Letters

Ping Pong gaze in reversible coma due to overdose of monoamine oxidase inhibitor

Sir: The term Ping Pong gaze was first used by Senelick in 1976[1] to describe the periodic alternating gaze of an unconscious patient. There are now six cases reported in the literature[1-6] which manifested this disorder of rhythmic and pendular conjugate horizontal deviation, which alternates from one extreme of gaze to the other, with a cycle time of between three and seven seconds duration. Five of the six cases described have been in coma. These cases have all had structural brain damage, generally affecting both hemispheres and have all died. In contrast, the patient reported by von Cramon and Zihl,[4] who had suffered multiple bilateral infarcts and showed periodic alternating gaze for several months, retained consciousness.

We report a case of a similar periodic alternating gaze induced by deliberate overdose of tranylcypromine which resolved spontaneously leaving no lasting impairment.

A 55 year old man was admitted having taken an overdose of 500 mg tranylcypromine. In the past he had been treated with this drug for endogenous depression, but had not been an any regular treatment for six months. His family, who were unaware of the overdose, described a gradual onset of drowsiness over a period of hours, accompanied by sweating and generalised shaking.

On admission he was unconscious with no response to speech and making non purposive withdrawal responses to painful stimuli. The most striking finding was that his eyes were spontaneously open and exhibited periodic alternating gaze. They moved conjugately and smoothly from one extreme of gaze to the other, returning without a pause, in a cycle lasting 3–4 seconds. There was no concomitant head movement and no response to the doll's head manoeuvre. Pupillary responses were normal as was the remainder of the neurological examination. Cardiac and respiratory function was unimpaired, the patient was normotensive.

The eye movements persisted continuously for about twelve hours, after which they gave way to intermittent rapid horizontal pendular nystagmus.

Investigations did not reveal a cause. CT head scan and lumbar puncture were normal. EEG was abnormal with widespread rhythmic runs of slow activity. After 48 hours consciousness was regained and the patient confirmed clinical suspicions by admitting to the overdose. At this stage eye movements became normal.

This patient showed eye movement abnormalities very similar to those cases reported previously, the greatest similarity being with Senelick's case in which the eyes were spontaneously open and the ocular deviation reached both extremes of gaze. This is in contrast with some of the other cases where the eyes were closed,[5] or where the deviation was from one extreme of gaze to just beyond the mid line.[5,6] Most authors have felt that Ping Pong gaze is related to bilateral cerebral hemisphere dysfunction and their autopsy results support this. Bilateral cerebral infarction has been present in all cases except Senelick's, where the lesion was a mid line posterior fossa haemorrhage. Previously Ping Pong gaze when seen in an unconscious patient was taken as indicating structural and irreversible brain damage. The case reported here demonstrates that Ping Pong gaze may be seen in coma and yet be associated with complete recovery.

Ping Pong gaze has not been reported as a consequence of drug therapy or toxicity. There are no reports either of monoamine oxidase inhibitor overdose producing specific abnormalities of eye movement, although Baloh *et al*[7] reported a case of smaller amplitude macrosaccadic oscillations in a fully conscious patient resulting from a single dose of L-triptophan in a patient pre-dosed with tranylcypromine. They postulate increased inhibition by serotonergic neurons of the median raphe nuclei resonsible for control of saccadic eye movements.

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References

1 Senelick RC. "Ping-Pong" gaze: Periodic alternating gaze deviation. *Neurology* 1976; **26**:532–5.
2 Fisher CM. Some neuro-ophthalmological observations. *J Neurol Neurosurg Psychiatry* 1967;**30**:383–92.
3 Lapresle J, Said G. Deviation forcée des yeux vers le bas et en de dans et mouvements oculaires periodiques au cours d'une hemorragie aneurysmale de la calotte mésencephalique. *Rev Neurol (Paris)* 1977;**33**: 497–503.
4 von Cramon D, Zihl K. Das Phanomen der periodisch alternierenden Bulbus deviation. *Arch Psychiatr Nervenkr* 1977;**224**:247–57.
5 Stewart JD, Kirkham TH, Mathieson G. Periodic alternating gaze. *Neurology* 1979;**29**:222–4.
6 Nasucci EF, Fabara JA, Saini N, Kurtzke J. Periodic alternating Ping-Pong gaze. *Ann Ophthalmol* 1981;**13**:1123–7.
7 Baloh RW, Dietl J, Spooner JW. Myoclonus and ocular oscillations induced by L-Tryptophan. *Ann Neurol* 1982;**11**:95–7.

