known that steroids, especially glucocorticoids, can modify body fat composition. Excessive epidural fat growth causing spinal stenosis has been reported during both high and low dose glucocorticoid treatment. Progesterone also causes enlargement of body fat deposits, and levels of this hormone rise steadily during pregnancy. It might be therefore, that the elevated progesterone levels during pregnancy stimulated the growth of the lipoma, leading to further compression of the sacral roots.

Whatever the mechanism, in some forms of occult spinal dysraphism, especially a tethered cord syndrome or lumbosacral lipoma, symptoms can start after childbirth. When the clinical picture is restricted to urinary dysfunction, especially stress incontinence, diagnosis may be delayed and the patient submitted to needless urological interventions.

Paraspinal myoclonus due to spinal root lesion

Sir: Myoclonus is a symptom produced by a wide variety of neurological diseases. Most commonly, myoclonus is generated from cerebral cortex, thalamus, or spinal cord. By contrast, localised myoclonus arising elsewhere is less familiar. As an example of unusual localised myoclonus I report a case in which segmental mid-thoracic paraspinal myoclonus occurred as the only sign of an advanced spinal nerve root lesion.

A 38-year-old man was admitted with unilateral involuntary jerking in the dorsal back muscles. In the early 1960s he had had paralytic ileus, operation for which revealed a neurosarcotoma. Later, mesenteric and pelvic metastases were found. The patient's general condition had remained good until 4 months prior to admission, when he experienced slight superficial pain below the left scapular region for a few weeks. The pain disappeared spontaneously. Thereafter, he was asymptomatic until he experienced jerking in the muscles of the back on the left side. The vigorous but painful jerks were present occasionally, varying from one day to another, sometimes being absent for weeks. On admission, cranial nerve and cerebellar functions were unaffected. Muscle tone and strength and deep tendon reflexes of the abdominal reflexes were normal and symmetric. The plantar responses were flexor. No sensory disturbances could be detected, and in particular, no thoracic segmental abnormality was seen. The only abnormal sign observed was myoclonus in the paraspinal muscles at the level of the 5th thoracic segment on the left side. Extensive laboratory investigations showed normal values. No abnormality was found in the plasma radioisotopes of skull, chest and spine. Computed tomography (CT) of the brain and EEG were normal. Electromyography (EMG) showed a normal pattern in the muscles of the extremities and in the paravertebral muscles in the thoracic area. Unfortunately, the jerking was not present and could not be elicited at the time of the EMG investigation. Normal values were also obtained in the measurement of nerve conduction velocities and in the somatosensory evoked potentials which were examined using median, ulnar and posterior tibial nerve stimulation. Metrizamide myelography revealed a mass at the level of the 4th thoracic vertebra. CT examination showed destruction of the left pedicle of the 4th
thoracic vertebra and a tumour mass which filled up the corresponding intervertebral foramen (fig). The spinal cord was not compressed but was slightly dislocated to the right. The patient was successfully treated with local radiotherapy. He has been followed so far for over one year, remaining asymptomatic.

The well known classical clinical manifestations of spinal root compression are pain and segmental motor and sensory abnormalities. At levels such as the mid-thoracic region the signs may, however, be few and atypical. There is considerable overlapping in the distribution of the muscular and cutaneous innervation supplied by adjacent thoracic spinal nerves. Thus, for instance, section of a single thoracic root does not necessarily produce any detectable sensory disturbance. The paraspinous musculature consists of a number of long and crossing muscles and an injury to one root has little effect upon function.

Although the clinical picture of our patient was unusual, there had been a previous period of segmental pain, most probably due to root irritation. Since the symptom disappeared spontaneously, more detailed examination had been considered unnecessary at that time. It was only the later appearance of local dorsal muscle jerking which led to investigation, and it was the identification of myoclonus which led to the elucidation of the root lesion and the tumour. Myoclonus is a well established consequence of spinal lesions but its appearance in the paraspinous muscles is uncommon. Its appearance as the only manifestations of a lesion primarily involving a spinal root also is unusual. In our patient, there were no upper motor neuron signs, myelography and CT examination showed no cord compression, and the SSEPs showed no abnormal latency values at the spinal level. Therefore, the origin of myoclonus was most probably at the nerve root level. Although not likely, the possibility of spinal cord compression cannot, however, be absolutely excluded.

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References


Failure to promote spinal cord regeneration in rats with immunosuppressive treatment

Sir: We have published several reports of minor improvement in central nervous system (CNS) regeneration in rats treated in ways that would alter their immune response. Our most successful regimen consisted of treatment with 75 mg/kg of cyclophosphamide 48 hours after spinal cord transection. While no clinical return of sensory or motor skills was noted in treated rats, electrophysiology, axon-plasmic flow studies, and anatomic studies indicated that some fibres did pass through the area of complete spinal cord transection in treated rats, but not in control rats. We recently found that an earlier axon-plasmic flow study indicating some recovery in long-term untreated control rats was a misinterpretation of data. Regeneration has been seen only in rats that have been treated in ways thought to suppress their immunologic responsiveness. While all evidence of regenerating long tracts in the spinal cord has been meager, the best results have been found in those rats treated with cyclophosphamide.

The advent of the immunosuppressant cyclosporin, which is reported to be much less toxic and much more successful in inhibiting immunologic responsiveness, led to our testing this new drug in rats with complete spinal cord transection to see if it would facilitate spinal cord long tract regeneration. We compared control rats with a complete spinal cord transection performed at T-9 cord level as described earlier with rats given one of two immunosuppressive treatments. Control rats received no specific therapy. A cyclophosphamide treated group received