rapidly.[2] This makes it unlikely that increased local blood flow will result in increased tissue concentrations in the steady state, though there is the possibility of an effect where frequent intravenous injections result in rapidly rising plasma levels. In 1987 our patient had three intravenous injections of phenytoin, two of 200 mg and one of 300 mg, but not before or since. While acute ischaemia can cause hyperperfusion,[3] this usually persists longer than the four days between the original SPECT scans.

Cerebral and cerebellar damage in association with seizures may be due to hypoxia, whether absolute, due to hypoventilation, or relative, due to the high metabolic demand of the brain tissue involved in the seizure.[4] More recent work has shown that pathological changes thought to be due to hypoxia can occur where brain oxygenation and perfusion is adequate,[5] and positron emission tomographic (PET) studies have shown that perfusion and oxygenation of discharging epileptic foci is adequate for their metabolic demands.[6] Meldrum[4] proposed that sustained neuronal overactivity in itself may lead to neurological damage, whether widespread or localised (as in mesial temporal sclerosis), and there is evidence that glutamic acid, an excitatory neurotransmitter with neurotoxic properties, may mediate the effect.[7]

This case demonstrates structural and functional damage in an area only secondarily activated by the epileptic discharge, and supports the concept that neural damage in epilepsy is partly or wholly due to neural overactivity.

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Syringomyelia with spontaneous resolution

Patients with syringomyelia have a range of physical and neurological symptoms and signs[1 2] which usually progress gradually with occasional abrupt exacerbations in some patients and involves long periods without change.[3 4] The size and the shape of the syrinx can be shown by CT myelography and/or MRI to vary during a follow up period of many years. Operation may reduce the size in some cases, but patients treated surgically sometimes show recurrent enlargement of the syrinx.[5] On the other hand, spontaneous disappearance of the syrinx is not well documented. We report a case of syringomyelia with spontaneous resolution.

A 16 year old right handed boy was seen at our outpatients clinic in August, 1986 with numbness of the left side of his chest and upper limb. He was born at full term by normal delivery. At the age of 11 years, he slipped and fell down a flight of stairs, resulting in a pain on the left side of his back which lasted for a few weeks. The following year he was thrown down and fell on his back while playing Judo at school; he felt a tingling and lancinating pain in the left side of his chest and back. Following this episode, he started to experience similar pain during exercise. Several months later he noticed hypalgesia and hypoaesthesia of the left side of his chest; this spread to affect the whole of the left arm.

In April 1985, aged 13, we examined him for the first time at another hospital. Positive neurological findings at that time included equivocal weakness of extension of the left fingers, areflexia of the left arm, hyperreflexia of both legs, bilateral extensor plantar responses and dissociated sensory disturbance over the left C3–T7. CT myelography of the cervical cord revealed definite delayed uptake of the contrast material into the left dorsal parts of the spinal cord, consistent with a syrinx. In April 1986 he complained of a lancinating pain in his left finger radiating from his neck, when he sneezed, coughed, or strained at stool. On 28 July 1986, MRI (0·15 Tesla) was performed. There was a syrinx from C2 to T9 near the left dorsal column, as well as a Chiari malformation of Type 1 (fig 1a). He was referred to our clinic on 1 August 1986. Neurological examination at that time showed a slight weakness of the left finger extensor, abductor and adductor, diminished left hand grasping power (right 28 kg, left 16 kg), loss of upper and middle abdominal reflex with diminution in the lower part, and dissociated sensory disturbance over left C2–T9. Sweating was absent on the left arm and chest. He had bilateral extensor plantar responses.

In September 1988 (aged 16), the patient was evaluated at our clinic again. He did not complain of any weakness or pain. Neurological examintion revealed some changes. His muscle strength was full throughout. His hand grip power was 33·0 kg (right) and 30·5 kg (left). He showed areflexia of his left arm and hyperreflexia of both legs. He did not have extensor plantar responses. Dissociated sensory disturbance was observed from C2–L1, but the degree was less than previously. MRI (1·5 Tesla) was performed on 10 November 1988 (fig 1b). On a T1 weighted image we could find neither a Chiari malformation nor any abnormal intensity in the

