

Letters

Intradural herniated cervical disc associated with chiropractic spinal manipulation

Sir: We read with interest the report by Parnell,¹ on cervical intradural disc protrusion, and describe a new case in which severe myelopathy followed spinal manipulation.

A 31 year old man developed torticollis followed by right cervico-brachial neuralgia at C8-T1 level. Cervical spine manipulation was performed on 7 May and 14 May 1985. On 17 May he developed a sensory-motor deficit in the lower limbs, then the upper limbs, and sphincteric dysfunction. There was a spastic tetraparesis, worse in the lower limbs, with a bilateral Babinski sign. The response of the right triceps reflex was reversed. There was hypoaesthesia below the T6 level and superficial sensation was blunted at the extremities of the upper limbs. There was also bladder paralysis and retention of urine. Myelography with contrast showed a lateral defect of extra-dural type at C5-C6 disc level and a complete block in front of the lower part of the C6 body. A CT scan, performed immediately afterwards, confirmed a posteromedial protrusion of the C5-C6 disc. At that level, the premedullary space was not visible. At C6-C7 level (fig), the subarachnoid spaces were interrupted by a right posterolateral disc herniation which pushed back the spinal cord. After myelography, the spasticity of the lower limbs and the motor deficit in the upper limbs increased.

Surgical treatment was performed urgently through an anterior approach. At C5-C6 level, the protrusion was curetted; at C6-C7 level, there was an extra-intradural stud-like herniated disc; removal of this was

followed by a leak of CSF through the dural defect. After operation the patient improved slowly; three weeks later he could stand upright.

An intradural (in fact extra and intradural) disc herniation is very rare and most often observed in the lumbar regions.² In 1988, we found only six cases¹⁻⁷ at the cervical level. The symptoms were those of an acute radiculo-medullary compression, often initially lateral. According to Parnell¹ the association of a lower motor neurone arm weakness paraparesis, a dissociated sensory loss (or a Brown-Sequard syndrome) and a Horner's syndrome point to the diagnosis. The diagnosis may be suspected when myelography shows an intradural irregular block but the dural sheath is not pushed back and, as in our case, was readily established by CT following myelography. At the lumbar level, in patients with a long history, an intradural disc herniation may be explained by the adherence of the dural sheath to the posterior longitudinal ligament or by its progressive erosion by a free fragment of disc.⁵ At the cervical level, it is the result of a strong physical effort. Our case is the first in which spinal manipulation was responsible. We agree with Lanska *et al*:⁸ doctors and chiropractors should be aware of this complication which may cause a severe neurological deficit requiring an emergency operation.

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Sciatic nerve damage due to toilet seat entrapment: another Saturday night palsy

Sir: We report two patients who suffered sciatic nerve palsy due to pressure from a toilet seat during a period of unconsciousness.

A 33 year old man was admitted as an emergency complaining of swelling, numbness, and weakness of his left leg. He had administered a solution of crushed barbiturate tablets by injection into his left femoral vein 15 hours earlier, in addition to taking oral barbiturates. Six hours after the injection, he had been found by the police slumped in a public toilet, with his left buttock and thigh wedged in the toilet seat. Some effort was required to extract him from this position. He recovered consciousness after being given intravenous naloxone in the local casualty department, and spent the night in police custody, at which point his symptoms of left leg weakness became apparent.

On examination, he was a well nourished man with multiple venous varicosities and injection marks, and a swollen, tense left leg. Erythema of the skin was evident on his left buttock. Neurological examination of the right leg was normal. In the left leg, hip flexion was normal, but knee flexion was weak, (MRC grade 3-4), and movements of the ankles and toes virtually absent (MRC grade 2). Sensation to light touch and pinprick was absent over the posterior aspect of the thigh and calf, and over the lateral aspect of the calf and the dorsum and plantar aspect of the foot, sparing the medial malleolus. The knee jerk on the left was reduced compared with the right side, and the ankle jerk

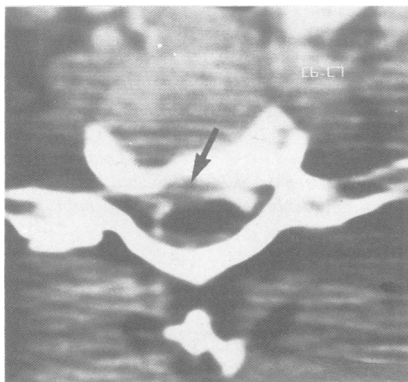


Fig CT myelogram showing the disc protrusion (←) at C6-C7 level.



