Paraplegia 19 (1981) 96–97 0031-1758/81/00350096802.00

Summary of paper given at the Annual Scientific Meeting of the International Medical Society of Paraplegia 1980

PROGRESSIVE LATE POST-TRAUMATIC SYRINGOMYELIA

By ALAIN B. ROSSIER, M.D.[1], DOMINIC FOO, M.D.[2], JOHN SHILLITO, M.D.[3], MOHAMMAD H. NAHEEDY, M.D.[4], WILLIAM H. SWEET, M.D.[5], FRANCES DYRO, M.D.[6], and MEHDI SARKARATI, M.D.[7]

Spinal Cord Injury[1, 2, 7] and Rehabilitation Medicine[6] Services, West Roxbury VA Medical Centre, Dept. of Orthopaedic Surgery[1], Dept. of Neurosurgery and Surgery[3, 5] and Div. of Neuroradiology, Dept. of Radiology[4], Brigham and Women's Hospital, and Massachusetts General Hospital, Harvard Medical, Boston, Massachusetts.

Key words: Spinal cord injuries; Syringomyelia; Air myelography.

BETWEEN May 1, 1973 and March 30, 1980, 659 spinal cord injury patients were admitted to our centre. During that period the same patient may have been hospitalised several times. However, he was counted only once. Syringomyelia was diagnosed in six male patients, *i.e.*, an incidence of 0.9 per cent close to Barnett and Jousse's figure of 1·2 per cent (Barnett and Jousse, 1973). In comparison to the incidence of true syringomyelia which ranges from 0·01 per cent to 0·4 per cent (Brewis *et al.*, 1966; Schliep, 1979), this ratio is high and militates against a congenital form of syrinx. The world literature was reviewed and in addition to the present six patients of the author, 143 cases of syrinx secondary to a fractured spine with spinal cord damage were gathered. Since identical cases happened to be reported several times in different journals, particular attention was paid not to count them twice. Syringomyelia was diagnosed in our six patients 5 to 23 years (mean: 13 years) after the spinal cord injury. Patients were between 19 and 45 years old at the time of injury (mean: 29 years). The initial neurological deficit was sensory motor complete in two patients, sensory incomplete (sacral sparing) but motor complete in three patients and sensory motor incomplete in the remaining patients. The very initial symptom was pain in three patients and numbness followed by pain in the other three patients at or about the level of injury six months to 20 years post-injury (mean: 9 years). The first signs of muscle weakness appeared 2 to 9 years later (average: 4·5 years). Only in two patients did paresia follow within a few weeks of patient's subjective sensory complaints. In four of the six patients pain was elicited by coughing, sneezing and straining. The diagnosis of syrinx was suggested by the presence of an ascending and dissociated sensory level up to C2, however, with an initially 'suspended' normal sensory zone in three of the six patients. In one of these three patients 3 years after the onset of the symptoms the suspended normal zone started to fade out and became analgesic over the next 2 years. The ascending level was on the right and left sides in two patients each and was asymmetric bilaterally in the remaining two patients. Electrically there was preserved sensory potential in the face of lost sensation and a decrease in the amplitude of the motor response. Needle EMG showed evidence of anterior horn cells loss with some fasciculations and

fibrillations. Motor units were generally of high amplitude and the interference pattern was markedly reduced. In all the patients the diagnosis was confirmed by the presence of a dilated cord in gas myelography which was suggestive of a non-communicating syrinx in five patients. In the last patient at the end of the examination the complete disappearance in the head down position of the initially marked C2–C6 cord enlargement strongly raised the possibility of a communication between the syrinx and the fourth ventricle, an infrequent finding in this post-traumatic cavitation group of patients. Brain CAT scan was within limits in four of six patients where this examination was carried out, including the patient with the communicating syrinx. Air myelogram accurately demonstrated the area of maximal enlargement of the syrinx. Due to the intramedullary hydrodynamic changes which took place during the head up and head down positions in two patients, it also allowed one to 'visualise' the extent of the cavitation within the cord. The syrinx was located in the cervical region in four patients and extended from the cervical to the mid-dorsal cord in the other two patients. Decompression of the cyst was carried out in four patients with a *Y* or *T* silastic tubing and with a silastic sponge in the remaining one. The cysts were entered and drained between the posterior columns. The lowest protein contents of the cystic fluid which was clear in four patients was 35 mg per cent. Surprisingly the highest value (167 mg per cent) was found in the clear fluid of the last patient whose air myelogram studies suggested a communicating syrinx. Decompression was followed by prompt relief of pain in all patients. Motor recovery was seen in two patients without change in the sensory level whereas a major motor and sensory recovery occurred in one patient, whose EMG also showed improvement in the number of motor units on volition and a reduction in fibrillation potentials. In another patient there was sensory improvement with motor deterioration $1\frac{1}{2}$ years later. Re-operation was followed by motor improvement. In the remaining patient an early major motor deterioration alone in the left upper extremity took place which was thought to be possibly related to too large a size of silastic tubing. Unhappily, re-exploration with partial removal of the tubing did not improve the motor loss which remained complete. Two patients with initial dorso-lumbar and dorsal cord injuries, respectively, also developed in the left shoulder a Charcot's joint which was discovered 4 and 5 years, respectively, after the syrinx was diagnosed.

This study demonstrates the long interval between the very first onset of symptoms and the diagnosis of the syrinx, the value of pain and numbness symptoms, especially following an increase of the intra-abdominal pressure, the late appearance of muscle weakness, the importance of a normal 'suspended' sensory zone within a dissociated sensory area, and the role of ascending sensory dissociation as initial feature. The diagnostic importance of air myelography is stressed.

REFERENCES

BARNETT, H. J. M. & JOUSSE, A. T. (1973). Syringomyelia as a late sequel to traumatic paraplegia and quadriplegia—clinical features. In *Syringomyelia. Major Problems in Neurology*. Editors H. J. M. Barnett, J. B. Foster and P. Hudgson, Saunders, London, Vol. 1, pp. 129–153.

BREWIS, M., POSKANZER, D. C., ROLLAND, C. & MILLER, H. (1966). Neurological disease in an English city. *Acta Neurol. Scand.*, **42**, suppl. 24.

SCHLIEP, G. (1979). Probleme der Syringomyelie. *Fortschr. Neurol. Psychiat.*, **47**, 557–608.