Incidence, management, and outcome of post-traumatic syringomyelia
In memory of Mr Bernard Williams

W S El Masry, A Biyani

Abstract

Objectives—To determine the incidence of clinically diagnosable post-traumatic syringomyelia (PTS).

Methods—A population of 815 consecutive patients with traumatic spinal cord injuries was studied between January 1990 and December 1992.

Results—Reviews of all records, full clinical evaluation, and thorough neurological examination of all patients disclosed 28 patients in whom PTS was confirmed radiologically (3-4%). The incidence of the presenting symptoms, including bladder dysfunction, is described. The level and density of cord lesion was correlated with incidence and it was found that post-traumatic syringomyelia was twice as common in patients with complete injuries than in patients with incomplete injuries. The highest incidence was found in patients with complete dorsal and complete dorsolumbar injuries. The interval between injury and diagnosis ranged from six months to 34 years (mean 8-6 years). This interval was shortest in patients with complete dorsal and incomplete cervical and dorsolumbar cord injuries.

Conclusions—Reduction of the size of the syrinx seen on postoperative MRI correlated well with a satisfactory clinical outcome in 85% of patients.

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Keywords: post-traumatic syringomyelia; management; outcome

Syringomyelia is potentially a disastrous complication of spinal cord injury with reported incidence of 0-3 to 2-2%. Since the description by Barnett et al. in 1966, post-traumatic syringomyelia (PTS) is being increasingly recognised and its diagnosis has become easier and more accurate with the availability of MRI. However, PTS as an entity is still not widely appreciated, its incidence and aetiopathogenesis are not well understood, and management guidelines have not been firmly established.

The incidence of PTS is higher in the dorsal spine in some series6,7 and higher in the cervical spine in others.6,7 No correlation between the severity of cord injury and the incidence of PTS was found by some authors,1,8–10 but others6,7 reported a two to three times higher incidence of syringomyelia after complete cord injury than after incomplete cord injury. Rossier et al.12 reported a 3-9% and 2-4% incidence of PTS after complete and incomplete spinal cord injury respectively. The incidence of PTS after incomplete and complete cord injury separately at each spinal level has not been published so far.

The interval between spinal injury and syringomyelia is highly variable, ranging from two months3 to several years. Lyons et al.15 reported earlier onset of syringomyelia after complete cord injury than after incomplete injury. This contradicts previous reports.1 It has been reported that PTS occurs after surgical11–13 and conservative14 treatment. Unfortunately, many of the larger studies6,8,15,16 do not outline the mode of initial treatment of the spinal cord injury. The important factors causing variability in the reported incidence of PTS may be related to the difference in treatment regimes in different institutions and the difference in clinical and neuroradiological criteria for the diagnosis of PTS. This retrospective study considers some of the controversies in the incidence characteristics of PTS. In addition, it outlines the clinical and MRI features, evaluates the results of surgical treatment, and correlates the postoperative MRI appearances and the clinical outcome.

Materials and methods

Since 1965 the Midlands Centre for Spinal Injuries (MCSI) has provided a “cradle to grave” service to patients with spinal cord injury. Most of the patients are regularly reviewed and neurologically examined every one to two years in the centre. Between June 1990 and December 1992, a total of 815 consecutive patients with old or new spinal cord injuries (390 incomplete and 425 complete) were critically examined for evidence of syringomyelia. They included all the new patients sustaining spinal cord trauma and all the old patients, who were regularly followed up from 1965 onwards with at least one review during this three year period. Patients with syringomyelia after non-traumatic spinal paralysis and iatrogenic and stab injury to the spinal cord were excluded. There were 28 patients with clinically diagnosed and radiologically confirmed PTS. Nineteen patients had complete cord injuries and nine incomplete spinal cord injuries with an overall cumulative incidence of 3-4%, twice as common after complete (4-5%) cord injury than after incomplete cord injury (2-3%).

There were 24 men and four women with a
mean age of 26.9 (range 17–45) years at the time of sustaining the spinal cord injury. Road traffic accidents (17 patients) and falls (six patients) were the commonest causes of injury. Four cases had been treated surgically (one each Harrington instrumentation, Hartshill rectangle, laminectomy, and cervical fusion) and the rest were treated conservatively at the time of the injury.

