Paraplegia **19** (1981) 389–390

ABSTRACTS OF SELECTED PAPERS

Thoraco-lumbar spine fractures—results of treatment, by W. R. Osebold, S. L. Weinstein and B. L. Sprague. *Spine* (1981), **6,** pp. 13–34.

The authors present their experience over a 6-year period in the management of thoraco-lumbar spine injuries. The review includes only those who had either neurological deficit or were considered radiologically to have posterior spinal column disruption. Of 63 patients who were selected for this study clinical details of 12 were obtained indirectly since they could not be reviewed. Only seven patients were treated conservatively, five of whom had cord lesions. Eighteen patients were treated by fusion and instrumentation and 17 by laminectomy in addition to fusion and instrumentation. Twelve patients were treated by laminectomy alone, four by fusion alone and the remaining five by a combination of the two procedures without instrumentation. Sixteen patients had their primary surgical procedure carried out on the day of injury. Sixty-five per cent of all the operated patients complained of postoperative pain at follow-up. Of 20 patients whose initial neurological deficit was Frankel Grade A only three made some improvement post-operatively (two Grade B and one Grade C). Overall, 25 out of 56 patients (45 per cent) who had some form of surgery to their spines, and six out of seven patients treated conservatively, made neurological gains at the end of treatment.

The authors have drawn 14 conclusions from this well-written documentation. The increased incidence of postoperative pain in their series is most likely due to the selection criteria of the clinical material.

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Resemblances and differences between traumatic and non-traumatic acquired paraplegia,* by A. Heilporn, P. Khoubesserian, P. Dossin and I. Umbach. Brugmann Hospital, Brussels, Belgium.

* (Summary of paper read at the Annual Meeting of the I.M.S.P., Beekbergen, July 1980.)

One hundred and five patients with spinal lesions were admitted during 1978–79, 77 after trauma, 28 due to disease. The traumatic cases are younger and the proportion of male patients higher.

The level of neurological disturbance was commonest in the mid-thoracic region for diseases of the spinal cord, and in the cervico-thoracic and thoraco-lumbar junctions after trauma. More severe lesions are found after trauma. In the heterogenic group of diseases vascular lesions are the more usual.

The possibilities of recuperation are very similar in the two groups, but three medical cases worsened. Complications of pain and chronic urinary tract infections are more frequent after disease than after trauma. On the other hand, P.O.A. and urinary calculi were found only in traumatic cases.

The results of rehabilitation are better after trauma and the prognosis is easier to make. Patients with disease of the spinal cord require a longer period for rehabilitation and hospitalisation.

Effect of trauma dose on spinal cord edema, by F. C. Wagner and W. B. Stewart. *Journal of Neurosurgery* (1981), **54,** pp. 802–806.

Adult cats were subjected to impact trauma of various intensities to determine how changes in trauma magnitude affect the formation and distribution of oedema within the spinal cord in the first 8 hours post-injury. The cord segments corresponding to the T5–T7 vertebrae were exposed by laminectomy and an impact injury was inflicted at T6 by dropping a weight down a vented tube on an impounder resting on the exposed dura. Fourteen cats were injected with fluorescein-labelled albumin 10 minutes before having the spinal cord subject to 260, 360, 500 or 700 gm-cm injury. Cats were sacrificed 8 hours after trauma. Twelve cats were injected with fluorescein-labelled dextrans of 20·000, 40·000, 70·000 or 150·000 molecular weight 10 minutes before receiving a 500 gm-cm cord impact. They were sacrificed 8 hours after trauma. Serial cord sections from both groups were carried out to study the pattern of fluorescent spread which was evaluated by fluorescence microscopy. Nine cats were subjected to an impact injury of 260, 360, or 500 mg-cm at T6 and 8 hours after trauma 1-cm cord sections were assayed for dry weight.

Five hundred and 700 mg-cm trauma doses sufficient to produce a permanent paraplegia resulted in not only tissue damage in the area of impact, but an extension of tracers and increases in tissue water rostrally and caudally from the site of impact. In contrast, the lowest trauma dose of 260 mg-cm which produced only transient paraplegia was not followed by increased water or extravascular fluorescence outside the region of trauma. The longitudinal distribution of increased tissue water was found to be consistent with the distribution of fluorescent markers. The findings of this study demonstrate that the longitudinal spread of post-traumatic oedema is directly related to the amount of initial trauma.

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