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Clinical features, investigation and treatment of post-traumatic syringomyelia

NORMAN SHANNON, LINDSAY SYMON, VALENTINE LOGUE, DAVID CULL, JOON KANG, AND BRIAN KENDALL

From the Gough Cooper Department of Neurological Surgery, Institute of Neurology and the National Hospital, Queen Square, London

SUMMARY Thirteen patients who sustained spinal cord trauma causing persisting disability, developed new symptoms, the chief one of which was severe pain unrelieved by analgesics. The clinical diagnosis of post traumatic syringomyelia was confirmed in each case by means of myelography, as well as endomyelography in seven patients. In every case exploration of the spinal cord syrinx was performed. Ten patients were troubled by severe pain while three patients were mainly subject to altered sensation in the upper limbs. Of the six patients who had initially sustained complete cord transections, three were treated by cord transection and three were treated by syringostomy. The seven patients who sustained incomplete cord lesions were all treated by syringostomy. The patients who initially sustained incomplete sensory motor spinal cord damage had a better symptomatic response to surgery than those who had sustained a complete spinal cord lesion. The ten patients whose main symptom was severe pain were completely relieved of their symptoms by surgery.

The development of a progressive neurological deficit extending to segments beyond the site of cord injury, months or years after the original trauma and shown subsequently to be due to the development of a syrinx cavity within the cord, is a rare but well documented occurrence.[1-3 6-9] Barnett[7] found clinical features suggesting syrinx formation in 1·8% of a series of 319 post-traumatic paraplegic patients. On the basis of the similarity of clinical manifestations to those seen in syringomyelia, he concluded that the lesion was a cystic degeneration of the spinal cord, although such a cyst was confirmed in only two of his eight cases, in one at operation and in another at necropsy.

Our recent experiences suggest that some of the features of post-traumatic syringomyelia are amenable to surgical intervention and the purpose of this paper is to call attention to this relatively rare complication of spinal cord trauma, and the possibility of partial relief.

Address for reprint requests: Professor Lindsay Symon, Institute of Neurology, Queen Square, London WC1 3BG.

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Method of study

Twenty case records with the diagnosis of post-traumatic syringomyelia were examined. Seven of the patients had been judged unsuitable for further investigation and potential surgery. This paper is concerned with those in whom investigations and surgery seemed warranted. In the course of the study all patients returned to hospital for re-examination and assessment, and all neuroradiological studies were re-examined in the light of current practice.

Observations

Clinical features The details of the initial injury sustained by the 13 patients in the study are given in table 1. A summary of the clinical findings in patients with complete and incomplete lesions is given in tables 2 and 3. The term "complete cord lesion" is used to describe patients who remained paraplegic, and up until the development of new symptoms, had a cleary defined sensory level on clinical testing. An "incomplete cord lesion" was one in which the patient regained lower limb power, but remained paretic. The term "dissociate sensory loss" describes a state wherein the patient had objective sensory loss or impairment of sensation of hot and cold stimuli, but touch was preserved. The clinical features of post-traumatic syringomyelia did not differ from those of