Table 1 shows the injury to PTS interval and the frequency of PTS at each spinal level in the patients with neurologically complete and incomplete lesions. Complete dorsal and cervical cord injuries were most commonly complicated by PTS. Four patients (two incomplete cervical and one each complete dorsal) had developed PTS within six months of cord injury. They had been treated conservatively in the routine manner for the spinal injury and they did not have any identifiable adverse factors.

The clinical examination included the assessment of type, duration, and severity of pain, hyperhidrosis, and spasticity as well as a complete neurological examination including cranial nerve function. The commonest clinical features of PTS were pain and ascending sensory level (table 2). Two patients had developed hyporeflexic urinary bladder, as the sole clinical feature in one and in combination with other clinical features of PTS in another.

The diagnosis and extent of PTS (table 1) was confirmed by CT myelography in two patients and air myelography in one patient in the earlier part of the study. Magnetic resonance imaging with T1 and T2 weighted axial and sagittal scans of the spinal cord and the medulla were performed in the remaining 25 patients. The syrinx extended above and below the vertebral fracture in all but two patients, whose syrinx extended only below the level of the fracture. Both these patients had developed PTS within six months of sustaining cervical cord injuries.

Preoperative MRI was performed in 21 patients. Three of the scans done elsewhere were not available. Preoperative CT myelography in two patients in the earlier part of the study delineated the length of the syrinx but one preoperative air myelogram done in 1973 did not. In these 20 patients treated surgically for PTS the syrinx spanned over a mean of 10.0±5 (range 2–19) vertebral segments (table 1). The syrinx extended over a mean of four segments in four conservatively treated patients with small asymptomatic, non-progressive syringomyelia. These four patients were followed up with serial MRI at six to 12 month intervals.

Surgical treatment was indicated in the remaining 24 patients with symptomatic and progressive PTS (table 3). Two of these patients were awaiting surgery at the time of data collection for this study. The operative procedures in the remaining 22 patients included one myelotomy, one cordectomy, one cord transection, seven syringosubarachnoid shunts, and 13 syringopleural shunts. More recently, omental grafting was performed either primarily or after a failed surgical procedure. The patients were reviewed at three to six months and subsequently at 12 to 24 months with serial MRI to assess the effectiveness of the drainage of the syrinx.

The outcome of the decompressive surgery for PTS on the postoperative MRI was graded as good, fair, and poor. Complete or nearly complete drainage of the syrinx was graded as good. Mild to moderate postoperative reduction in the size of the syrinx was considered fair. When there was no change or increase in the diameter or extent of the syrinx this was graded as a poor result.

### Table 1  The incidence, injury to PTS interval, and extent of lesion according to level and density of cord injury

<table>
<thead>
<tr>
<th></th>
<th>CC</th>
<th>CI</th>
<th>DC</th>
<th>DI</th>
<th>DLC</th>
<th>DLI</th>
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<tbody>
<tr>
<td>Incidence</td>
<td>3.6%</td>
<td>1.2%</td>
<td>4.8%</td>
<td>4.8%</td>
<td>5.6%</td>
<td>3.8%</td>
</tr>
<tr>
<td>Mean interval (y) between injury and diagnosis of syringomyelia</td>
<td>10-7</td>
<td>6-5</td>
<td>6-5</td>
<td>12-0</td>
<td>12-6</td>
<td>5.3</td>
</tr>
<tr>
<td>Range</td>
<td>(6m-34y)</td>
<td>(6m-10y)</td>
<td>(6m-22y)</td>
<td>(2-18y)</td>
<td>(8-19y)</td>
<td>(3-7y)</td>
</tr>
<tr>
<td>Longitudinal extent of the syrinx</td>
<td>18 MRI and 2 CT</td>
<td>9-3</td>
<td>9-3</td>
<td>11-5</td>
<td>8-0</td>
<td>12-7</td>
</tr>
</tbody>
</table>

**CC** = Cervical; complete.  **CI** = cervical, incomplete.  **DC** = dorsal, complete.  **DI** = dorsal, incomplete.  **DLC** = dorsolumbar, complete.  **DLI** = dorsolumbar, incomplete.

