LETTERS TO THE EDITOR

Fear can interrupt the continuum of memory

I had always thought that the existence of post-traumatic amnesia, characterised by gaps in the patient's memory of events after an accident, particularly if the remaining "snapshots" of memory were particularly vivid, meant that the patient must have been knocked out, albeit briefly. I had always rejected the concept that fear, acute anxiety, panic, that which the layman calls "shock", could be responsible for the phenomena of post-traumatic amnesia. That is until about 6 months ago.

I was driving in the wet, too fast, in a convoy in the outside lane. The conditions were appalling; I was alert. Six or seven cars in front of me were involved in a "front to rear shunt", precipitated by a car that had been stuck on the inside lane behind a slow moving vehicle, darting without warning into the outside lane.

I remember seeing this car do this, I have a vivid image of seeing a car, perhaps two away from me, suddenly appearing at right angles—I could see its rear end to the left of, and the front end to the right of the car in front of me. I have no recollection of putting my brakes on, and then I have a very clear image of the car in front of me coming towards me as I skidded. This was a crystal clear isolated image, associated with an absolute conviction that I was about to die—the car was a small red Honda saloon, the front passenger was wearing a flowery hat, and there was a black and white soft toy dangling in the rear window. I have no recollection of avoiding this car, or steering or skidding to the offside, I have no recollections until that of my car slithering to a bumpy halt on the grass verge at the central reservation of the road. I remember thinking that the antiskid mechanisms had not worked. My next recollection is of looking down through my opened door (without any memory of opening it) at the thick mud of the central reservation and thinking that I would have to clean my shoes after walking on it. At that point clear and continual memory is restored and I went to see if I could assist the people in the other cars, and eventually I drove off.

As I continued on I ruminated on events, and realised that there was no doubt about it—there were significant blanks in my memory of events, and that furthermore those memories I did have were extremely vivid—the hat, the dangling toy, the sure fire knowledge that I was about to die, and the light brown slimy mud on the central reservation.

There is no question at all of my having hit my head, or being subjected to any violent to and fro acceleration or deceleration forces—this was peritraumatic amnesia in which the trauma was wholly psychological.

I am grateful to my anonymous referees for drawing my attention to two reviews—by van der Kolk and Fislér¹ and by Kopelman²—on the subject of psychological trauma and amnesia. Neither papers describe precisely what I am describing and neither accentuate what to me is a most remarkable phenom-

enon and that is the very vivid nature of these memories. Now, 6 months later, some of the memories are beginning to fade, but interestingly it is not the conviction of impending death that persists but it is the image of the mud. This vivid nature of post-traumatic amnesic memories was described by Russell and Nathan.³ I think that this is what differentiates my experience from a dissociative state—although I am obviously open to correction on this.

In answer to the inevitable question from my fellow medicolegal report writers—no I did not develop any of the features of "post-traumatic stress disorder".

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Folate in CSF and age

Low serum and red cell folate concentrations have been reported in up to 42% of elderly healthy subjects in the community or acute geriatric medical admissions, and in 20%–82% of psychogeriatric admissions.¹ Although widely attributed to dietary causes, we have long reported a causal link between folate deficiency, depression, or dementia.¹⁻³ Recent community studies have suggested a significant rise in serum total homocysteine and fall in serum folate with age⁴; and there has been renewed interest in a link between Alzheimer's disease, depression, folate deficiency, and raised serum total homocysteine.^{5,6} Other studies suggest that high homocysteine and low folate concentrations are independent risk factors for premature occlusive vascular disease, including cerebrovascular disease.^{4,5}

Remarkably, folate in the form of methyl folate is concentrated in CSF by an active transport process at concentrations about three times that in serum.³ We have had the opportunity to study CSF folate in a group of elderly patients undergoing spinal anaesthesia.

Folate in CSF was measured in 41 patients undergoing surgery at Northwick Park Hospital in a study of spinal anaesthesia and postoperative spinal analgesia by the Department of Anaesthetics. The operations were hip replacement (22), knee replacement (four), hernia (eight), haemorrhoids (three), and varicose veins (four). There were 17 men and 24 women, mean age (SD) 74.6 (13.4) years. The patients were otherwise healthy and were not taking medication other than analgesics before surgery.

With informed consent, 2 ml CSF were withdrawn at L3/4 in the fasting recumbent position under local anaesthetic before spinal anaesthesia, and was immediately stored at -70°C. Folate in CSF was measured by microbiological assay using a chloramphenicol resistant strain of *L. casei*.