Table 1 *Details of the initial injury sustained by the patients*

Cases	*Date of injury*	*Mode of injury*	*Spinal column injury*	*Neurological deficit*	*Initial management*
1 JM 21M	November 1975	Road traffic accident	Fracture dislocation C7 on D1	Tetraplegia improved to paraparesis	No operation
2 JH 40M	January 1976	Fall	Crush fracture L1	Paraplegia improving to paraparesis	No operation
3 RP 40M	June 1962	Fall	Crush fracture L1	Paraparesis Ambulant	No operation
4 BH 47M	April 1957	Road traffic accident	Posterior dislocation C6 on C7	Paraparesis Ambulant	No operation
5 JR 22M	August 1972	Thrown from horse	Crush fracture D6	Paraplegia. No improvement	No operation
6 KT 37M	May 1972	Road traffic accident	Crush fracture D7	Paraplegia. No improvement	No operation
7 RH 25M	July 1971	Road traffic accident	Crush fracture L1. Posterior dislocation of D12	Paraplegia. No improvement.	No operation
8 RS 46F	June 1951	Gunshot injury	D7, D1 dislocation	Paraplegia. No improvement	Exploration D7, D1 Laminectomy
9 GV 51M	June 1975	Road traffic accident	Fracture L1	Paraplegia. No improvement	No operation
10 AB 41M	September 1975	Road traffic accident	Fracture D12	Paraparesis Ambulant	No operation
11 DK 49M	1955	Industrial accident	Crush fracture D12	Paraparesis Non-ambulant	Plate fixation of fracture
12 KA 59F	June 1966	Gunshot wound	Laminae of C8 and C7	Initially tetraplegic improved to weakness upper limbs limited hand function. Paraplegia	D1/D12 laminectomy Wound explored
13 MP 53M	September 1965	Road traffic	Fracture D7	Paraparesis improved to spastic weak left lower limbs. Ambulant	Wound explored. Extradural and intradural haematoma removed

Table 2 *Patients with complete cord lesions after trauma*

Name/age at time of injury	*Column level*	*Interval to new symptoms*	*New symptoms*	*New motor and/or sensory signs*	*Surgical treatment*	*Results*
5 JR 22M	D7	2 years	Numbness with loss of pain and temperature sensation, L upper limb	Dissociate sensory loss C4 to D4 left side	January 1975 Laminectomy D4, D5, D6. Syringostomy Pudenz catheter	June 1979. No change
6 KT 37M	D7	5 years	Loss of sensation of pain and temperature left trunk and left arm	Weakness of left finger flexors and intrinsic muscle. Dissociate sensory loss left side D6 to C3 left	D4, D5, D6 laminectomy. Cord sectioned through syrinx sparing anterior spinal artery	Sensory level fell to C7 left side
7 RH 25M	L1	6 years	Pain right hypochondrium R neck and chest	Dissociate loss C3 to D5 right	April 1979. C4 to D7 laminectomy Syringostomy Pudenz catheter	August 1979. Relief of pain
8 RS 46F	C7	21 years	Pain left face. Rise in numbness to C2 left C4 right. Weakness in hands	Dissociate loss C7 to D2 left. Weak intrinsic muscles in hands	October 1978. Re-exploration of C7 and D1 region. Cyst found and syringostomy performed	June 1979. Developed headaches. No change.
9 GV 45M	L1	5 years	Pain on coughing side of neck R hand and R chest wall	Right C3 to D10. Dissociate loss	June 1975. D11, D12, L1 laminectomy. Cord transected through cyst at D10 sparing the anterior spinal artery	May 1979. Pain relieved 18 months and returned
12 KA 59F	D1	8 years	Pain left neck, shoulders, arm increasing weakness left hands	Dissociate loss left L2 to C8. Wasted triceps and hand intrinsic numbers	D3, D4 laminectomy. Cord incised in two places sparing anterior spinal artery	Improvement in power left arm 12 months post-surgery