### Table 2  Clinical presentation of PTS

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<tr>
<th></th>
<th>CC</th>
<th>CI</th>
<th>DC</th>
<th>DI</th>
<th>DLC</th>
<th>DLI</th>
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<tbody>
<tr>
<td>No</td>
<td>6</td>
<td>3</td>
<td>10</td>
<td>3</td>
<td>3</td>
<td>28</td>
</tr>
<tr>
<td>Pain</td>
<td>5</td>
<td>2</td>
<td>8</td>
<td>2</td>
<td>2</td>
<td>21</td>
</tr>
<tr>
<td>Sensory changes</td>
<td>4</td>
<td>1</td>
<td>10</td>
<td>2</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>Loss of reflexes</td>
<td>3</td>
<td>—</td>
<td>6</td>
<td>2</td>
<td>—</td>
<td>11</td>
</tr>
<tr>
<td>Loss of temperature sensation</td>
<td>1</td>
<td>1</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Motor deficit</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Hyperhidrosis</td>
<td>3</td>
<td>1</td>
<td>4</td>
<td>—</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Increased spasticity</td>
<td>—</td>
<td>1</td>
<td>3</td>
<td>—</td>
<td>—</td>
<td>5</td>
</tr>
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For abbreviations see table 1.
The effect of surgery on neurological functions, pain, hyperhidrosis, spasticity, or any other associated feature of syringomyelia was also noted postoperatively and at each review. The results at the last follow up were assessed by criteria modified from those used by Tator et al.17 and Barbaro et al.18 and were graded as good, fair, and poor. A good result was defined as improvement of neurological signs and symptoms along with reduction in pain and hyperhidrosis. A fair result was identified when the neurological deficit was stabilised with improvement or no change in the severity of pain or hyperhidrosis. If the patient required repeat surgery for recurrence of syringomyelia or any other reason, the results were described as poor.

Epinfo 5.00 Statacalc (1990) software was used for statistical analysis.

Results
A diagnosis of PTS was made in nine patients after incomplete and in 19 patients after complete spinal cord injury after mean postinjury intervals of 6-9 years and 9-4 years respectively. Two patients with complete cord injury had developed PTS more than 30 years after the original injury, thereby significantly increasing the injury to syringomyelia interval in this group. Student's t test did not show any statistically significant difference in the injury to PTS interval between incomplete and complete spinal cord injury (P = 0.47) or between the cervical (mean 8-7 years) and the dorsal (mean 8-5 years) spine (P = 0.96). The injury to PTS interval was the shortest in the patients with complete dorsal and incomplete cervical and dorsolumbar injury (table 1), but the number of patients in each group was too small to draw any statistical conclusions.

Pain and sensory changes were the commonest clinical features of PTS and there was no significant preoperational difference in any one clinical feature of PTS at any spinal level, with either incomplete or complete spinal cord injury. The diagnosis of PTS had been delayed in two patients treated elsewhere and sympathectomy for hyperhidrosis had been carried out with good improvement in one and unsuccessful ulnar nerve decompression at the cubital tunnel in the other. Retrospectively, both these patients had clinical features suggestive of PTS at the time of misdirected surgery.

Four conservatively treated patients did not have any significant symptoms and the syrinx did not progress over a two year follow up period. Two patients were awaiting surgery at the time of data collection. Nineteen of the remaining 22 surgically treated patients (86, 4%) had good and fair improvement. Pain and motor deficit (table 4) were the commonest modalities to recover. Hyperhidrosis and spasm also improved in most patients but sensory recovery was less consistent (table 4). The least improved modality was the return of tendon reflexes (14%). Two patients with hyporeflexic urinary bladder did not have any change in their bladder function. One patient with fifth nerve involvement and Horner's syndrome improved postoperatively. Postoperative MRI in 20 patients showed good drainage of the syrinx in 10, fair drainage in eight, and poor drainage in two patients. Clinically, the results were good in 13, fair in six, and poor in three patients after a mean postoperative follow up of three years (range six months to 15 years). One patient who was followed up for 15 years after syringosubarachnoid shunt with a good MRI and clinical result had developed lung carcinoma but was asymptomatic from the syringomyelia. Six patients who had been followed up for less than two years had three good and three fair clinical results. Of the 13 patients who underwent syringopleural shunt, nine had good, two fair, and two poor outcome. For seven patients with syringosubarachnoid shunt, four had good results, two had fair results, and there was one poor result. One patient each had a fair result after omental grafting and cordectomy.