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Autonomic neuropathy in systemic lupus erythematosus

Sir: Acute autonomic neuropathy has been described in association with autoimmune diseases,[1-3] but has been rarely reported as a complication of systemic lupus erythematosus (SLE).[4]

A 72 year old woman with systemic lupus erythematosus diagnosed 2 years previously,[5] who had suffered other CNS complications of SLE (visual and auditory hallucinations, and an ischaemic stroke with good recovery) developed painful acute urinary retention after 6 days of dysuria. She could no longer void despite the use of the Crédé manoeuvre and required catheterisation. She was given prednisone 10 mg qd and diazepam 10 mg qd. At the time of admission she had xerostomia, xerophthalmia and intestinal constipation. Examination showed normal sized symmetric pupils that reacted to light and constricted poorly with accommodation. There was no weakness, profound reflexes in the upper/lower members were present and symmetric and all modalities of sensation were normal. There were no orthostatic changes in blood pressure and heart rate. The Schirmer test was abnormal in both eyes. Electrocardiogram showed no R-R interval variability at rest, during deep breathing, during Valsalva manoeuvre and after standing.[6] Complete blood count, electrolytes, blood urea nitrogen, creatinine, glucose, hepatic transaminases, CSF examination, including protein electrophoresis, were all normal. Hepatitis B surface antigen was negative. Erythrocyte sedimentation rate (Westergren) was 45 mm/h. Rheumatoid factor (Waaler-Rose), anti SS-A antibody, anti SS-B antibody, anti RNP antibody, anti Sm antibody, and anti ds-DNA antibody (Chritidia lucillae) were

negative. Antinuclear antibodies (1:128) revealed a diffuse homogeneous pattern and LE cells preparations were positive. The titre of serum complement (C3) and IgG were normal. Electromyography and nerve conduction studies of the upper/lower limbs were normal. Urodynamic assessment was carried out by cystometry at a filling rate of 50 ml/min. Intravesicular pressure during filling did not exceed 15 cmH20. Bladder capacity was 420 ml. Although the patient had normal sensation of bladder distension, she could not initiate micturition even with the use of the Crédé manouevre. A coaxial needle inserted percutaneously into the striated muscle of the anal sphincter showed a continuous hyperactivity, without relaxation during the effort of voluntary micturition, denoting a detrusor-sphincteral dyssynergia. A trial with prednisone 80 mg qd and pyridostigmine 30 mg tid for 2 weeks was unsuccessful. Prednisone was lowered to 20 mg at alternating days. She was maintained with vesical catheterisation and laxatives. After 5 weeks her condition slowly improved and she could void with the use of suprapubic manual compression. We reviewed the patient 4 months later and she was well, but dryness of mouth and eyes and intestinal constipation remained unchanged.

We could find only one report of a previous case of acute autonomic neuropathy in association with SLE,[4] in a 21 year-old-female, who showed a rapid clinical response to steroids, which was not observed in our patient. The pathogenesis of this condition is unknown, although both humoral and cell-mediated immune mechanism may be implicated.[7] In some respects this condition resembles experimental autonomic neuropathy.[8] A prospective study of the prevalence of autonomic dysfunction in SLE patients is suggested.