Table 3 *Patients with incomplete cord lesions after trauma*

Name/age at time of injury	*Column level*	*Interval to new symptoms*	*New symptoms*	*New motor and/ or sensory signs*	*Surgical treatment*	*Results*
1 JM 21M	C7	3 years	Pain left upper limb and left chest wall. No sweating in these parts	Dissociate sensory loss C2 to D3 left. Weak extension elbow left MRC Grade 3. Anhidrosis C2 to D3 left	November 1978 C7 to D4 laminectomy. Syringostomy and Pudenz catheter	March 1979. Pain relieved. Power improved Sensory signs unchanged
2 JH 40M	L1	2 years	Pain right upper limb. Numbness right side of face	Dissociate Déjerine loss R trigeminal plus R C2 to L1 and left C3 to L1. Weak hand, intrinsic muscles	August 1978. D11, D12, L1. Laminectomies Pudenz catheter. Syringostomy	June 1979. Pain relieved completely apart from occasional twinges of facial pain. Sensory signs unchanged
3 RP 59M	L1	12 years	Bilateral mid dorsal girdle pain. Deterioration of power in lower limbs to MRC Grade 3	Sensory loss dissociate D5 to D8. Able to elevate lower limbs off couch 1 minute only	January 1976. D10 to L2 laminectomy. Pudenz catheter. Syringostomy	April 1976. Pain relieved. Power in lower limbs unimproved
4 BH 47M	C6	12 years	Pain both arms L face weakness increasing lower limbs	Déjerine L face. Dissociate loss down to D7. Grade 4 weakness upper limbs	June 1978. C7, D1. Laminectomy. Syringostomy	July 1979. Pain relieved. Power deteriorated lower limbs for 2 months then returned to pre-operative state
10 AB 42M	D12	19 years	Right upper limb pain pain on coughing. Loss of pain, temperature, right hand	C4 to D10 dissociate loss. Weak R hand intrinsic muscles	October 1978. D8 to D11 laminectomies	August 1979 pain relieved
11 DK 49M	D12	14 years	Paraesthesia right hand weakness numbness right hand	Impaired light touch L2, L3 right. Dissociate sensory loss RV to D8. Weakness of extensors	D10, D11, D12 laminectomy Syringostomy D10	Improvement in power right upper limb, 1978
13 MP 53M	D7	7 years	Severe pain. Numbness left arm and upper trunk. Weakness left upper limb	Global weakness left upper limb especially shoulder girdle muscles. Dissociate loss left C2 to D11	C6 to T3 laminectomy Syringostomy through Pudenz catheter draining into subarachnoid space	Improved gait. Pain relief. Improved power left upper limb, 12 months post-operatively

patients who have syringomyelia from other causes. The presenting symptoms occurred in varying combinations of pain, weakness, hypaesthesia and dysaesthesia, involving usually one or both upper limbs and the trunk. The period between developing symptoms and investigation ranged from six months to six years, but in five of the cases was less than 18 months. Pain in many cases began in the neck and radiated into the arms. It was often initiated or made worse by coughing, sneezing or straining, and the distribution of the pain corresponded to the areas affected by dysaesthesia and sensory impairment. Two patients had impairment of trigeminal sensation and one had a unilateral partial Horner's syndrome. In none of the cases in whom the initial paraplegia was incomplete, was there worsening of the lower limb sensory or motor loss, when superadded symptoms of syrinx occurred. The dermatome symbols used are those of the MRC handbook[10] on peripheral nerve injury and the muscle power grades are on the MRC scale 0–5.

In table 2 details of new clinical symptoms and signs occurring in patients who sustained complete cord lesions are set out, while table 3 gives the same kind of information on patients who sustained incomplete lesions.

Radiology Plain radiographs were obtained and myelography was performed in all cases. The contrast medium used was Myodil (7) or metrizamide (6). Endomyelograms[11 12] were performed in six cases, usually through midline punctures, near the lower end of a cord swelling in order to penetrate as few functioning fibres as possible (fig 1). One case was punctured laterally at C2 level. If no cord swelling was present, a puncture would be made near the site of the trauma. Cranial Computed Tomography (CT) was performed on two cases, before and after intravenous contrast medium. Spinal CT using the EMI 5005 machine was performed after combined water soluble myelogram and endomyelogram in two cases (fig 2).

The results of the radiological studies are tabulated in table 4.