There were no significant differences in the results of surgical treatment of PTS at the cervical, dorsal, or dorsolumbar levels (Yates' corrected χ² test 0.02, P = 0.88; Fisher's exact P = 0.5). Similarly, there was no difference in the surgical outcome of syringomyelia after either complete or incomplete cord injury (Yates' corrected χ² 0.53, P = 0.46; Fisher's exact P = 0.22).

The preoperative extent of the syrinx did not influence the surgical outcome but good correlation (85%) was noted between the postoperative MRI appearance of the syrinx and the final clinical outcome in 18 patients (table 5). Satisfactory (fair and good) reduction in the size of the syrinx as judged by postoperative MRI correlated well with a satisfactory (good and fair) clinical outcome in

<table>
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<th>Clinical outcome</th>
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<tr>
<td>Poor</td>
<td>Fair</td>
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<tr>
<td>1 (OM)</td>
<td>1 (SS)</td>
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<td>1 (SP-OM)*</td>
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<td>1 (SS)</td>
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*Two patients had poor clinical outcome after syringopleural shunt initially but had a fair clinical result after omental grafting.
†Postoperative MRI of patient 11 (cordectomy with a fair result) and patient 16 (syringopleural shunt with a good clinical result) were not available. Ss = syringosubarachnoid shunt; sp = syringopleural shunt; OM = omental grafting.
all but three patients. In the first patient the size of the syrinx had slightly increased after omental grafting, with a fair clinical outcome. In the second patient the size of the syrinx on MRI had not changed noticeably after syringosubarachnoid shunt but clinically the patient had a good result. The third patient had almost complete drainage of the syrinx after syringosubarachnoid shunt on the postoperative MRI but without amelioration of the symptoms, due to pronounced arachnoiditis.

Three patients had a poor outcome after surgery. The first patient discussed above had a poor result, probably due to myelomalacia. There was no improvement in either the sensation or the motor function and the pain had become worse postoperatively. The second patient had undergone four surgical procedures for PTS after incomplete dorsal spinal cord injury. The cyst was drained elsewhere in 1973, followed by posterior decompression and fusion at the same centre in 1985. Syringopleural shunt performed in 1987 had decompressed the syrinx satisfactorily on postoperative MRI but the clinical result was poor, probably due to gliosis and meningeal adhesions. He subsequently underwent omental transposition with a fair final clinical result. The last patient with a poor initial result (due to refilling of the syrinx) after syringopleural shunt in 1989, underwent omental transposition in 1991. Two years later, he had a good MRI result without any recurrence of the cyst but the omentum had partially atrophied. His clinical result was fair but there was mild visceral herniation through the track of the omental flap.

There were relatively few operative complications. Two patients developed air embolism, one requiring abandonment of a syringopleural shunt which was successfully carried out at a later date, and another patient had epileptic fits after syringopleural shunt due to air embolism in the omentum. Neither of these patients had any long term complications related to air embolism. In one patient, the placement of the syringopleural shunt was incorrect and was not draining into the pleural cavity as seen on chest radiographs but he had a good postoperative MRI appearance and a fair clinical result. One patient had methicillin resistant Staphylococcus aureus infection, which was treated effectively with appropriate antibiotics.

Discussion

The true incidence of PTS is probably higher than that documented in our study. Only patients with suggestive symptoms and/or alteration of sensation, motor power, or reflexes on neurological examination had MRI. The overall clinically diagnosed and confirmed incidence of PTS in the present study (3-4%) is, however, higher than previously reported.¹ Many earlier studies were conducted before PTS as an entity was well established and before the general availability of MRI. The present study, although retrospective in nature, represents the incidence characteristics of PTS in a conservatively treated spinal cord injury population with uniformity of the treatment plan of the spinal cord injury centre. A previous review, high index of suspicion of PTS, and MRI in clinically suspicious cases. Most (about 85%) of the patients at our centre were treated conservatively. Therefore our data may not be applicable to patients with surgically treated spinal cord injuries. The trend in the incidence of PTS in the conservatively and surgically treated patients seems to be the same in our study, but the number of patients treated surgically is too small to draw any firm conclusions.

There are no reported cohort studies comparing the incidence of PTS in the patients undergoing conservative and surgical treatment of spinal cord injury. Similarly, there is a scarcity of data on the incidence of PTS in a largely surgically treated spinal cord injury population. It is therefore difficult to recommend initial conservative or surgical treatment of spinal trauma with the aim of minimising the incidence of PTS. Because of the retrospective nature of the present study and the lack of a surgically treated control group, it was not possible to evaluate the role of kyphoscoliosis and instability in the initiation or propagation of the syrinx. There are no studies pertaining to these points in the aetopathogenesis of PTS.