Fig 1



Fig 2

Figs 1 and 2 Linear bruises over posterior aspects of both thighs (Case 2).

and plantar response were absent. A clinical diagnosis of sciatic nerve palsy was made, but electromyographic studies were not performed because the patient was Hepatitis B antigen positive. The extensive sensory loss may have been due to damage to the posterior cutaneous nerve of the thigh, which runs close to the sciatic nerve in the upper part of the thigh. The reduced knee jerk may have been due to muscle necrosis. Over a period of 6–8 weeks the patient made an almost complete recovery, although his course in hospital was initially complicated by acute oliguric renal failure with greatly elevated serum creatine phosphokinase, due to muscle necrosis, necessitating peritoneal dialysis.

The second case was a 33 year old labourer who presented as an emergency on a Sunday morning complaining of difficulty in walking, with painful calves and vomiting. He gave a history of regular alcohol consumption of up to 20 pints of beer daily, for a number of years. On the evening prior to admission, after a particularly heavy drinking binge, he fell asleep on the toilet, and awoke several hours later with both buttocks stuck in the toilet seat.

On examination, he was well nourished, with no cutaneous signs of liver disease. There were linear bruises over the posterior aspects of both thighs (figs 1 and 2). He had slurred speech, horizontal nystagmus, and intention tremor. He had bilateral foot drop

(MRC grade 2) with weak hip extensors, knee flexors, and foot dorsiflexors (MRC grade 3–4). Cutaneous sensation was impaired bilaterally below the knees, with sparing of the medial aspects of both calves. Ankle jerk and plantar responses were absent. Electromyography confirmed the diagnosis of sciatic nerve palsy, with denervation potentials observed in all muscles supplied by the sciatic nerve after its emergence from the sciatic notch. The cerebellar signs improved after several days. Acute renal failure with elevated serum creatine kinase was treated with fluids, mannitol, and calcium resonium. Renal function subsequently returned to normal. However, six months after presentation, there was a persistent neurological deficit.

These cases illustrate the hazards of prolonged immobilisation, with nerve damage which may be due to direct pressure, or to a compartment syndrome. Lesions of the sciatic nerve are usually traumatic. They have been described following orthopaedic surgery, particularly hip replacement¹, sitting for a prolonged period on a hard surface² and in drug or alcohol-induced coma.^{3,4} The sciatic nerve originates from roots L4 to S2, and leaves the pelvis through the sciatic notch, passing under the piriformis and over the obturator muscle. Direct pressure may damage the nerve at this level by compressing it against the sciatic notch. In these cases, the compression may have

been worsened by hip flexion, since this tightens the nerve against the sciatic notch.

Alternatively, or in addition, damage may result from haemorrhage and oedema due to a compartment syndrome.^{4,5} This term refers to the local manifestations and sequelae of ischaemic damage to nerve and muscle due to compromised tissue circulation, resulting from increased pressure (from oedema and haemorrhage) within a fascial compartment. This is usually associated with a crush syndrome, which refers to the systemic effects of massive muscle necrosis, with myoglobinuria, shock and acute renal failure. The extent of the soft tissue compression is illustrated by the severity of the rhabdomyolysis in both patients described, and it is likely that both direct pressure and compartment syndrome may be involved in the pathogenesis of neuronal damage of this type. The rapid recovery in function in the first patient suggests that the nerve damage was predominantly neuropractic in type, while the continued disability in the second patient suggests more severe, structural damage due to Wallerian degeneration of the nerve axons.

Compression from a toilet seat has, to our knowledge, been described as a cause of sciatic neuropathy. In both cases muscle hypotonia in association with unconsciousness allowed descent of the buttock within the ring of the toilet seat. These two cases demonstrate another hazard of unattended coma, and widen the textbook concept of "Saturday Night Palsy".

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Schizophrenic psychosis associated with benzhexol (artane) therapy

Sir: We wish to report the first case of a schizophrenic illness associated with the therapeutic use of high dose benzhexol.