Surgical treatment

In 1892 Abbe[13] demonstrated at necropsy how syringostomy could be performed. The subject

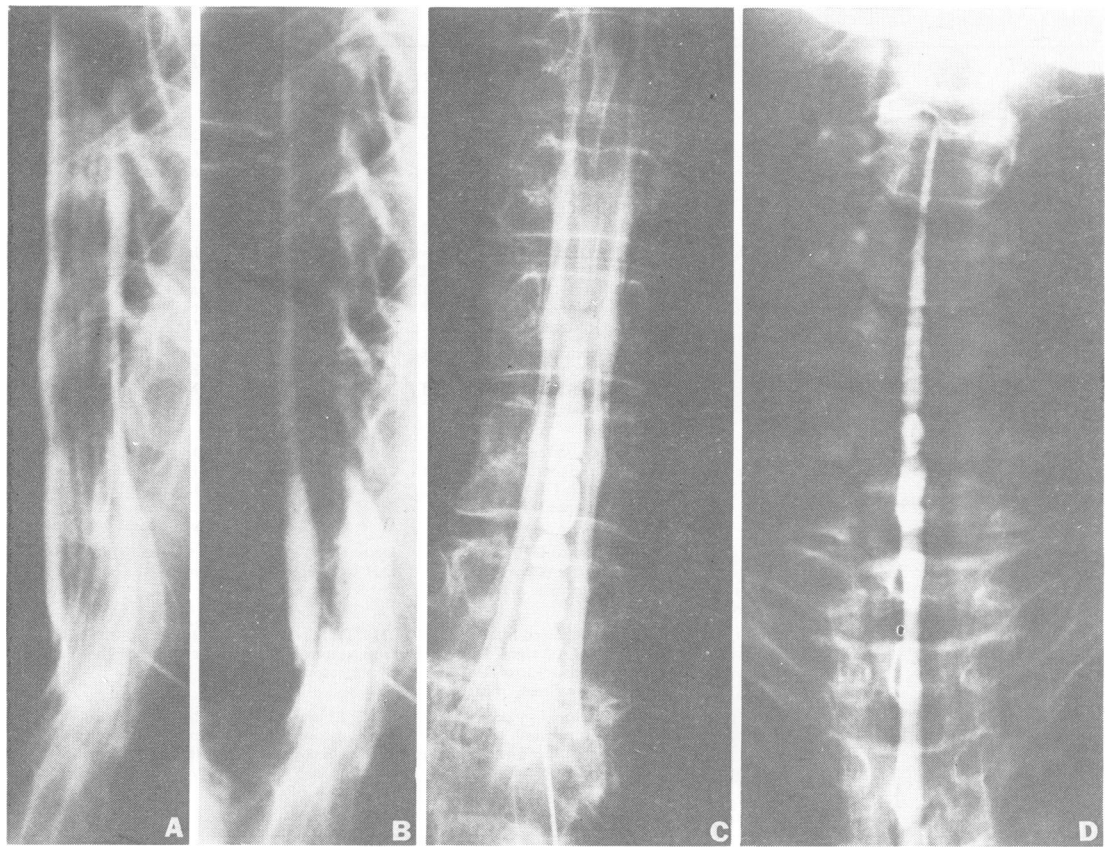

Fig 1 *Post-traumatic syringomyelia associated with compression fracture of D12. Combined myelogram and endomyelogram. (a) Lateral projection. Puncture at D11/12. Contrast medium injected into subarachnoid space showing the expansion of the lower dorsal cord with obstruction at D12 level. (b) Needle advanced to puncture syrinx into which a small amount of contrast medium has been injected. (c) and (d) There is expansion of the lower segment of the spinal cord with syrinx extending to second cervical level. The lobulations are typical of a post-traumatic syrinx widest adjacent to level of trauma.*

had syringomyelia not associated with trauma. Elsberg[14] gave the first description of syringostomy on "one case of hydromyelia and intramedullary cyst formation and one case of syringomyelia with marked glial formation." Further reports of syringostomy were given by Pusepp[15] and Frazier[16]. However, Freeman[4,5] must be given credit for rationalising surgical treatment after performing experimental cord concussive injuries on cat and dog spinal cords. He showed that syrinx formation frequently followed experimental trauma to the spinal cord of cats and dogs and, after a series of controlled experiments, demonstrated that blunt myelotomy allowed the egress of necrotic material from the cord and reduced the likelihood of syrinx formation. By 1959 he had extended his experimental findings and used syringostomy to treat a patient with post-traumatic syringomyelia. Love and Olofsen[17] elaborated the operation by inserting tantalum wire into the syrinx.

