nerve, mixed median nerve and mixed ulnar nerve potentials were 24, 23 and 38 μ V respectively. Following 9 months treatment they were 19, 25 and 38 μ V respectively. The minimum F response latency (median nerve stimulation) and the triceps surae H response latency (popliteal fossa stimulation) were normal before treatment (26.2 ms, 32.4 ms respectively) and did not change significantly after treatment (25.5, 30.8 ms respectively).

Somatosensory cortical responses, referred to Fz, were recorded from 2.5 cm behind the vertex following posterior tibial nerve stimulation at the ankle, and from a point just posterior to C3 and C4 on the 10–20 system, following median nerve stimulation at the wrist. Erbs point and spinal (C2 spine) recordings were also made but are not shown. Stimuli sufficient to cause a small muscle twitch were delivered at 3.1 Hz and two runs of 256 trials were averaged. The N20, P25 and P40 components of the SSEP from right median nerve stimulation, before and after treatment, are shown in the fig. Before treatment the peak latencies were approximately 25, 35 and 56 ms respectively. After treatment these latencies fell to 22.5, 31.2 and 48 ms respectively. In addition to shorter latencies, the entire waveform was better synchronised after treatment. Erb's point potentials were normal (latencies to peak, 10.2 ms before, 9.9 ms after treatment). The cervical potentials were poorly defined and could not reliably be measured. Following right posterior tibial nerve stimulation no cortical event could be discerned

before treatment but a P45 was seen clearly at 54 ms, after therapy.

This patient presented subacutely with the signs and symptoms of spinal cord disease due to Vitamin B12 deficiency. The subacute presentation may have been precipitated by the coincident metabolic stress of surgery⁸ or by the nitrous oxide anaesthesia.⁹ In addition to the clinical evidence, she had electrophysiological evidence of spinal cord dysfunction which has been documented previously in three patients.⁷ Two of these three cases were elderly and had absent sural nerve action potentials which may have affected the latency of their cortical responses. The patient reported here was elderly but had normal peripheral nerve sensory and mixed nerve action potentials. Thus the cortical delay demonstrated in the fig must be due to some pathological process rostral to the dorsal root ganglion, perhaps rostral to the dorsal root as the H responses were within normal limits. Although some delay remained after 9 months' therapy (fig b), a definite improvement has been recorded. This conclusion concurs with the neuropathological data which suggest that the initial neurological abnormality in Vitamin B12 deficiency is a myelopathy.¹⁴ Although the delayed arrival of somatosensory information at the cortex suggests a demyelinating process and a similar delay has been demonstrated in the nerves of the lower limb once Vitamin B12 deficiency has become established,^{2,6} this may be due to a selective fallout of large diameter myelinated nerves, as is seen in the peripheral nervous system.¹⁰

Fig Somatosensory evoked potentials (referred to Fz) from just posterior to C3 following right median nerve stimulation at the wrist in a 73 year old female with pernicious anaemia before (A) and after (B) 9 months' Vitamin B12 therapy. Consecutive runs of 256 trials have been averaged on each occasion. The latencies of the N20, P25 and P40 are 25, 35 and 56 ms respectively before treatment and 22.5, 31.2 and 48 ms respectively after treatment.

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References

- Pant S, Asbury AK, Richardson EP. The myelopathy of pernicious anaemia: a neuropathological reappraisal. *Acta Neurol Scand* 1968;44:Suppl 35, 1–36.
- Pallis CA, Lewis PD. *The Neurology of Gastrointestinal Disease*. London: WB Saunders, 1974:34–41.
- Shorvon S, Carney MWP, Chanarin I, Reynolds EH. The neuropsychiatry of megaloblastic anaemia. *Br Med J* 1980;281:1036–8.
- Victor M. Vitamin B12 Neuropathy. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds.

Peripheral Neuropathy 3rd ed. Philadelphia: WB Saunders, 1984:1925–6.

- Gilliat RW, Goodman HV, Willison RE. The recording of lateral popliteal nerve action potentials in man. *J Neurol Neurosurg Psychiatry* 1961;24:305–18.
- Cox-Klazinga M, Endtz LJ. Peripheral nerve involvement in pernicious anaemia. *J Neurol Sci* 1980;45:367–71.
- Fine EJ, Hallett M. Neurophysiologic study of subacute combined degeneration. *J Neurol Sci* 1980;45:331–6.
- Amess JAL, Burman JF, Murphy MF, Paxton AM, Mollin DL. Severe bone marrow change associated with unsuspected mild Vitamin B12 deficiency. *Clin Lab Haematol* 1981;3:231–7.
- Schilling RF. Is nitrous oxide a dangerous anaesthetic for Vitamin B12 deficient subjects? *JAMA* 1986;255:1605–6.
- McCleod JG, Walsh JC, Little JM. Sural nerve biopsy. *Med J Aust* 1969;2:1092–6.

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Polyneuropathy cranialis following cervical epidural anaesthesia

Sir: Epidural anaesthesia is a relatively safe and useful method of pain control but complications may infrequently arise. Most of the reported neurological complications of epidural or spinal anaesthesia primarily affect the lower extremities.¹ Paraplegia may result from spinal cord ischaemia due to arterial hypotension during surgery or compromise of blood supply to the cord by the procedure or condition of the patient, or by the production of an epidural or subdural haematoma.² Direct injury to the spinal cord or the nerve roots has also occurred following inadvertent subarachnoid administration of toxic chemicals, or chemical contamination of anaesthetic solution.² Cranial nerve palsies are rare. Unilateral trigeminal nerve palsy has been reported after lumbar epidural anaesthesia; the proposed mechanism being an ascending local anaesthetic effect.³

Hypoaacusis, after spinal anaesthesia, has also been reported and it was postulated that a decrease in cerebrospinal fluid pressure produced a distortion of the structure of the inner ear by pressure imbalance, resulting in a dampening of the response to auditory stimuli.^{4,5} (There is a free communication across the cochlear aqueduct between the cerebrospinal fluid and the perilymph of the cochlear apparatus, thus changes in cerebrospinal fluid pressure are accompanied by changes in cochlear perilymph pressure).

Letters

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.

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References

- 1 Dawkins CJM. An analysis of the complications of extradural and caudal block. *Anaesthesia* 1969;24:554-63.
- 2 Kane RE. Neurologic deficits following epidural or spinal anaesthesia. *Anesth Analg* 1981;60:150-61.
- 3 Shigematsu T, Wang H, Nagano M. Trigeminal nerve palsy after lumbar epidural anaesthesia. *Anesth Analg* 1985;64:646-53.
- 4 Gordan AG. Hypoacusis after spinal anaesthesia. *Lancet* 1983;ii:793.
- 5 Panning B, Mehler D, Lehnhardt E. Transient lower frequency hypoacusis after spinal anaesthesia. *Lancet* 1983;ii:582.
- 6 Hardy PAJ. Hypoacusis following extradural injection. *Br J Anaesth* 1985;57:573.
- 7 Burn JM, Guyer PB, Langdon L. The spread of solution injected into the epidural space, a study using epidurograms in patients with the lumbosacral syndrome. *Br J Anaesth* 1973;45:338.

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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.

trophic 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 astrogliosis 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