LETTERS TO THE EDITOR

Spasms

It all happened a long, long time ago. I had seen the first case shortly after my arrival in Baghdad. A colonel in his early fifties, he positively glowed in a grey uniform. He was a leading member of the ruling Party and spoke reasonable English, displaying in conversation that sinister mixture of sycophancy and suspicion that I had come to expect of “political” officers in various parts of the Middle East.

His twitch had come on some two years earlier, fairly abruptly, under circumstances he could not or would not recall. At times, it was most distressing, seizing him and compelling him against his wish, to point his third chin in the general direction of his right shoulder. He was most aware of his impotence, and found no solace in the sympathy of those in authority. It afflicted him little in the Staff College to which he was attached. The twitch had disappeared completely during a recent visit to London, where he had felt faintly ridiculous consulting neurol ogists for an involuntary movement he had been unable to show them.

In the hotel, later that evening, I hardly gave the matter a second thought. I was tired, had seen many patients that day, and the colonel had left no deep impression upon me, either as a person or as a problem. Spasmodic torticollis was a rare condition, singularly refractory to treatment. It had never excited my clinical curiosity. Neuropathologists and neuropharmacologists had had little of substance to say concerning its aetiology and my psychiatric colleagues, over the years, had proved quite unable to help those few cases I had guided their way.

The Tigris flowed quietly through the city, its banks a double necklace of delightful delight. The heated houses and gardens of buff and ochre, grey and pink—were enlivened at intervals by nuggets of brilliant green or turquoise: the fronds of palms and the leaves of various hibiscus. But the peace was deceptive. The city had known only a few months of civilian rule since the revolution of 1958. One group of officers after another had held power in the name of varying creeds, all dutifully—if somewhat implausibly—proclaimed to be radical variants of the same belief. Deposed rulers often met bloody ends. The corpse of the hated Nuri Said had been dragged by the feet through the streets of the city, to reassure the multitude that he was totally and irreversibly dead.

The second case had been shown to me about a week after the first. He was a much younger man, twenty-five (if that), lean and tense, and with that periodic glint of fanatism in the eye that proclaimed the true believer. I found it hard to elicit his precise occupation. He had something to do with the Ministry of the Interior. Given the machine gun turrets I had seen rather obviously displayed outside that particular building I probed the point no further.

He had already slowed a long, long way. A long, long way. A long, long way. A long, long way.

Pontine myelinolysis presenting with acute parkinsonism as a sequela of corrected hypothermia

We report an unusual case of pontine myelinolysis associated with severe hypothermia and transient hypotension. The presentation was one of acute parkinsonism, despite the presence of a large pontine lesion on magnetic resonance imaging (MRI).

A 66-year-old woman presented with a "flu-like" illness and was treated with oral erythromycin. Six days later she became confused and drowsy. She was referred to hospital and found to have a chest infection associated with a syndrome of inappropriate antidiuretic hormone secretion (in serum: albumin 33 g/l, sodium 94 mmol/l, potassium 1-6 mmol/l, urea 23 mmol/l, osmolality 305 mosmol/kg, in urine: sodium 45 mmol/l, potassium 35 mmol/l, osmolality 407 mmol/l kg). There was no history of alcohol abuse and she had not received diuretics. She was treated with twice normal saline with added potassium, intravenously, until her serum sodium was 122 mmol/l and her serum potassium was 3-3 mmol/l. She became orientated and cooperative, intravenous fluids were stopped and she was treated with oral fluid replacement. The rate of serum sodium correction was 19 mmol/l in the first 24 hours (6 mmol/l in the first 10 hours and 13 mmol/l in the subsequent 14 hours), 10 mmol/l in the next 24 hours and then an average of 3 mmol/l per day over the next five days.

Twelve days after admission she again became confused and agitated, with paranoid ideas. She had developed impulsive facies, bradykinesia, rest tremor, cogwheel rigidity in the limbs and a parkinsonian gait. No biochemical abnormality was detected and she had not received any neuroleptic drugs. CT scan was normal but MRI showed a large area of pontine myelinolysis (fig). Imaging of the basal ganglia was inadequate due to movement artefact. A small dose of levodopa (Madopar 62-5 mg four times daily) improved her gait and abolished the tremor. The levedopa was withdrawn after two months and she is now neurologically normal and fully independent. During her hospital admission she was found to have mild primary hypothyroidism and was treated with thyroxine.