In the present series the two forms of surgical treatment employed were cord transaction and syringostomy. In the cases where cord transection was performed particular care was taken to spare the anterior spinal artery and emergent roots immediately above the level of transection. This dissection in the presence of arachnoiditis at the level of injury may be difficult and where there is residual cord function clearly is inappropriate. In the cases treated by tube syringostomy, dissection was performed through the thinned posterior

columns under the microscope to expose the glial cavity, and a small Pudenz ventricular catheter, led into this cavity cephaled for a distance of four to five cm, was then anchored to the arachnoid or to the dura by an encircling stitch. The multiperforate end of the catheter was then carefully placed in the subarachnoid space distal to the site of syringostomy. These operations were performed some segments proximal to the level of trauma, and usually in the upper to mid dorsal region. The cord is easily accessible here and disturbance of hand segments is avoided. The usual operation finding was of a considerably dilated cord which often collapsed with respiration. Occasionally where the cord appeared only a little expanded, knowledge of a central cavity shown by endomyelography was of considerable help in justifying dissection in the approximate midline. The advantage of a choice of a mid-dorsal position for syringostomy is that joint position sense in the legs is, in our experience, not usually at risk. The cysts encountered in this series were usually lined by grey gliotic tissue though occasionally there was a more diffluent gelatinous appearance. The aspirate from the cyst material was in 10 of the 13 cases clear and indistinguishable from CSF. Where chemical examination was performed the protein level of the cyst fluid was that of the accompanying CSF; in two cases the cyst contained yellow fluid with a high protein content. The position of the syringostomy tube in the distal theca was arranged under the microscope to ensure close relationship to the denticulate ligament, and in one case two tubes were placed in a large syrinx,

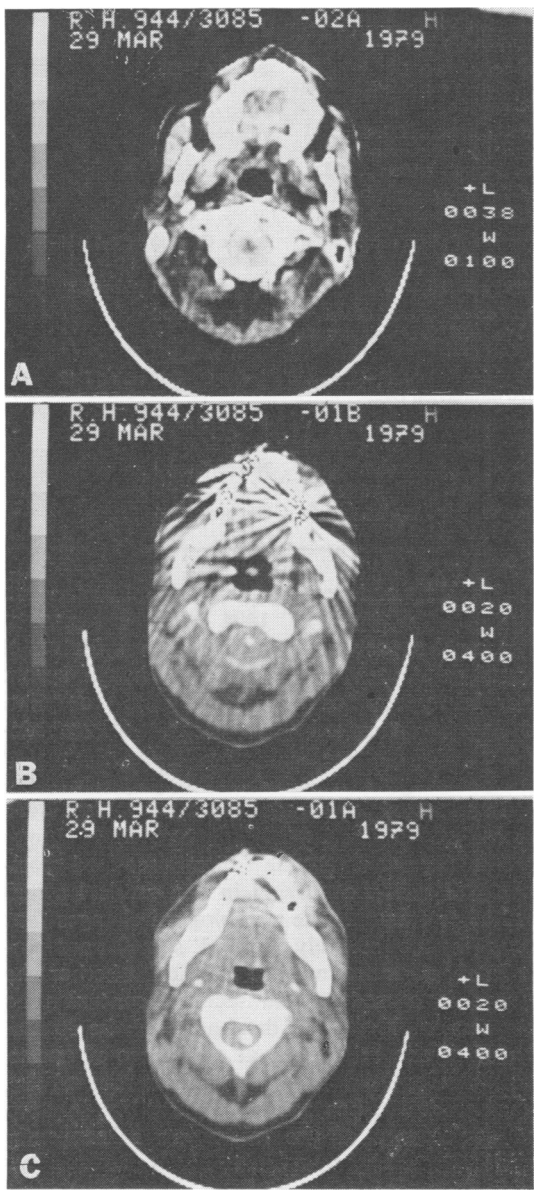

Fig 2 *Computed tomogram after myelogram and endomyelogram. (a) At the level of the upper part of C1 the cord is small and the syrinx is absent. (b) and (c) At lower border of C1 and (c) at C2 the syrinx is present and expands the cord.*