The figure shows a highly significant decline in CSF folate with age. The mean CSF folate (28 (SD 7.4) µg/ml) between ages 40 and 59 fell 5% between age 60 and 69 (26.6 (SD 8.1) µg/ml). The greatest fall was

Relation of age to CSF folate concentrations in elderly surgical patients.

after the age of 70, with a 27% fall between 70 and 79 (20.3 (SD 5.8) µg/ml) and a 54% fall between 80 and 99 (13.0 (SD 5.4) µg/ml). (analysis of variance (ANOVA) $F=11.73$, $p<0.001$). The correlation between age and CSF folate was -0.72 ($p<0.001$).

Our findings provide further evidence of a link between folate concentrations and age and, for the first time, directly in the nervous system. Although we did not measure serum folate concentrations, others have already reported a fall in serum folate and rise in plasma homocysteine with age.⁴ Our subjects were not anaemic or macrocytic and were not known to have medical or psychiatric disorders. It is unlikely, therefore, that the significant decline in CSF folate can be attributable simply to dietary deficiency. Other possibilities include a gradual failure with aging of the active transport mechanism which carries methyl folate into the nervous system.³

Whether due to aging or acquired deficiency, there is evidence causally relating folate status to mental function, especially depression and dementia.^{1-3,5,6} Controlled and uncontrolled clinical trials suggest a specific effect of folate on mood and cognitive function.¹⁻³ The effects of folate on mental function are mainly mediated through numerous methylation pathways in the nervous system and are similar to the effects of S-adenosylmethionine (SAM), the major methyl donor in the brain which obtains its methyl group mainly from methyl folate.¹

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Anterior superior alveolar neuropathy: an occupational neuropathy of the embouchure

A 31 year old French hornist was referred for evaluation of a 9 month history of pain in her right upper lip.

A member of a major symphonic orchestra, she regularly played 4-6 hours a day. Her symptoms began after a heavy period of playing, with a sensation of excess pressure in the right upper lip followed shortly thereafter by frank pain in this area. She spontaneously distinguished between two types of pain. The first was a constant dull ache, extending from the midline 1 cm laterally onto the vermilion lip. She noticed that brushing her teeth or eating could trigger pain in this region. Her second pain sensation was different from the first—electric, lancinating pain originating in the medial portion of the right lip and extending to her right nostril. There were no paraesthesias of the lips, cheeks, or jaw, and she had no symptoms of facial weakness.

She compensated for her difficulties and maintained her usual high level of professional performance. Briefs periods of rest produced temporary improvement; however, her symptoms recurred when she resumed performing. There were no other complaints and her family history and medical history were unremarkable.

Except for her right lip, neurological examination was normal including careful sensory evaluation of the anterior teeth, gums, and palate. Her right first incisor was noted to protrude slightly in front of her left. As shown in the figure, light touch sensibility was reduced in the right lip (wide cross hatching). A smaller region (narrow cross hatching) was painful to touch, lying directly over her prominent first incisor and below the contact point for the circular mouthpiece of the horn (fine stippling). Application of viscous lidocaine to the lip temporarily relieved her symptoms. An MRI of the head was obtained, showing no abnormalities along the path of the trigeminal nerve. A brief course of gabapentin at 900 mg/day was ineffective, and she declined further drug treatment. Filing down her prominent right first incisor in combination with 6 weeks of complete rest produced marked improvement in her symptoms. Her sensory loss improved as well, and she was able to return to work.

Musicians who play brass instruments (trumpet, French horn, trombone, and tuba) spend their professional lives perfecting control of the embouchure, the muscles of the lower face, lips, and tongue that control the direction and intensity of airflow into the mouthpiece. The lips and face of a brass

player experience tremendous pressure from the mouthpiece, sometimes sufficient to cause muscle strains or even traumatic tears in the orbicularis oris.¹

This patient's sensory disturbance localised to the anterior superior alveolar nerve. The superior alveolar nerve divides into a posterior, middle, and anterior group. Branches from these divisions form a plexus that innervates the dental pulp, periodontal membrane, adjacent labial and buccal gingiva, and the upper teeth. The posterior, middle, and anterior branches innervate the molar, premolar, and incisor or canine teeth, respectively. Only the anterior superior alveolar nerve travels with the infraorbital nerve to exit through the infraorbital foramen.² In our patient, her prominent right first incisor predisposed a terminal branch of this nerve to focal entrapment by the overlying mouthpiece rim.

Injuries related to occupation are increasingly recognised among performing artists. Focal entrapment neuropathies of the arm have been well described in musicians. This patient shows that similar occupational neuropathies may affect the lips and face. We are aware of two other reports of patients similar to ours.^{3,4} Given that the force exerted on the lips by the mouthpiece of a French horn has been measured to be as high as 50 N, with displacements of the central incisors of up to 180 µm during playing, it is not surprising that these neuropathies occur.⁵

French horn and trumpet mouthpieces are significantly smaller than those of the trombone and tuba, and so mouthpiece forces are concentrated onto a very small lip surface area. We suspect that there are other French horn and trumpet players who have experienced similar symptoms to our patient.

Entrapment neuropathy is a potentially reversible cause of disability in brass players, and should be considered in the differential diagnosis of embouchure dysfunction.

I thank Drs Jonathan Aviv, and Michael Gelb, for their assistance in caring for the patient.

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Clinical and MRI discordance in a case of delayed radiation myelopathy

Delayed radiation myelopathy (DRM) is a rare complication of radiotherapy, especially when the total dose delivered to the spinal cord is less than 50 Gy.¹ From the limited data reported on the radiological features of DRM, typical MRI changes of cord swelling and gadolinium enhancement on T1 weighted images and increased intramedullary signal on T2 weighted images often correlate well with progression of neurological

symptoms. We report a case of DRM in which the clinical features remained static at a level corresponding only to the lower end of the abnormal intramedullary lesion seen on MRI, without radiological evidence of blood-brain barrier breakdown, suggesting that pathological changes to the spinal cord after radiation are not always displayed as visible changes on MRI.

A previously well 71 year old woman presented in December 1997 to the ear, nose and throat department with hoarseness and difficulty swallowing, having discovered a lump in the right side of her neck. Laryngoscopy showed a right pyriform fossa tumour, histologically confirmed as a lymphoepithelioma after biopsy. Staging neck CT showed extensive tumour in the right pyriform sinus and lateral pharyngeal wall with associated lymphadenopathy extending to the thoracic inlet.

Between April and May 1998, she received chemotherapy comprising 5-fluorouracil and cis-platinum. Radiotherapy was started in June 1998 with fields covering the pyriform sinus and neck, anterior supraclavicular fossa, and posterior neck. The calculated dose delivered to the cervical spinal cord was 42.5 Gy in 2 Gy equivalent fractions.

Over the next 4 months, she improved clinically and on review in October 1998 there were no signs of tumour recurrence, confirmed on laryngoscopy. She remained well until November 1998 when she began to notice progressive numbness and weakness spreading proximally up both legs over a period of 6 weeks, such that she was unsteady when walking. Soon thereafter, she developed urinary incontinence and constipation. At this time, cervical and thoracic spine MRI (figure) disclosed cervical cord swelling with a spindle shaped cavity of low signal intensity on T1 weighted images and high signal intensity on T2 weighted images extending from C4 to the upper end of T1. There was no cord enhancement after the administration of gadolinium.

On neurological review in January 1999, she was incontinent of urine and faeces and was able to mobilise only with the aid of a walking frame. There was generalised wasting in both legs with mild reduction in strength of hip flexion, knee flexion, and extension, but no detectable weakness in the arms. There was a loss of spinothalamic sensation below the level of T5 bilaterally, with a subjective feeling of altered sensation in both hands.

Over the ensuing 4 months, her level of disability progressed such that she was unable to walk or transfer independently. Further examination disclosed flaccid paralysis of both legs and minor weakness of the intrinsic muscles of both hands. There was reduced perception of pain and temperature sensation below the level of C8. She commenced hyperbaric oxygen therapy given on a daily basis for 3 weeks but there was no symptomatic recovery.

After a recurrence of her original swallowing difficulties, she was found to have a tumour recurrence in her pyriform fossa at biopsy, with associated lymphadenopathy. No further radiotherapy was given. At last review in February 2000, she had no movement in her lower limbs and marked weakness of the intrinsic muscles of both hands, with preserved power in proximal upper limb muscles. Sensation above the level of C8 was intact.

Diagnostic criteria of DRM as outlined by Pallis et al² stated that the spinal cord should

Sensory loss (wide cross hatching), focal pain (narrow cross hatching), and lancinating pain (arrow) are shown overlying a diagram of the lips. Dashed lines indicate the position of the first incisors, and the contact of the french horn mouthpiece with the lips is shown as a stippled circle.