course of Parkinson's disease.⁶ In favour of this viewpoint is the finding that MPTP

exposed parkinsonian patients had severe loss of dopaminergic neurons and developed dyskinesias rapidly after starting levodopa therapy.⁷ Caveats about this conclusion must be placed, relative to the fact that both patients had prominently akinetic disturbances, and thus prevalent tremorogenic parkinsonisms might have different courses with different occurrences of complications during levodopa treatment.

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Reversible hydromyelia in a synchronised swimmer with recurrent thoracic girdle pains

Synchronised swimming is considered a low injury competitive aquatic sport for all ages, although stress related symptoms such as knee or shoulder pain are common.¹ We report a case of recurrent thoracic girdle pains in a professional instructor of this sport due to reversible hydromyelia.

The patient was a 40 year old woman with 15 years experience as an instructor of synchronised swimming. She was in good health up to 15 December 1996 when she developed insidious left thoracic pains 2 days after an underwater exhibition performance that was longer and more strenuous than usual. The pains spontaneously disappeared over the next 10 days. On 6 February 1997, she again experienced similar thoracic girdle pains 2 days after prolonged lessons and a week later came to our hospital. The pain was dull and increased intermittently, in particular when she turned over in bed at night. On examination she was alert, afebrile, and normotensive. Neurological examination disclosed no abnormalities in her cranial nerve, motor, sensory, and autonomic functions. There was no nuchal rigidity. Routine laboratory findings for blood, urine and CSF were

Thoracic MRI showing areas of abnormal signal intensity in the central portion of the spinal cord at the T4-5 level. (A) sagittal, T2 weighted; (B) sagittal, T1 weighted; and (C) axial, T1 weighted. (D) T1 weighted cervical MRI showing a Chiari malformation type I.

all normal. Bleeding and whole blood clotting times were normal. Tests for oligoclonal bands and myelin basic proteins of CSF were negative. Thoracic cord MRI detected areas of abnormal signal intensity (high in the T2 weighted and low in the T1 weighted images with no enhancement by Gd-DTPA) in the central portion of the spinal cord at the T4-5 level (figure A, B, C). The intensities were linear-elliptic in the sagittal plane, and small and round in the axial plane, indicative of hydromyelia (central canal dilatation). Myelography and MRI of the cervical and lumbar cord and of the brain showed no abnormalities except for a Chiari malformation type I (figure D). Only a recommendation not to strain or hold her breath was given, and the pains resolved spontaneously over the next 10 days. Two months later follow up MRI of the thoracic cord showed the absence of the initial abnormal finding.

Synchronised swimming requires flexibility, kinesthetic awareness, and aerobic conditioning. Few acute injuries occur in the participation of this sport, but overuse injuries such as knee pain associated with the eggbeater kick and shoulder pain associated with sculling are becoming more common.¹ Therefore the thoracic pain in our patient might have been wrongly diagnosed as having a musculoskeletal origin.

The combination of breath holding and the performance of compulsory figures, such as those that involve hyperextension of the spine, can markedly raise intrathoracic and intracranial pressures during prolonged underwater performances, and may cause dangerous

hydromyelia (central canal dilatation) due to changes in the CSF dynamics, especially in those who have a Chiari malformation.² We have no idea why this hydromyelia developed at the upper thoracic level.

We conclude that synchronised swimming rules should not encourage prolonged underwater performances and unnatural compulsory figures, and that prior checks for risk factors such as a Chiari malformation should be made.

The lumbar puncture, myelography and follow up MRI were done at the Department of Neurology, the National Institute of Neuroscience Kohnodai Hospital, Ichikawa, Chiba. We thank Dr Takeshi Sato and his colleagues for their expert help.

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Barium carbonate intoxication: an electrophysiological study

Barium carbonate is an uncommon poisoning agent in India. This whitish coloured powder is available over the shelf from the

chemists and is used mainly as a rodenticide (rat poison). We report an unusual case of barium carbonate poisoning. The nerve and muscle electrophysiological studies are reported for the first time in barium carbonate intoxication.

A 19 year old boy presented to a hospital emergency department with sudden onset tetraplegia. He was referred to our centre as a case of Guillain-Barré syndrome. The patient presented with tingling in the right upper arm, which was immediately followed by weakness in all four limbs. The weakness progressed and involved trunk muscles also within a few minutes. There was no history suggestive of bulbar or facial nerve involvement.

On examination, higher mental functions, cranial nerves, and sensory and cerebellar examination were normal. Examination of the motor system showed a power of 0/5 globally. No deep tendon reflex could be elicited. Surprisingly, with complete areflexic paralysis there was increased tone in the lower limbs. A diagnosis of Guillain-Barré syndrome was considered, but there were some pointers against this diagnosis. The onset and progression to complete tetraplegia was very rapid and the weakness started from the upper limbs followed by that of the lower limbs. Increased tone in the lower limbs was also not consistent with the diagnosis of Guillain-Barré syndrome.