The patient, a 30 year old female, was diagnosed with spasmodic torticollis at the age of 17. For several years she was treated with various agents, including amantadine, clonazepam, trifluoperazine, haloperidol and baclofen, but without success. At the age of 23 she was admitted to a psychiatric hospital, with an obsessive-compulsive disorder (OCD), which persisted for several years. This was originally associated with depression and dysmorphophobic beliefs concerning her appearance, but only the OCD persisted. She was again admitted at the age of 26 with depression associated with dysmorphophobic concerns.

Three years later she was referred to a specialist neurology clinic for a second opinion concerning her movement disorder. Spasmodic torticollis was again diagnosed, and benzhexol therapy started. Over a three month period the dose was increased to 45 mg daily, and there was a complete resolution of the torticollis.

Four months after starting the benzhexol, she developed a paranoid psychosis, associated with delusions of persecution, delusions of reference and delusions of having AIDS, in addition to her previous dysmorphophobic fears. She was admitted to a psychiatric hospital, where she was irritable and abusive but there was no evidence of a confusional state. She was treated with chlorpromazine and her benzhexol therapy was reduced to 15 mg daily.

Over the next two months her condition deteriorated. She developed thought broadcasting and third person auditory hallucinations, including voices instructing her on her behaviour. She became convinced that the hospital was a concentration camp, and that her husband had been turned into a Nazi

officer who had murdered her family. She was also convinced that the members of her family who visited her in hospital were impostors. She accused the nurses of pouring alcohol down her throat. There were also features suggestive of a mood disorder, with irritability, aggression and elation. At no stage was there any alteration in consciousness.

She was treated with increased doses of chlorpromazine, thioridazine and lithium, and made a gradual recovery, leaving the hospital after 4 months. When she was seen in outpatients, still taking benzhexol 15 mg, she was improving. However, six months later confusion over dosages resulted in the prescription of benzhexol being increased to 15 mg tds. Within one month she had deteriorated. The principal complaint was of thought broadcasting, but Capgras's syndrome was again present. All treatment was stopped, but there was no change in her condition over the eight week period before neuroleptic therapy was again instituted. She still complains of thought broadcasting six months after stopping benzhexol. Her torticollis has not returned. There is no family history of neurological or psychiatric illness, and there is no history of alcohol or illicit drug abuse.

Benzhexol toxicity is well known. The principal features are those of a toxic confusional state, in which visual hallucinations and disorientation of time and memory are prominent.^{1,2} As benzhexol has euphoriant properties it is a frequent drug of abuse, and it is in this context that most psychiatric side-effects are reported. A recent series³ included three cases in which a brief psychosis occurred after overdosage of between 25 mg and 50 mg. It is not clear, however, if this was associated with clouding of consciousness. There are fewer reports of psychiatric side effects occurring after the therapeutic use of benzhexol, although an early case is of particular interest. Bolin⁴ described a 32 year old female who was prescribed a regular dose of 30 mg benzhexol for spasmodic torticollis (with therapeutic benefit), who developed a toxic confusional state. Rechallenge with a lower dose (8 mg) led to a reappearance of irritability and ill-defined ideas of reference.

In our current case there is little doubt about the diagnosis of a schizophrenic psychosis, made by the persistence of several first rank symptoms without a disorder of consciousness. The important question is whether this was precipitated by benzhexol. It is possible that the psychosis was unrelated to therapy, since dysmorphophobia may precede schizophrenia.⁵ However, the principal psychiatric disorder before either the

development of a psychosis or the introduction of benzhexol, was that of an obsessive-compulsive disorder (OCD). Marks⁶ concluded that there is no association between OCD and schizophrenia, although there may be an association with basal ganglia disorders.

There are substantial grounds for suggesting a link between the psychosis and benzhexol therapy. After 12 years of torticollis and six years of OCD, the psychosis developed within three months of starting high-dose benzhexol therapy. When a high dose therapy was inadvertently restarted her psychosis returned. Although the development of first-rank symptoms has not been recorded previously, other features of her illness, in particular the development of elation, are in keeping with previous reports of psychosis resulting from benzhexol therapy. It is unlikely that benzhexol is the sole cause of the psychosis, instead it may have precipitated a schizophrenic episode in a predisposed patient.

High dose anticholinergic therapy is being increasingly used in the treatment of several movement disorders.⁷ Clinicians should be aware of the possibility of a schizophrenic illness.

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