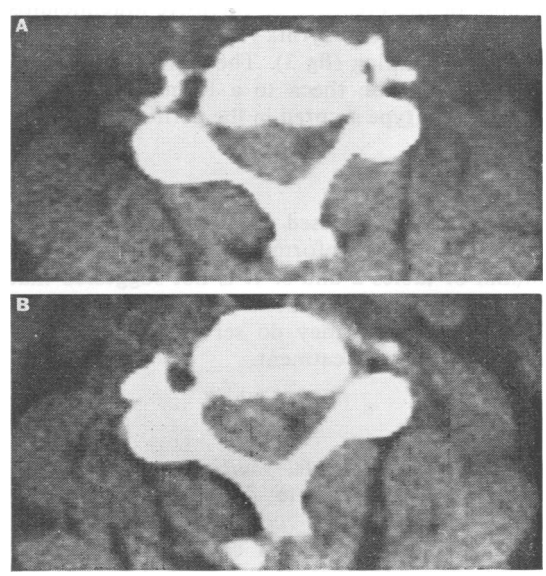

Fig 3 *Post-traumatic syringomyelia following fracture of body D5. CT of spine at C4 and C5 levels shows syrinx extending through the right side of the cord.*

Table 4

Cases	*Plain x-ray*	*Contrast*	*Abnormalities*	*Endomyelogram*
1 JM	Fracture C7 Dislocation C7/T1	Metrizamide	Obstruction C7 Expansion lower cervical and upper dorsal cord	Syrinx C1–D3
2 JH	Crush fracture L1	Metrizamide	Partial obstruction of L1 Expansion of cord D10–D12/L1	Syrinx D11–L1
3 RP	Crush fracture L1	Myodil	Obstruction L1 Expansion of whole cord	Not performed
4 BH	Posterior dislocation C6 on C7	Metrizamide	Cord compression C6 Expansion of cord C3–D2 Arachnoiditis	Syrinx C3–D5
5 JR	Crush fracture D6	Myodil	Partial obstruction D6. Cord compression D6. Expansion cervical and thoracic cord to D8	Not performed
6 KT	Fracture D7	Myodil	Partial obstruction D7. Cord dilated to upper cervical region	Not performed
7 RH	Crush fracture L1. Posterior discoloration D12	Metrizamide	Expansion of whole cord. Partial obstruction D12	Syrinx C2–D12 widest D11–12. CT shows syrinx up to C1
8 RS	C7/D1 dislocation	Metrizamide	Atrophy cervical cord	Syrinx C1–D11 widest C7
9 GV	Fracture L1	Myodil	Expansion cervical and dorsal cord, varied slightly with posture. Arachnoiditis.	Not performed
10 AB	Fracture D12	Metrizamide	Expansion of cord D10–12. Partial Partial obstruction D12.	Syrinx C2–D10 widest D12
11 DK	D12	Myodil	Partial obstruction D12. Marked increase in width of cervical cord	Not performed
12 KA	D8	Myodil	Complete obstruction C7. Cord wide C3 to C7	Not performed
13 MP	Collapse body of D7	Myodil	Expanded cord C3 to C8. Local arachnoiditis at D8	Not performed

directed proximally and distally. In two more recent cases (not included in the present series, because of the presence of extensive arachnoiditis in the theca), the syrinx was demonstrated by plain CT scanning (fig 3). The syringostomy tube was led from the theca to a Raimondi valve of low pressure type secured in the peritoneal cavity.

