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 1976¹ to describe the periodic alternating gaze of an unconscious patient. There are now six cases reported in the literature¹⁻⁶ 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 on 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 nor-

mal. 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-tryptophan in a patient pre-dosed with tranylcypromine. They postulate increased inhibition by serotonergic neurons of the median raphe nuclei responsible for control of saccadic eye movements.

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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 (Waller-Rose), anti SS-A antibody, anti SS-B antibody, anti RNP antibody, anti Sm antibody, and anti ds-DNA antibody (Chitridia lucillae) were