However, Hardy reported acute hypoacusis in three patients at the end of injection of local anaesthetic solution in the lumbar extradural space and postulated that the hypoacusis in his cases was a consequence of the increase in perilymph pressure which accompanied the increase in cerebrospinal fluid pressure.⁶

We report an unusual case of reversible bilateral abducens nerve, right facial nerve and bilateral vestibulocochlear nerve palsies following epidural anaesthesia. A 65 year old woman who had been having regular haemodialysis for chronic renal failure for many years was referred for management of intractable and increasingly severe pain in her neck and hands. Neurological examination and upper limb electromyographic studies were normal. Epidural block was performed through an indwelling catheter at the C6-7 level. Marcain, 8 mls of 0.25% solution, was injected through this catheter together with 40 mgs (1 ml) Depo-Medrol. The patient was placed in a head-down position and her blood pressure remained constant at 100/60 mm Hg. After 5 minutes she became dyspnoeic due to ventilatory failure. She was mechanically ventilated with 100% oxygen via a bag and mask for 30 minutes. She was then able to breath spontaneously but complained of deafness. Neurological examination revealed bilateral abducens nerve palsies, right lower motor neuron facial nerve palsy and bilateral vestibulocochlear nerve palsies. The remainder of the neurological examination was normal. The patient was observed and managed conservatively. After 3 hours her lower cranial nerve palsies had completely resolved.

The absence of long tract signs in this case indicated that the integrity of the spinal cord was preserved. It is considered that the initial mechanical ventilatory failure was due to anaesthesia of the C3, 4, 5 nerve roots that constitute the phrenic nerve, resulting in diaphragmatic paralysis. As diaphragmatic function returned it was noted that the abducens nerves and vestibulocochlear nerves were involved bilaterally in addition to the right facial nerve at the nuclear or infranuclear level. The complete return to normal function of these nerves after 3 hours (the expected duration of action of Marcain) was most consistent with an ascending local anaesthetic effect of the Marcain on the upper cervical nerve roots and lower cranial nerves.⁷ It is suspected that the Marcain solution was injected into the extra-arachnoid/subdural space and transfer of this medication in a cranial direction was facilitated by gravity as the patient had been placed in a head-down position whilst being mechanically ventilated.

Intrathecal injection would have probably resulted in more profound neurological dysfunction, involving the upper and possibly the lower limbs. Epidural injection would have been unlikely to result in lower cranial nerve palsies because these nerves exit through the dura which is attached to the base of the skull.

The occurrence of cranial nerve palsies following cervical epidural anaesthesia is an uncommon complication of this form of anaesthesia which fortunately is of benign nature with a favourable outcome, providing reassurance to both the attending clinician and patient. The timing of the onset of symptoms from anaesthesia should allow differentiation of the underlying pathogenesis into either a mechanical effect of injection or a local anaesthetic effect on nerve roots.

GRAEME J HANKEY

Department of Neurology

DAVID PERLMAN

Department of Anaesthesia,

Royal Perth Hospital,

Perth 6001, Australia

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Myokymia in motor neuron disease

Sir: In motor neuron disease, spontaneous motor unit discharges tend to be isolated (fasciculations), rather than continuous muscle fibre activity or repetitive bursts (myokymia). Occasionally, grouped discharges and doublets are seen in such patients.¹ We report a patient with amyotrophic lateral sclerosis-dementia complex and myokymia.

A 62 year old man presented with a 4 month history of progressive dysarthria, confusion, muscle cramps, and fatigue. He was moderately demented. Prominent slow rippling movements (myokymia) were evident diffusely, but were most prominent in the muscles of his lower extremities. These movements persisted during sleep. There was mild, symmetric muscle weakness and wasting that was worse in the lower extremities. Muscle relaxation was normal. Deep tendon reflexes were symmetrically brisk, and Babinski's sign was present bilaterally. Snout, glabellar, grasp, and palmomental reflexes were prominent. The following were normal: thyroid function tests; serum protein electrophoresis; cerebrospinal fluid studies; head CT scan with and without contrast; EEG; urinary lead, mercury, and arsenic determinations from a 24 hour collection; motor and sensory nerve conduction velocities, distal sensory latencies, and amplitude of evoked motor unit potentials. EMG showed abnormally increased insertional positive waves and continuous muscle fibre activity at rest. Most of the motor unit potentials appeared normal in form, but some were polyphasic and some were of increased amplitude. There were some rhythmic repetitive bursts of normal-appearing, rapidly-firing motor units (myokymic discharges), as well as occasional positive sharp waves and fibrillations. An ulnar nerve block at the elbow did not abolish the continuous muscle activity in the first dorsal interosseous muscle. Carbamazepine produced no apparent change in the spontaneous movements.

