We thank Dr Sheila for her expertise in performing the MDMA assay.

It is evident that this patient had a pre-existing 'berry' aneurysm. After ingestion of MDMA, however, it is possible that there was an acute sympathetically mediated surge in blood pressure which caused the aneurysm to rupture. It is also possible that due to repeated use of the drug recurrent surges in blood pressure led to progressive weakening of the vessel wall resulting in aneurysm instability. As noted previously this patient was a regular user of MDMA over a two to three year period before presentation. Such a mechanism has previously been suggested in cocaine abuse which has a recognised association with subarachnoid haemorrhage. Although current usage of MDMA is generally associated with the 'rave scene' it is of note that in the patient we describe, no strenuous physical activity or sexual intercourse was associated with the onset of symptoms.

This case emphasises the importance of taking a full drug history including the possible abuse of illicit drugs in patients presenting with subarachnoid haemorrhage. Subarachnoid haemorrhage may therefore be yet another serious adverse effect of the abuse of MDMA and adds to the increasing number of reports of serious morbidity and mortality associated with this drug.

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Opsioclonus showing only during eye closure in hereditary cerebellar ataxia

Opsioclonus is an abnormal eye movement characterised by a burst of saccades in all directions without an intersaccadic interval. Opsioclonus only during eye closure, and opsioclonus in hereditary cerebellar ataxia, as in our case, have not been previously described.

A 34 year old man complained of gait disturbance and difficulty with fine finger movements. At the age of 28 he gradually noted a deterioration in his baseball playing skills. At the age of 30 he began to have difficulty in walking down stairs, and has since had to use a hand rail. He also experienced difficulty with handwriting and manipulating small objects, and when watching baseball he frequently lost sight of the ball. He had no history of alcohol abuse. On admission on 12 March 1990, physical examination revealed a scaphoid skull, high arched palate and small auricles. Neurological examination showed that he was mentally alert and cooperative. The pupils were equal, round and reacted to light. He had no limitation of eye movement, but there was a horizontal fine ocular oscillation during fixation. When he closed his eyes abnormal ocular movements were noted together with an upward deviation of the eyes (Bell's phenomenon). The eyes showed frequent irregular rapid conjugate oscillations in all directions. He had saccadic pursuit eye movement and ocular dysmetria. He had moderate to severe limb and gait ataxia, and ataxic dysarthria. There was no muscle atrophy or weakness. Tendon reflexes were normal and the plantar response was flexor. There were no involuntary movements, and no sensory or autonomic disturbances.

Routine laboratory examinations for blood and urine showed nothing abnormal. Chemical analysis revealed a two year old 'Ecstasy' and MDMA, however, existing 'berry' (Flamm 1986). Brain CT and MRI showed only that the left parieto-occipital area and upper hemisphere. EEG showed frontal dominant 7 waves during hyperventilation. Electro-oculographic findings were as follows: square-wave jerks were noted during visual fixation, and its amplitude and frequency were increased in darkness. On closing the eyes irregular sharp waves without an intersaccadic interval appeared horizontally as well as vertically (fig). These spontaneous abnormal eye movements persisted during the voluntary eye closure, decreased in drowsy state, and disappeared during sleep. Smooth eye tracking test revealed saccadic pursuit which overlapped the square wave jerks. Saccadic latencies and speed were normal. Optokinetic pattern test showed poor increase of slow phase of nystagmus in both directions. Caloric test showed normal vestibular response with normal visual suppression.

A 29 year old man, a brother of the patient we have described, had no subjective complaints and no history of alcohol abuse. He was mentally alert and the pupils were equally round and reacted to light. There was no limitation of eye movement and convergence was normal. Although there was no nystagmus, horizontal fine ocular oscillation was noted during fixation. On closing the eyes, abnormal irregular rapid conjugate oscillations in all directions were seen as in his brother. He had saccadic eye movement and ocular dysmetria. His speech was slightly dysarthric and.

Figure 2 Digital subtraction carotid angiogram illustrating the left posterior communicating artery aneurysm.

