a child donating his or her bone marrow then a tribunal or court decision is unnecessary. If, on the other hand, the psychological benefits to the donor are dismissed then consent cannot be given by either the parents, a tribunal, or a court.

The wellbeing of the child donor must be emphasised, and it is important to provide information appropriate to the child's developmental level in

order to reduce anxiety, dispel misapprehension, and minimise pain and trauma. It is difficult to see how invoking legal procedures such as a tribunal review would facilitate the support of the child donor. It is far more likely to increase a family's distress.

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Lesson of the Week

False localising signs in the spinal cord

D R S Jamieson, E Teasdale, H J Willison

Normal radiological examination at the clinical site of a cord lesion should prompt investigation at higher levels

Acute spinal cord compression is an emergency where irreversible neurological damage can occur within hours. The diagnosis should be considered in any patient with an acute paraparesis, parasthesiae, or urinary retention, but there is commonly a delay because neurological examination is incompletely performed or the urgency of the condition is not appreciated. In the days of myelography the whole spinal axis up to the foramen magnum was routinely examined, but nowadays the use of more sophisticated imaging may miss the lesion because of the tendency to restrict the imaged field to the likely clinical site. If the signs are falsely localising there is a danger of missing a treatable cause. We describe a case where the sensory level localised the lesion 11 cord segments distal to the abnormality, leading to a serious delay in diagnosis.

Case report

A 68 year old right handed retired joiner was admitted with acute retention of urine. There had been no prostatic symptoms and no constipation. Three weeks earlier he had developed numbness on the anterior aspects of his thighs. In the initial examination he was reported to have Medical Research Council grade 4 power globally in the legs with normal tendon reflexes and plantar responses. There was impairment of pinprick sensation bilaterally below the inguinal ligament with sacral sparing. Rectal examination and chest x ray examination were normal.

Two days later he was unable to stand and he was transferred as a neurosurgical emergency with a provisional diagnosis of an acute central disc at L1. Emergency magnetic resonance imaging of his lumbar and thoracic spine up to T4 (fig 1, left) gave normal results, and a neurological opinion was sought. He was found to have a pyramidal pattern of weakness in both legs, increased knee and ankle jerks, absent abdominal reflexes, and bilaterally upgoing plantar responses. There was loss of pinprick sensation bilaterally in the L1 to L4 dermatomes, but normal pinprick sensation in the L5 to S3 dermatomes. Vibration sensation was diminished up to the iliac crests, and joint position sense was impaired at the toes. There was no spinal tenderness and examination of the arms showed no abnormality. Routine biochemical investigation including globulins, and haematological investigation showed nothing abnormal, and the erythrocyte sedimentation rate was 35 mm in the first hour. Neurophysiological examination showed electromyographic changes in the left iliopsoas muscle comprising fragmentation of motor units and a very reduced recruitment pattern. These abnormalities were interpreted as indicating interruption of the L1/L2 motor neurone

pool with noticeable recruitment block more caudally, in keeping with a focal cord lesion at about L1. The cerebrospinal fluid cell count and protein concentration were normal and there were no malignant cells or oligoclonal bands. Three days later he was found to have a confluent sensory level to light touch and pinprick at T9. Further local magnetic resonance imaging of the cervical spine showed marrow infiltration of T2 affecting the vertebral body and posterior neural arch and an extradural mass affecting the levels above and below that vertebra extending posterolaterally to affect the paravertebral soft tissues on the left (fig 1, right). This was shown histologically to be a plasmacytoma and he was treated with radiotherapy. After a prolonged period of rehabilitation he could walk with a frame and was discharged home.

Fig 1—Left: T1 weighted sagittal magnetic resonance image of the thoracic and upper lumbar regions of the spine is normal. Right: T1 weighted sagittal magnetic resonance image of the brain stem and cervical and upper thoracic spine showing marrow infiltration of T2 affecting the vertebral body and the posterior neural arch. An extensive extradural mass affecting the level below and above this vertebra is evident

Discussion

This case illustrates the often overlooked observation that there may be a large disparity between the clinical sensory level and the level of the lesion in spinal cord disease. Other falsely localising symptoms or signs that may occur in spinal cord disease include low back and leg pain simulating lumbar disc disease but actually caused by cervical cord compression¹ and wasting of the intrinsic muscles of the hands innervated by the T1 segment yet caused by a lesion at the foramen magnum.^{2,3} Benign extramedullary tumours at the foramen magnum are notorious for producing signs which do not accurately identify the anatomical

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site of the tumour, with sensory loss below C5 occurring in 15% of patients and atrophy localised to the hands in 13% of patients.³ A sensory discrepancy has been well described in cervical disc herniation,^{4,5} and in one series of five patients with painless cervical myelopathy the sensory level was consistently below T5.⁶ This has been explained on the basis of pain and temperature fibres crossing obliquely to reach the contralateral spinothalamic tracts two or three segments higher.⁷ Although this is true, it accounts only for sensory levels a few segments below the lesion, and does not explain the large discrepancies of up to nine segments below the lesion that have been described in cervical myelopathy,⁸ of seven segments in other forms of cord compression,³ or of 11 segments in our case.