This case illustrates the dangers of rapid correction of hyponatraemia.1 Hypernatraemia did not occur at any time and the suspicion that myelinolysis will not occur providing hyponatraemia is avoided appears invalid.

Only one previous case of pontine myelinolysis presenting with dopa-responsive park-
insonism has been reported; that patient also had pyramidal signs. A further patient exhibited dopa-responsive tremor and facial impassivity during recovery from a more typical presentation of pontine myelolysis with supratentorial eye movements and tetraparesis. Pathological changes in the basal ganglia have been well documented in typical cases of pontine myelolysis, and it has been suggested that the pontine lesion masks the extra-pontine clinical features. In our case the large lesion in the pons was clinically silent. MRI is clearly the investigation of choice in patients presenting with neurological syndromes associated with hypopontaxemia; subclinical or clinically atypical pontine myelolysis may be more common than is currently realised.

We are grateful to Dr S P Kane for permission to report a patient under his care.

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<th>Cortical cholinergic transfer activities in senile dementia of Lewy body type&lt;sup&gt;a&lt;/sup&gt; mean, SD</th>
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<td>SDLT</td>
<td>10.8, 5.2</td>
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<td>(7)</td>
<td>(11)</td>
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<td>Without hallucinations</td>
<td>5.1, 2.7</td>
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<td>(17)</td>
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<tr>
<td>With hallucinations</td>
<td>1.6, 1.4</td>
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*CHAT appeared as mmol/mg protein<sup>b</sup> in Brodmann areas 39 or 40 (parietal), 21 or 22 (temporal) and 17, 18 or 19 (occipital). Case numbers in parentheses.

<sup>a</sup>Parietal and temporal but not occipital cortex.

Cholinergic therapy (those for neurofibrillary therapy and amenable to treatment with the presence of visual hallucinations. SDLT archicortical pathology of pontine myelinolysis, a further patient under his care. CT scanning and brainstem imaging have been reported; that patient also had profound hyponatremia and was resuscitated and had experienced several serious falls.

The right hip prosthesis had loosened in a recent fall and she was unable to walk. Her eye movements were full and there was no nystagmus in the primary position. There was down beating nystagmus on down gaze and obliquely down beating nystagmus on lateral gaze to either side. Palatal movement and gag reflexes were absent although palyngeal sensation and tongue movements were preserved and speech was normal. Fluid aspiration occurred consistently and she was fed through a nasogastric tube. Her limbs were ataxic, but there were no pyramidal features.

A sleep study demonstrated considerable periodic nocturnal hypoxia (SaO<sub>2</sub> < 80%). MR imaging showed cerebellar hypoplasia and tonsillar herniation down to the level of the second cervical vertebral body (fig). The medulla was elongated and kinked over the odontoid peg. There was mild associated hydrocephalus. Her femoral magna was deformed, the dura incised and a fascial graft was inserted. Although there was no change in the physical signs following operation, she was able to swallow fluids normally without choking, aspirating or regurgitating. The nasogastric tube was successfully removed and she was discharged from hospital.

2 Perry RH, Irving D, Blessed G, Perry EK, Fairbank AF, Cholinergic and neuropatho-

Type 1 Arnold-Chiari malformation in a 77 year old woman

Chiari malformations may present in a variety of ways, most commonly between the ages of 20 and 60 years. Onset of symptoms after the seventh decade is distinctly unusual. A 77 year old woman had fallen and was found unconscious by her general practi-

tioner. She was thought to have aspirated. She stopped breathing before reaching hospita-

l but was successfully resuscitated and needed artificial ventilation for a week. As she recovered, frequent choking and aspiration of fluids were noted. On closer questioning she admitted having dysphagia with nasal regur-

gitation over a two year period. During the preceding six months she had become steady and dizzy, with a tendency to fall, and there had been fleeting episodes of diplopia. Some years before she had had bilateral total hip replacement and had experienced several serious falls.

The right hip prosthesis had loosened in a recent fall and she was unable to walk. Her eye movements were full and there was no nystagmus in the primary position. There was down beating nystagmus on down gaze and obliquely down beating nystagmus on lateral gaze to either side. Palatal movement and gag reflexes were absent although pharyngeal sensation and tongue movements were preserved and speech was normal. Fluid aspiration occurred consistently and she was fed through a nasogastric tube. Her limbs were ataxic, but there were no pyramidal features.

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J Neurol Neurosurg Psychiatry 1990 53: 87-88
doi: 10.1136/jnnp.53.1.87-a