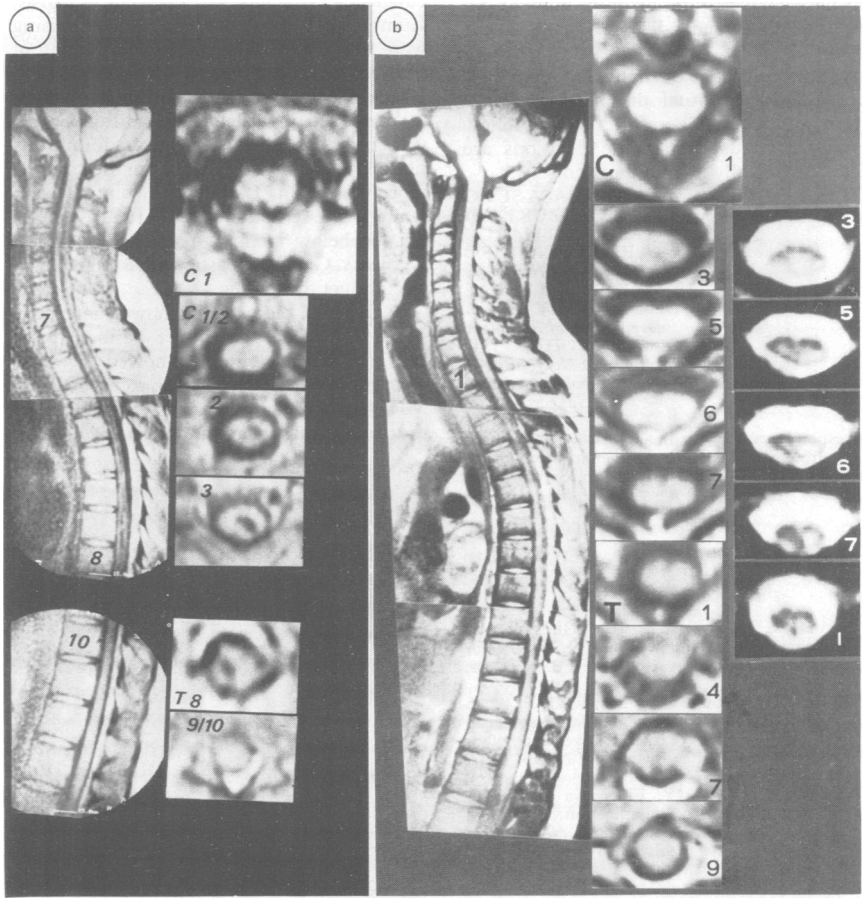

Figure 1a 1st MRI of whole spinal cord (left: TR = 500 TE = 40 sagittal, right: TR = 500 TE = 30 axial image) showing a Chiari malformation and syrinx in the spinal cord.

Figure 1b 2nd MRI (left: TR = 1000 TE = 15 sagittal, middle: TR = 600 TE = 15 Axial image), reveals neither a Chiari malformation nor syrinx in the spinal cord. However, TR = 3300 TE = 90 T2-weighted images in the right of the figure showed a high signal intensity area in the spinal cord, indicating the previous location of the syrinx.

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spinal cord; also the cord was not atrophic. The images of the brain were also judged to be normal. On a T2-weighted image there was an abnormal high intensity area in the centre of the spinal cord where the syrinx had been. There was no CSF flow-void sign or abnormality of cord size.

On MRI, after a follow up period of two years and three months, the Chiari malformation and syrinx had disappeared on the T1-weighted image. On the T2-weighted image, however, a high intensity area was clearly visible in the spinal cord. A high intensity area on T2-weighted image showed a non-specific change and could be due to gliosis, oedema, microcystic changes and/or demyelination.[6 7] We therefore assumed that the syrinx had collapsed and been replaced by gliosis.

The reduction of pain, hand muscle weakness, pathological reflexes and severity of anaesthesia are likely to reflect the disappearance of the syrinx, even though the hyporeflexia of the left arm remained unchanged and the area of sensory disturbance was somewhat enlarged.

There are several theories about the pathogenesis of syringomyelia.[1-3] They are based on the concept of the block of the flow of the CSF around the posterior fossa due, for example, to Chiari malformations or arachnoiditis. Decompression of the posterior fossa may reduce the symptoms and signs as well as the size of the syrinx.[3 4] We presume that in our case, the Chiari malformation was mild and that growth of the patient and enlargement of the posterior fossa may have led to restoration of the flow of CSF and the subsequent disappearance of the syrinx.

We conclude that the spontaneous reduction and the eventual disappearance of a syrinx can occur. Therefore, careful follow up and cautious therapeutic decisions are needed, especially in young patients.

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Syringomyelia associated with a spinal schwannoma: a case report

Schwannomas are the most common intraspinal tumours. They constitute 16 to 30 per cent of all spinal tumours in reported series.[1] The presence of syringomyelia in association with a spinal schwannoma is rare being more often seen with intramedullary tumours. There are only two reports of the former in the literature.[2 3] However, it is possible that with the increasing availability of MRI, the association will be recognised more frequently. We report a case of a schwannoma of the filum terminale associated with syringomyelia that was demonstrated on the preoperative MRI.