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References

1 Edelman J, Gubbay SS, Zilko PJ. Acute pandysautonomia due to mixed connective tissue disease. *Aust N Z J Med* 1981;**11**:68–70.
2 Guddesblatt M, Goodman AD, Rubenstein AE, Bender AN, Choi H-SH. Automonic neuropathy associated with autoimmune disease. *Neurology* 1985;**35**:261–64.
3 Takayama H, Kazahaya Y, Kashihara N, *et al.* A case of postganglionic cholinergic dysautonomia. *J Neurol Neurosurg Psychiatry* 1987;**50**:915–8.
4 Hoyle C, Ewing DJ, Parker AC. Acute autonomic neuropathy in association with systemic lupus erythematosus. *Ann Rheum Dis* 1985;**44**:420–4.
5 Tan EN, Cohen AS, Fries JF, Masi AAT, McShane DJ, Rothfield NF, Schaller JG, Talal N, Winchester RJ. The 1982 revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum* 1982; **25**:1271–7.
6 Ewing DJ, Clarke BF. Diagnosis and management of diabetic autonomic neuropathy. *Br Med J* 1982;**285**:916–8.
7 Morgan SH, Kennett RP, Dudley C, Mackworth-Young C, Hull R, Hughes GRV. Acute polyradiculoneuropathy complicating systemic lupus erythematosus. *Postgrad Med J* 1986;**62**:291–4.
8 Appenzeller O, Arnason BG, Adams RD. Experimental autonomic neuropathy: and immunologically induced disorder of the reflex vasomotor function. *J Neurol Neurosurg Psychiatry* 1965;**28**:510–5.

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Korsakoff's psychosis in the presence of multiple sclerosis: an unusual cognitive state

Sir: The typical Korsakoff's psychosis, an amnesic syndrome following the acute confusional state of Wernicke's encephalopathy, is well known and easy to recognise in chronic alcoholics. This classical picture is however uncommon, and in practice the clinician is often confronted with patients in whom memory deficits of variable severity are accompanied by other cognitive abnormalities and in whom the history of Wernicke's encephalopathy is often missing. In such cases it is tempting to explain the symptoms as a result of dual pathology caused by thiamine deficiency and by the direct toxic effects of alcohol on the brain. The subacute onset, often seen in such cases, could be interpreted as the result of the overlapping effects of these different lesions, which need to reach a certain threshold to manifest clinically. This explanation, likely to be accurate in most cases, carries the danger of preventing clinicians from looking for other aetiological factors which could be responsible for these atypical combinations of symptoms.

We describe here a patient in whom multiple sclerosis may have contributed to an atypical picture of Korsakoff's psychosis. The patient was a 50 year old, twice divorced man who had been admitted for further assessment. He had been referred initially to his local psychiatric hospital by the social services as they had become concerned by his rapid social decline and seeming indifference to it. Prior to his admission the patient had been unemployed and at times living as a vagrant.

Background information was limited by the lack of informants and the patient's poor memory. His early development was seemingly normal and he had left school with 3 A levels to study engineering at London University. After completing his training he had worked for a number of large engineering companies, but had lost his job on frequent occasions because of drink. His two marriages had failed for the same reason and he had lost contact with his 2 children from his first marriage. He admitted to drinking over half a bottle of spirits a day for several years and was able to recall an admission to a private clinic for detoxification some years previously. His mother and one of his half-brothers were said to have abused alcohol. His previous physical health appeared to have been good except for a transient period of double vision accompanied by an unusual sensation in his feet, "as though walking on cobblestones" which had taken place perhaps two years previously while working in Bahrain. On his return to the UK he remembered being admitted to hospital and receiving ACTH injections with subsequent improvement of his symptoms. The diagnosis of multiple sclerosis was mentioned at the time. He denied further neurological symptoms.

On admission the patient appeared surprised at being in hospital and had few spontaneous complaints. On questioning he admitted to having a poor memory for dates and past personal events. Physical examination revealed a dishevelled, unkempt man who had neglected his personal hygiene. A pale optic disc on the left and an afferent pupillary defect on the same side were the only other signs. There were no stigmata of alcoholic liver disease. At interview he appeared relaxed and cooperative, but rapport was difficult to establish. Answers were rapidly given, inconsistent and often contradictory. He was unconcerned about his problems and denied being depressed. No abnormal beliefs or perceptions were elicited. He was disorientated in time and short term memory was severely impaired both on verbal and non-verbal testing. He did not confabulate spontaneously, but did so occasionally when provoked by questioning, these answers being inaccurate rather