The incidence of PTS in the present study was twice as common after complete cord injury than after incomplete cord injury, in accordance with the findings of some authors⁴-⁷ and in contradiction to those of others⁸-¹⁰ who did not find any correlation between the incidence of PTS and the severity of the cord injury. Injury to the dorsal spinal cord was more commonly associated with the formation of the syrinx than injury to the cervical cord in our study, and some others,²⁻⁵ as supported by the findings of Rossier et al.¹⁰ and Lyons et al.³ who reported higher incidence of PTS after injuries to the cervical cord. The incidence of PTS after complete and incomplete cord injury at each spinal regional level has not been previously reported.

The injury to PTS interval in the complete dorsal and incomplete cervical cord and dor-solumbar injuries was the shortest (table 1), supporting the data of Vernon et al.,¹ but contradicting the findings of Lyons et al.³ who reported much earlier occurrence of PTS after complete cord injury compared with incomplete injury. Why some patients develop PTS within a few months of sustaining the spinal cord injury whereas others may not develop it until several years later cannot be explained from our study, as these patients were not significantly different from others in any way. However, early onset PTS tended to have a more rapidly progressive course and required surgical treatment.

Pain and ascending sensory level were the most common clinical presentations of the PTS in this study, whereas increasing motor deficit, hyperhidrosis, and increased spasticity were present less often (table 2). These find-
ings are in agreement with previous descriptions. Occasionally the syrinx may only descend downwards from the fracture level as seen in two patients (one with a closed fracture, one incomplete with cervical cord injury). Both these patients were diagnosed as having developed PTS within six months of injury against the expectations of late presentation of a descending PTS because they were still undergoing inpatient rehabilitation after the spinal injury.

Hyporeflexic urinary bladder has been previously described in association with PTS in one patient but this was considered to be an aetiological factor by increasing the venous pressure rather than a result of PTS. However, downward extension of the syrinx may render the urinary bladder hyporeflexic as noted in two of our patients, who presented with increased difficulty in voiding, as the sole presenting feature in one patient and in combination with other clinical features of PTS in the other.

Rare but other potentially serious clinical presentations of PTS include bowel dysfunction due to downward extension of the syrinx, which may necessitate a colostomy, and diaphragmatic paralysis due to extension proximal to the C4 level. A partial Horner’s syndrome may be present which may show anisocoria only (B Williams, personal communication).

The clinical diagnosis of PTS should be confirmed by MRI, which gives an accurate non-invasive assessment of the longitudinal extent and the distension of the syrinx. Spinal cord cysts after spinal injury are very common and may be seen on MRI in 51% of patients. They rarely require any treatment. The relevance of these small cysts is not well established and their natural history remains unresolved. Some authors believe that all the patients on conservative treatment for spinal cord cysts will eventually show signs of progression and neurological deterioration if followed up for long enough. On the other hand the neurological status may remain unchanged for years despite the presence or slow progression of the cyst or there may even be an occasional patient showing spontaneous resolution. It is clear from the study of Backe et al that mere demonstration of a spinal cord cyst does not constitute a clinically relevant syrinx. In the present study, the criteria of Tobimatsu et al and Williams were adhered to, which require a cystic cavity to extend over at least two vertebral segments, before a diagnosis of PTS can be made.

Surgical treatment for relatively smaller syrinx of the cervical cord has been advocated to avoid sudden respiratory embarrassment but small lower cervical cord syrinxes were kept under close observation and did not progress appreciably in our study. In the patients requiring surgical treatment MRI delineates the extent as well as the location of maximum distension of the syrinx, thus allowing correct surgical targeting. The technique also evaluates the degree of reactive gliosis and beading, which may prevent total collapse of the cavity and the cavity may refill. The septations, when present, create difficulty in effective drainage of all the loculated cavities.

Surgical treatment usually arrests clinical deterioration and can even lead to reversal of the neurological deficit. Reduction in the severity of pain and motor improvement was found in 89% of patients in the present study and is comparable with that reported by others. Sensory recovery has been reported to be poor in several series by contrast with our experience and that of Lyons et al. Hyperhidrosis reduces in severity but tends to persist in many patients postoperatively. Although it has been suggested that surgical treatment does not have any significant effect on spasticity, four out of five patients with increased spasticity in the present study reported postoperative improvement.