Routine blood investigations at the time of admission, including serum electrolytes, were normal. Nerve conduction study showed normal distal latency of the compound muscle action potential and motor nerve conduction. Amplitude of the compound muscle action potential was low on proximal and distal stimulation. No conduction block, dispersion of compound muscle action potential, or F wave response was recordable. Sensory conduction studies on median and ulnar nerves at both sides of the body were normal for distal latency and amplitude of sensory nerve action potential. No H reflex was recordable on stimulation of posterior tibial nerves on both sides. The patient now disclosed that he had accidentally consumed barium carbonate (15 g) on the day before admission. He ingested barium salt by mistaking it for dieting food given to him by a slimming centre. Ingestion was followed immediately by profuse vomiting. One hour later, the patient had watery diarrhoea. Four hours later he was taken to hospital where his stomach was washed out with magnesium sulphate solution. Blood investigations repeated 8 hours after admission showed normal blood counts, blood gas analysis, and renal and liver function tests. Serum potassium was 2.8 mmol/l and sodium was 140 mmol/l. An ECG showed prominent U waves. Based on available history and investigations, a diagnosis of barium carbonate induced hypokalaemic periodic paralysis was considered. The patient was moved to the intensive care unit and monitored for arrhythmias. He was started on intravenous potassium. On day 2, the patient had completely recovered clinically (power 5/5). Serum potassium was normal. Nerve conduction studies were performed as on day 1 with similar results except that the amplitude of compound muscle action potentials were in the normal range. The F waves and H-reflexes were still unrecordable. Electromyography was carried out with concentric needles in the biceps, deltoid, and quadriceps. It showed no abnormal insertional or spontaneous activity and motor unit action

potentials were normal in amplitude and duration. The recruitment pattern was reduced. Visual evoked potential, EEG, and brain stem evoked potential were all normal. Nerve conduction velocity and EMG were repeated on day 7 and were normal in all aspects.

Most patients with barium intoxication have gastrointestinal and cardiac involvement with tetraplegia. Barium carbonate is a rare cause of hypokalaemic periodic paralysis.¹ Diarrhoea and arrhythmias are due to direct stimulatory action of barium ions on smooth and cardiac muscles.² Heart failure and hypertension may occur in a few cases. Barium blocks the potassium channels and thus potassium efflux from the muscle is reduced whereas the sodium-potassium pump is intact. This causes increased potassium in the muscle and decreased resting membrane potential.³ Barium acts mainly at the neuromuscular junction by this mechanism.⁴ The fatal dose of barium carbonate is 0.8 g and death occurs within 2–12 hours.⁴

There is only one report of an EMG study done in four patients with paralysis because of barium carbonate poisoning. It was done within 1 week of admission and did not show any abnormality.⁴ In our case nerve conduction velocity studies and EMG were done on the first day and were sequentially repeated. Interpreting the nerve conduction velocity studies of day 1—namely, low amplitude of compound muscle action potentials—would indicate axonal neuropathy but similar findings can be found in profound muscle weakness because of neuromuscular junction blockade or myopathy. Loss of F waves and H reflexes on day 1 can be explained by the presence of lower amplitudes of compound muscle action potentials on distal nerve stimulation. Loss of F waves and H reflexes with normal distal nerve conduction velocity is suggestive of proximal demyelination or dysfunction at the spinal cord level. Loss of H reflexes and the F response has not been reported previously in patients with hypokalaemic periodic paralysis. As H reflex latency depends on several factors including the central delay in the cord involving conduction, synaptic transmission, and activation of anterior horn cells, any of these processes might be involved in barium carbonate poisoning. The exact mechanism for their absence is still unclear. These responses reverted back to normal on day 7. The concentric needle EMG study on the second day showed a reduced recruitment pattern which is found in neuropathies but has also been reported in hypokalaemic periodic paralysis.⁵

In most cases of barium ingestion the recovery from weakness occurs rapidly and uneventfully.⁴ Oral magnesium sulphate or sodium sulphate precipitate barium in the gut and thus reduce absorption. Large amounts of intravenous potassium are needed to restore the pump electrogenesis and to displace barium from potassium channels.¹ Hypertension caused by barium cannot be reversed by this treatment. Haemodialysis is a useful therapeutic adjuvant in severe barium carbonate poisoning. Ventilatory support may be required due to respiratory muscle involvement.⁴

In summary, this is the first electrophysiological study done on day 1 in a case of barium carbonate poisoning with paralysis. Previously involvement of neuromuscular junctions has been considered as a cause for weakness. In our study, we found that barium

acted at more than one level—that is, muscles, proximal segments of the reflex arc, and probably γ efferents as the tone was increased.

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Central pontine myelinolysis causes bilateral loss of deep sensitivity and pseudochoreoathetosis

Central pontine myelinolysis is characterised as symmetric and selective destruction of myelin sheath of the basis pontis. Central pontine myelinolysis often results from rapid increase of serum sodium after hyponatraemia. Patients with central pontine myelinolysis often show symptoms such as conscious disturbances, tetraparesis, and pseudobulbar palsy.^{1–3} We present a case of central pontine myelinolysis due to compensation of hyponatraemia, resulting in acute onset of pseudochoreoathetosis and sensory disturbances.

A 39 year old woman was submitted to a department of internal medicine of another hospital due to a long history of alcoholism, resulting in bad general condition accompanied by severe anaemia, toxic gastritis, and acute pancreatitis. She was alert without any mental disturbances. Laboratory data disclosed metabolic acidosis and hyponatraemia of 126 mmol/l. She received bicarbonate and physiological saline intravenously to compensate for the metabolic disturbances. Serum sodium increased to 136 mmol/l within 3 days. On the third day she suddenly developed severe choreoathetotic movements. Therefore she was transferred to our department of neurology. On the day of submission to our department she remained alert, but she had difficulties in swallowing and speaking. Examination of the cranial nerves showed bilateral ptosis and a dissociated nystagmus on the left side. We also found weakened corneal and pharyngeal reflexes. There was a general reduction of deep tendon reflexes and muscle tone. Muscle strength was normal. Involuntary movements were absent but angle positioning during movements was choreoathetotic. She was unaware of the position of her limbs with closed eyes. We instructed her to watch her limbs and she was then able to reach her nose with the finger. When investigating her sensitivity we found a complete loss of touching, vibration, and position sense in all limbs. Pinprick sensation was unremarkable in the upper limbs, whereas testing of the legs caused a painful burning sensation. Thermal sensation and