A 38 year old female cashier had suffered mild back pain since her teens and for a year this had been associated with pain in the anterolateral and proximal aspects of her left leg. The pain was constant, burning in nature, worse at night and not related to exertion. In the same period walking became more difficult. She had no sphincteric disturbance. On examination, she had an obvious scoliosis convex to the left. There was no cutaneous stigmata of neurofibromatosis. Lumbar movements and straight leg raising were full. There was generalised wasting of the left leg and a spastic paraparesis with Grade 4 power, the weakness being a little more marked on the left. Knee and ankle jerks were brisk and plantars extensor. Joint position sense was slightly reduced in the legs, left more than right. Light touch, pinprick and temperature sensation were all reduced in the left leg below L1 and sparing the perineal segments. Gait was spastic with a tendency to circumduct the left leg.

Plain spinal radiographs showed an expanded lower dorsal and upper lumbar spinal canal. CT myelograph showed a mass adjacent to and displacing the conus at L1/2. MRI (fig a, b) showed a mass at the level of the conus, oval in shape and indistinguishable from the cord in terms of signal intensity and plane of separation. In addition, there was slightly irregular cavitation extending proximally from the lesion into the upper thoracic cord. The cervical cord was normal in shape and size with no evidence of syringomyelia. There were no abnormalities at the craniocervical junction, in particular, no Arnold Chiari malformation. The tonsils were in a normal position at the level of a line joining the inferior tip of the clivus and the posterio-inferior lip of the foramen magnum.

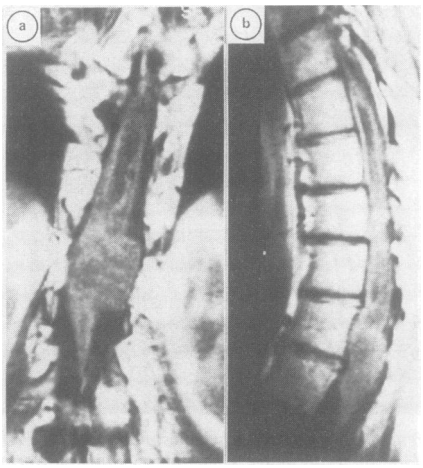

Figure T_1 weighted MRI, coronal (a) sagittal (b), show a mass indistinguishable from the cord at the level of the conus. There is a syrinx cavity above the lesion.

The patient had a D11 to L2 laminectomy. This revealed an intradural firm pinkish tumour sized 5 × 3 cm excavating the conus from the left. Above the lesion, the cord was expanded. A small mid-line myelotomy above the tumour revealed a syrinx containing clear fluid identical with cerebrospinal fluid (CSF). The tumour was totally excised with the help of a Cavitron Ultrasonic Surgical Aspirator and it appeared to arise from the filum terminale. After the tumour was excised, a small hole in the cord just above the conus was visable at the upper end of the tumour bed which appeared to communicate with the syrinx cavity. Histology confirmed a typical appearance of a schwannoma with no malignant changes. Post operatively, there was a temporary deterioration of the patient's paraparesis which has since improved.

Spinal schwannomas almost invariably originate from the posterior roots of the spinal nerves[4] and this may account for the high incidence of radicular pain as a presenting symptom. These tumours may be single or multiple and may or may not be associated with generalised neurofibromatosis. In the report of 115 cases of spinal schwannomas by Gautier-Smith,[5] only 12 cases had some cutaneous manifestations of neurofibromatosis. The presence of cord signs in association with lumbar schwannomas is uncommon since there must be spread upward of the tumour to produce this.[5] However, in our case the degree of spastic paraparesis was disproportionate to the extent of cord compression by the tumour which was effectively confined to the conus only. Presumably the ascending syrinx was responsible for the upper motor neuron signs.

In 1969 Williams[6] introduced the concept of communicating and non-communicating syringomyelia referring to any existing connection between the syrinx cavity and the fourth ventricle. The majority of patients with a communicating syrinx also have some form of hind brain abnormality such as Arnold Chiari malformation or Dandy Walker cyst with resulting obstruction of the normal outlets of the fourth ventricle.[4]

Non communicating syringomyelia is less common and relates to the extension of cystic cavities within the cord or to blockage of the central canal within the cord. The commonest causes are, intramedullary tumours, post traumatic paraplegia, and dense arachnoiditis.[7]

Syringomyelia occurring with intramedullary spinal tumours is a well known entity but extramedullary tumours causing syringomyelia are less common. In a review of the world's literature up to 1973, Barnett *et al*[7] reported seven cases of pathologically proven syringomyelia associated with extramedullary tumours, five of which were thoracic and two were cervical in location. Few cases have been reported since and these have been in the main meningiomas.[8] There were three cases of schwannomas.

In attempting to explain the pathogenesis

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