Various surgical procedures have been employed for the treatment of PTS. Cord transection or cordectomy at the level of the syrinx is possible only in the patients with complete lesions and is usually associated with satisfactory reversal of the symptoms of PTS. Drainage of the syrinx into the subarachnoid space or a low pressure system such as the pleural or the peritoneal cavity has been advocated. Although frequent blockage of the syringosubarachnoid shunt has been reported, it has been recommended by some authors and our results with the syringosubarachnoid shunt in seven patients (four good, two fair, one poor) have also been gratifying. The syringoperitoneal shunt has been advocated by some authors but this procedure was not performed in any of the patients in the present study. Instead, the shunt was drained into the pleural cavity in 13 patients with nine good, two fair, and two poor results.

We concur with Umbach and Heilporn that no one shunting procedure is superior to another. The results of different shunts in the present study are not significant, but these results have been published. Comparative studies are desirable. However, most studies are retrospective in nature and lack a control group. Another drawback in comparing the results of various surgical procedures in different studies has been the lack of uniformity of the outcome assessment criteria.

Vascularised omental transposition (one primary and two secondary procedures) stabilised three patients in the present study. It is believed to increase the vascularity of the damaged cord and is useful either as a primary procedure in patients with adhesive meningitis, myelomalacia with cystic changes, or in the patients with failed previous surgery. However, the operative technique is more complex and requires two surgical teams. Williams had mixed preliminary results of omental transposition in his patients and emphasised the need for caution. Omental atrophy, visceral herniation through the potential space of the tract of transposed omentum, and paradoxical increase in the size of the syrinx due to increased vascularity of the cord are some of the problems encountered with omental grafting. We admit however, that the few patients
who were treated with omental transposition does not allow us to draw meaningful conclusions about the efficacy of this operation.

Septated syringomyelia, which presents surgical difficulties in ensuring complete drainage of all the chambers, may be treated by perforating the septa under vision by using a flexible neuroendoscope. Laser myelolocotomy was recently described by Edgar and Quail to obviate the need for shunt placement. The technique involved creating multiple puncture holes throughout the cyst with the help of a laser. However, their preliminary results showed a 50% revision rate due to dural scarring, which obstructed the myelotomy sites.

Other surgical procedures such as the divisions of adhesions and the possibility of creating an artificial meningocele without shunting are said, in some cases, to be as effective.

Good correlation (85%) between the reduction in the size of the syrinx on postoperative MRI and a satisfactory clinical outcome was experienced in our study, in accordance with previous reports. However, complete or nearly complete drainage of the syrinx was not necessary for a good clinical outcome, which was possible even with a mild to moderate reduction in the size of the syrinx. Refilling of the syrinx in the presence of a persistent clinical improvement and good decompression of the syrinx in the presence of a poor clinical result due to arachnoiditis or myelomalacia were the factors identified in the mismatch between the clinical outcome and final MRI appearance of the syrinx.

We agree with Pilay et al. that it is difficult to predict the prognosis on the basis of the clinical features or preoperative MRI appearance. In our experience, preoperative MRI assessment of the size of the syrinx is not a useful predictor of the results of surgical treatment and a large syrinx is not necessarily associated with a poor result. Although the length of syringomyelia in the present study was higher than the previously reported mean longitudinal extent of the syrinx from 3-3 to 8-5 segments the results were comparable with other studies. The measurement of the absolute cord size at the level of the maximum distension of the syrinx is subject to considerable error and it is not helpful in predicting the outcome after surgery.

A further longitudinal study of similar size of population using MRI would yield very valuable information. This is in view of a recent unpublished study (D Wang, personal communication) at Stoke Mandeville Hospital, in which it was suggested that the incidence could be about 20%. Due to the relatively small number of spinal injuries (10–15/million/year) however, the cooperation of other spinal injuries centres would be required.

We are grateful to the late Mr Bernard Williams, consultant neurosurgeon, Smetwick, Birmingham, who performed the surgery on these patients, Drs I McCall and V Pullicino, consultant radiologists at the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, who have helped us with the radiological evaluation, and Drs M Tsu and S Katoh of the University of Tokushima, Japan for their help and support. This work was part of a thesis submitted by AB to the University of Liverpool, August 1993.

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