Another explanation is based on the lamination of the spinothalamic tract. Fibres originating in lower segments run laterally and posteriorly, unlike those from the upper segments, which are located medially and anteriorly. However, this explanation is also unsatisfactory, since it does not explain how central—as opposed to lateral—compressive lesions cause the phenomenon. Ischaemia after arterial occlusion is a further unsatisfactory explanation, because anterior spinal artery compression would not produce a complete lesion of the cord, and there is no supporting evidence for this mechanism from pathological studies.⁸ A more plausible hypothesis is that the myelopathy is caused by venous obstruction with ensuing stasis, resultant hypoxia, and subsequent loss of anterior horn cells.² Features of anterior horn cell damage rather than root compression at the level of the clinical signs have been shown by electromyography,⁹ seen pathologically,¹⁰ and induced experimentally at sites distant from the cord lesion.² The clinical neurophysiological signs in the case described above also suggest anterior horn cell damage far removed from the lesion. However, if raised venous pressure is the mechanism the site of the anterior horn cell damage would be expected to be where the arterial pressure—and hence tissue perfusion—in the cord is lowest, but there is no consistent falsely localising level described between cases which would support this hypothesis.

This case is a timely reminder of the classical, but

rare, neurological phenomenon that high thoracic or cervical cord compression from any cause can mimic disease of the lumbar spine. Those handling medical emergencies should be aware of falsely localising sensory signs, in order that the correct investigations are done. An emergency myelogram carries little risk of missing a lesion far removed from the symptoms and signs because the dye is routinely run into the cervical region. However, sensitive magnetic resonance sagittal sections of a limited area focusing on the presumed site of the lesion can occasionally fail to diagnose a treatable lesion and thus allow further irremediable neurological deterioration. Optimal magnetic resonance imaging is achieved if a limited section of the spine is examined with a local coil. In most cases this will easily cover the possible site of a compressive lesion if the sensory level has dropped by only a few segments. On those occasions where magnetic resonance imaging shows no abnormality and the physician's conviction remains that compression needs to be excluded then imaging at higher levels is indicated. The lesson from this case should lead to a reappraisal of emergency imaging policy in cases of acute spinal cord compression: imaging of the whole spine is obligatory if no lesion is immediately apparent.

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A MEMORABLE PATIENT

A fulfilling experience

Seeking escape, with the usual relief, my toothache quelled, I was halted abruptly by his request—would I please anaesthetise the next patient? I nodded reflexly as a silent, inward voice said: "I don't do this. I'm sorry . . . a matter of principle . . . in the dental chair." For I had not quite adapted to "consultant anaesthetist, UK."

The apparatus was a Walton number something, with simple controls for gas pressure and concentration—an immediate reminder of casualty years before, and that narrow zone of nitrous oxide "anaesthesia" between octopus like struggling and the seriously puce.

Now the patient was being ushered in—a thin girl, early 20s, too preoccupied with her swollen mouth to notice erratic chairside manners. My commitment was soundly signalled by a turn of taps, a compelling swish of gases, and a flick of the nitrous oxide and oxygen gauges.

Here I was, a guest; and how trusting of them, I thought, tenser than either. I had no special skill with a nose piece, so I waited for her ear lobes to turn dusky before adding 20% oxygen. The tooth was out in seconds, she was awake in two to three minutes, and I felt almost fraudulent at the ease of this—anaesthesia for the exhausted.

Hesitating as he escorted me to the door, my dental

"colleague" reached into a trouser pocket. Crisp paper was thrust into my hand. A 10 shilling note—my fee? I recoiled in fist clenched confusion.

A knowing onlooker would have seen the funny side of this role reversal, as I did soon enough. For my prejudice against British style dental anaesthesia was at a zenith in the mid-1960s. I had added to the published concerns about postoperative hypoxaemia (so much milder than with the nitrous oxide techniques of notoriety); and I had experienced Canadian dentistry (either competent local anaesthesia or the hospital—no makeshift in between arrangement). Moreover, I was quite unused to "outside" work. In sum, I had thought the business unsafe, outdated, and disreputable if not immoral.

Inescapable irony. And strange that I cannot remember whether, in the end, I took the money. I suppose that it is all different now.—RONALD A MILLAR is a retired anaesthetist in Surrey

We welcome filler articles of up to 600 words on topics such as *A memorable patient*, *A paper that changed my practice*, *My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk.