double-stranded DNA antibodies prompted speculation as to whether such antibodies, or other autoantibodies described in scleroderma, might be found in patients with "idiopathic" HFA.

It has been suggested that lesions to the ipsilateral cervical sympathetic innervation, whether peripheral or central, may cause HFA. Moss and Crikelair reported a rat model of progressive HFA following cervical sympathectomy. However, autonomic function testing in patients with HFA has produced conflicting results: although some patients have clear evidence of concomitant autonomic dysfunction (for example, ipsilateral Horner's syndrome) others give equivocal responses to standard tests of autonomic function. Nonetheless, Archambault and Fromm came to the conclusion that the sympathetic theory alone could explain most cases of HFA, even though no thorough evidence of autonomic dysfunction, a position which seems less tenable today. Only indefinite pathological changes have been found in the cervical sympathetic ganglia in the few patients examined thoroughly at post mortem. The evidence of autonomic dysfunction was found in our patient.

Facial trauma is a recognised antecedent of HFA (in up to a third of cases) but the mechanism(s) by which it might produce this condition remains to be clarified. Much remains to be learned of the aetiology of this condition, which probably should not be regarded as a uniform syndrome. Its clinical heterogeneity probably reflects an underlying aetiological heterogeneity. Some cases probably reflect a developmental abnormality, possibly with a genetic basis. Other cases may form part of the clinical spectrum of scleroderma, and others still may result from involvement of the cervical sympathetic chain. The suggestion has also been made that cases with prominent neurological complications may result from a slow viral infection.

We thank Dr R C Hughes for permission to report his patient.

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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 had been 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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Opsoclonus showing only during eye closure in hereditary cerebellar ataxia

Opsoclonus is an abnormal eye movement characterised by a burst of saccades in all directions without an intersaccadic interval. Opsoclonus only during eye closure, and opsoclonus 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 dystarhria. 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 nor autonomic disturbances.

Routine laboratory examinations for blood and urine were normal, as were thyroid function and lysosomal enzyme assays. Peripheral nerve conduction studies and needle electromyography of limb muscles were also normal. Brain CT and MRI showed diffuse atrophy of the thalamus and upper hemisphere. EEG showed frontal dominant theta 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 was normal and showed no 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 his 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 pursuit eye movement and ocular dysmetria. His speech was slightly dysarthric and