards deviation of the eyes, interspersed by slower drifts back to the primary position. Benzodiazepines have complex actions[6] on the brainstem circuits which may be responsible for the generation and maintenance of vertical gaze both under normal circumstances and during oculogyric crises,[1 5] and have been used to treat the latter.[7] If the reverse ocular bobbing is indeed a modified form of oculogyric crisis one would expect it to be abolished by intravenous anticholinergic drugs, which almost always abolish typical oculogyric crises. This was not attempted during the episodes of reverse ocular bobbing here but, if validated in future cases, might form the basis of a simple bedside test for combined phenothiazine and benzodiazepine poisoning.

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MATTERS ARISING

Treatment of lower urinary tract dysfunction in patients with multiple sclerosis

In this paper[1] we concluded that simple treatments could improve the bladder symptoms in multiple sclerosis, but before embarking on therapy it is important to detect incomplete bladder emptying which can exacerbate detrusor hyperreflexia. The paper was the result of discussions by a group of urologists and neurologists convened in March 1991 by the European Study Group of SUDIMS (Sexual and Urological Disorders in Multiple Sclerosis).

Although a consensus was reached then on which therapies are effective and desirable, there was no agreement about what pretreatment investigations were necessary or who should arrange them. At a second SUDIMS meeting in Copenhagen in May 1992 the subject of minimal investigations was again discussed. Again the views of the neurologists and urologists remained essentially irreconcilable but there was agreement on those investigations a neurologist might reasonably arrange for a patient with MS and bladder symptoms. These are: 1) exclude urinary tract infection; 2) detect incomplete bladder emptying.

Anticholinergic medication alone is unlikely to correct symptoms of urgency and frequency in the presence of either of these two disorders. The neurologist should arrange and follow up these investigations if possible. Urological advice should be sought if the patient does not get better or suffers recurrent urinary tract infections.

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1 Fowler CJ, van Kerrebroeck Ph EV, Nordenbo A, Van Poppel H. Treatment of lower urinary tract dysfunction in patients with multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1992;**55**:986–9.

The use of multiple anti-dystrophin antibodies in Duchenne and Becker muscular dystrophy

A recent paper by Muntoni *et al*[1] describes the use of multiple anti-dystrophin antibodies in Duchenne and Becker muscular dystrophy. I was particularly interested in the discordance in staining between the various antibodies in the very mild or preclinical Becker phenotypes. Presumably this is explained by small in-frame deletions that affect antigenic regions of dystrophin identified by one antibody and not the other. Although Western blot analysis was carried out, and said to confirm the immunofluorescence results, no data are shown.

The question I would like to put to the authors is whether dystrophin (presumably of slightly reduced mulecular weight) from such asymptomatic or mildly affected Becker patients could be distinguished from normal dystrophin on Western blot analysis using an anti-dystrophin antibody that gives normal immunocytochemistry pattern. If this were the case, then it would obviate the need to use multiple antibodies to avoid underdiagnosing the mild or asymptomatic Becker phenotypes.

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1 Muntoni F, Mateddu A, Ganchetti C, *et al.* Dystrophin analysis using a panel of anti-dystrophin antibodies in Duchenne and Becker muscular dystrophy. *J Neurol Neurosurg Psychiatry* 1993;**56**:26–31.

Muntoni et al reply:

Dr Kyriakide's observation gives me the opportunity to clarify my view on this important matter. The underlying question asked is: is there a single, preferred technique for detecting dystrophin abnormalities and are there occasions when other techniques should be considered instead?

Although our study[1] was not specifically designed to address this particular point, I have reached my conclusion on the basis of the following observations:

A) Western blot *qualitative and quantitative* abnormalities: these were observed in the great majority of the patients we have analysed (90%), including the ones with very mild or preclinical Becker muscular dystrophy (BMD) phenotypes. In particular, two preclinical BMD patients showed a slightly reduced molecular weight protein of slightly lower abundance on Western blot analysis. Immunocytochemical analysis with multiple anti-dystrophin antibodies was judged to be abnormal before the know–ledge of the immunoblot result.

B) Western blot *quantitative* abnormality: this was observed in patient BMD 17,[1] a boy still asymptomatic at the age of 10. There was also discordance in staining between the various anti-dystrophin antibodies.

It thus appears that while most BMD patients have an abnormal molecular weight dystrophin on Western blot, a few (in the very mild end of the spectrum of dystrophinopathies) will only have a reduced amount of a normal molecular weight protein. We have been able to pick up immunocytochemical abnormalities in these patients, but as concluded in our paper, a normal result with immunocytochemistry should not stop one from doing a Western blot, if a dystrophinopathy is still suspected. On the other hand, precise quantitation of dystrophin present on a Western blot can be rather difficult, especially in cases where it is close to normal. Few laboratories around the world routinely carry out dystrophin quantitation corrected for the myosin content of the muscle sample.[2]

Western blot analysis is a technically demanding, expensive and time consuming technique (taking a minimum of two and a half days). Its use cannot therefore be easily justified for individual samples. On the other hand, immunocytochemical analysis with a panel of anti-dystrophin antibodies can be done on individual samples in a couple of hours by a technician with routine histochemical skills.

Another important piece of information that can be derived from immunocytochemical analysis relates to detecting the location of genomic or transcription abnormalities.