Figure Electro-oculography of the first case: showing opsioclonus only after eye closure.
he had mild limb and gait ataxia. There was no muscle atrophy nor weakness, and tendon reflexes were normal, the plantar responses were flexor. There were no other neurological abnormalities, no sensory nor autonomic disturbances. Electro-oculographic findings were almost identical to that of his brother. The main clinical feature of our patient was adult onset slowly progressive cerebel-
lar ataxia, thought to be hereditary as there were no apparent causes including metabolic diseases. The most conspicuous finding of our cases was an abnormal ocular movement during eye closure. It was a rapid irregular multidirectional oscillation with no intersaccadic intervals, and appeared to fulfill the criteria of opsoclonus. To our knowledge, the appearance of opsoclonus, only during eye closure, has not been previously described. Opsoclonus can be caused by an abnormal activity of bursts cells and pause cells in the brainstem sac-
cadic pulse generator.3 A case of opsoclonus described by Hain et al17 was a case with neurodegenerative disease showing bursts of ocular flutter during blinks, and attributed it to a blink-related discharge of pause cells. As ocular flutter and opsoclonus form a continuum, frequent aggrava-tion of opsoclonus at attempted fixation5 seem to be due to a similar neural dysfunction. Opsoclonus only during eye closure could be explained by the presence of eye-closure related abnormality of sac-
cadic pulse generator, and increased opso-
clonus during eye closure seem to support this speculation.

Square wave jerks, saccadic dysmetria and saccadic pursuit seen in our cases seem to be common ocular motor abnormalities in hereditary cerebellar ataxia.1 Opsoclonus, however, has not been clearly described in hereditary cerebellar ataxia the absence of which was even thought to be a characteristic finding.3 Although opsoclonus often occurs with cerebellar ataxia, absence of opsoclonus in hereditary cerebellar ataxia is noteworthy. Most cases of opsoclonus are of acute or subacute onset, therefore a slowly progressive disease like a hereditary cerebellar ataxia may not usually cause opsoclonus, although there are exceptions, as in our cases.

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Preferential impairment of slow alternating movements in patients with mild cerebellar ataxia

Clinically, it is well known that patients with cerebellar lesions have difficulty pro-
ducing rapid movements. This has been confirmed quantitatively for fast, so-called 'ballistic' movements5 and is often apparent when the patient tacks, hits, or maintains postures.

However, slow movements are also impaired in cerebellar patients. We have recently shown, for example, that the time course of slow, accurate step-tracking movements in mildly ataxic patients is prefer-
entially impaired compared with move-
ments of the same amplitude but made at higher speeds.6 The performance of slow, pursuit movements has also been shown to be impaired both in cerebellar patients7 and following dente cooling in monkeys.8 Clinically, slow alternating movements are not usually tested, probably due to the assumption that one could expect more impairment at higher than at lower frequenc-
ies of voluntary alternating movements.

That this assumption is misleading will be demonstrated by the following study.

Using previously established selection cri-
Figure 1 Velocity records associated with 30° alternating elbow movements. Records on the left were obtained from a control subject, those on the right from a patient with chronic cerebellar degeneration of approximately 3 years. Movements made at maximum frequency are shown in the upper set, middle and lower sets correspond to target frequencies of 1-0 and 0-75 Hz respectively. (1-0 and 0-75 Hz records have been plotted at a higher gain.)

Figure 2 Effects of removing visual feedback on low frequency alternating movements. Individual records of velocity and acceleration are shown for a control subject and 2 cerebellar patients (CB1, CB2) performing 30° alternating elbow movements under "eyes open" and "eyes closed" conditions. Movements made at 0-5 Hz are shown on the left, 0-75 Hz movements on the right. Acceleration was obtained from 3 point digital differentiation of the velocity signal. Velocity and acceleration gains have been arbitrarily adjusted. Duration of each trial was 24 s.
Opsoclonus showing only during eye closure in hereditary cerebellar ataxia.

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