The patient continued to deteriorate, and 9 months after the onset of symptoms, he developed pneumonia and died. The neuropathology was consistent with amyotrophic lateral sclerosis-dementia complex.² There was loss of neurons and reactive astrocytosis in the motor cortex, the motor fifth and twelfth cranial nerve nuclei, and the ventral horns of the spinal cord. Spinal cord sections showed degeneration of the corticospinal tracts. Skeletal muscle sections showed grouped fibre atrophy. There were rare senile plaques in the cortex, but no neurofibrillary tangles.

Generalised myokymia is rare. When seen, it almost always reflects a diffuse injury or hyperirritability of peripheral nerves as can be seen in association with toxins,³ thyrotoxicosis,⁴ Guillain-Barré syndrome,⁵ and polyneuropathy.⁶ This concept is supported by several lines of evidence.⁵⁻⁷

Myokymic discharges can be produced locally in normal subjects with transient forearm ischaemia.⁷ In patients with inflammatory polyradiculoneuropathy, lowering serum ionised calcium may increase axonal excitability and lead to myokymic burst amplification and increased clinical myokymia.⁵ Finally, microneurographic recordings of two patients with myokymia and peripheral neuropathy resembling Charcot-Marie-Tooth disease demonstrated hyperexcitability of motor and sensory fibres.⁶

Similarly, a low excitability threshold of the axonal membrane appears to be necessary for the production of isolated fasciculations, and fasciculating neurons are often the first to be excited by stimuli of weak intensity.⁸ Grouped discharges and doublets have been seen occasionally in patients with motor neuron disease.¹ Our case suggests that motor neuron disease may cause sufficient nerve injury and hyperexcitability to produce myokymic discharges and generalised myokymia.

Myokymia and abundant fasciculations cannot be reliably distinguished clinically,⁴ and, as in our case, abundant isolated discharges and myokymic discharges may coexist.⁹ In any case, to avoid misdiagnosis, both fasciculations and myokymia need to be interpreted in light of other neurologic signs and symptoms, as well as appropriate laboratory investigations.⁴

DOUGLAS J LANSKA

ROBERT L RUFF

Department of Neurology,
University Hospitals of Cleveland,
2074 Abington Road,
Cleveland, Ohio 44106,
USA

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Methylprednisolone in multiple sclerosis: a comparative dose study

Sir: Pulsed therapy with intravenous methylprednisolone is widely used in patients with multiple sclerosis who have suffered symptomatic relapse. Open studies,¹⁻⁴ comparisons with ACTH^{5,6} and controlled trials^{7,8} have all supported this form of treatment, although a recently completed multicentre comparison of methylprednisolone with ACTH has shown no advantage of the former (Kennard C, personal communication). The mode of action of the drug is not clear. Recent work suggests that there may be no direct effect on immunological events,⁹ but other factors involved in cell damage and associated oedema may be influenced.

The optimum amount of steroid required may vary between individuals and between relapses. Upper limits are restricted by the anticipated side effects of very high dosage, but there has been little comparison of dose ranges. To explore one aspect of this question, a series of 32 patients in relapse were randomised to receive a single 1 g intravenous injection of methylprednisolone or five consecutive injections of 1 g daily. Relapse was defined as a clinical progression of the disease during the previous month. The two treatment groups were equally matched according to age, disease duration and disability. Kurtzke functional and disability scores¹⁰ were obtained before and after treatment and the study was conducted in an open fashion.

On comparing the assessments immediately before the injections with those one month after the treatment course, the mean change in the Kurtzke functional score was 2.8 in the 1 g group and 4.8 in the 5 g group. The corresponding mean changes in the Kurtzke disability scores were 0.8 and 1.9.

When analysed by the Mann-Whitney Test for non-parametric data, the differences in the disability scores were significant ($p < 0.05$). The 15 individuals in the five injection group either improved or in three instances were unchanged. Three of the 17 patients receiving a single injection continued to deteriorate after treatment, yet each improved following a further five injections.

It is unlikely that the open design of this study has caused a bias in the results and it is concluded that in many patients the lower dose of methylprednisolone is not suitable. Our present policy is to tailor the methylprednisolone dose to the patient. A regime comprising a five day course of daily 0.5 g injections, followed by 5 days of reducing oral prednisolone, appears satisfactory in most cases of relapse.

These data were presented at a Symposium on Steroids and CNS disease, and will be published in a book of the same title by John Wiley & Sons Ltd.

L BINDOFF

PR LYONS

PK NEWMAN

M SAUNDERS

Department of Neurology,
Middlesbrough General Hospital,
Middlesbrough,
Cleveland TS5 5AZ, UK

Some of this data will be published by John Wiley & Sons in a book entitled *Steroids and CNS Disease*.

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