Clinical assessment

Patients were reassessed after varying periods of follow up and this information is given in the last column of tables 2 and 3. It is not suggested that these objective assessments represent the final clinical state, but they do serve to indicate the value of surgical treatment.

Discussion

The term syringomyelia was introduced in 1827 by Oliver d'Angers[18] to describe a glial lined cavity in the spinal cord which may communicate with the central canal or the subarachnoid space. In 1871 Hallopeau[19] described the necropsy appearances of unconnected cystic lesions in the spinal cord following trauma while only six years later the first description of spinal arachnoiditis and syringomyelia after trauma was given by Joffroy.[20] In 1904 Minor[21] described degenerative vascular changes in the spinal cord following trauma and postulated that a syrinx after injury resulted from incomplete resorption of degenerate spinal cord tissue. Holmes[3] described, for the first time, an unspecified number of cases of gunshot wounds damaging the spinal cord and noted intramedullary cysts arising from the primary spinal injury and ascending up four or five segments. Strong[22] first described post-traumatic syrinx occurring in the cervical spinal cord after trauma. The pathological changes that occur in the severely injured spinal cord immediately after trauma are identical whether the trauma is caused by transection, contusion or compression. Kao[23] has demonstrated the formation of myelin microcysts at a distance of one to two mm from the experimentally transected dog spinal cord. He has postulated that the rupture and coalescence of the microcysts subsequently leads to syrinx formation. Syrinx formation seems to be associated with an abortive attempt at regeneration. It is likely that this process of microcyst formation occurs in contused, compressed and transected spinal cords resulting in the egress of fluid from the damaged axons.

Microcyst formation with coalescence of microcysts may occur in the damaged human spinal cord shortly after the time of injury, even though some years will elapse before the syrinx produces symptoms. However, not everyone agrees that Kao's observations in spinal cord injuries in dogs can be used entirely to explain cyst formation in the human subject. It has been postulated that the rise in central venous pressure which occurs during straining, sneezing and coughing is transmitted to the epidural veins so as to alter intracystic tension, resulting in extension of a cyst. While it is well known that deterioration in the neurological status of syringomyelia sufferers can occur after sneezing and coughing there was no evidence of this in any of the cases in this series, although pain was exacerbated by such neurones.

In none of our cases was myelographic abnormality of the posterior fossa and cervicomedullary junction discovered. No communication between the syrinx and fourth ventricle was demonstrable radiologically and at operation there was no sign of communication between the syrinx and the subarachnoid space. Our findings accord with the detailed necropsy description of two cases of post-traumatic syringomyelia by Jensen and Reske-Nielsen.[27] However, there are other possible causes of syringomyelia.[24 25]

There have been many reports in the literature describing the results of syringostomy in small series of post-traumatic syringomyelia.[5 6 9] Laha *et al*[26] described in three cases. In 1977 Jensen and Reske-Nielsen[27] reviewed 32 published cases of post-traumatic syringomyelia and noted that eight of 13 operated cases were improved. However, in the same year Lacert *et al*[28] reviewed 35 cases described in the literature including seven cases of their own. Although only two cases were subjected to operation they concluded that neurosurgical treatment did not modify the spontaneous evolution of the condition. It is very difficult from the published literature to form any clear idea as to the benefits to be gained by performing syringostomy on patients suffering from post-traumatic syringomyelia and it is certainly not possible to make any statement as to the effect of syringostomy on the natural history of the condition. From our relatively large series of cases we have drawn the conclusions that follow.

Conclusions

Post-traumatic syringomyelia is a rare sequela of spinal cord trauma of unknown incidence. However, it is important for the affected individual that the diagnosis be made. This series of cases shows that accurate delineation and the extent of the syrinx can be made by myelography and CT scanning. Syringostomy seems to be a predictable and simple method of relieving pain and carries a low morbidity. Sensory symptoms do not seem to be altered by syringostomy. Further examination of the cases described here will indicate whether the natural history of the condition is significantly altered by surgery.

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