Journal of Neurology, Neurosurgery, and Psychiatry 1986;**49**:844–846

Matters arising

The ominous discoloration of the spinal cord due to thoracic disk protrusions: a historical note

Sir: Hulme[1] presented a very serious sign of thoracic disk protrusions. The standard laminectomy used in the past[2–7] for removal of thoracic disk protrusions has given way mercifully to a variety of approaches all designed to avoid further encroachment or damage to a cord compressed by a hardened or calcified bit of cartilage.

The case we present was described 20 years ago; we believe the colored photograph (fig) confirming Hulme's description is unique and bears duplication. We have seen this appearance of the cord but once; since 1967 we have removed thoracic disks by a thoractomy or costotransversectomy approach. We believe this discoloration of the cord represents inadequate perfusion, with necrosis; its manipulation, no matter how gentle, leads to total and permanent paraplegia below the level of the surgery.

A 47-year-old male had numbness of his legs for three years thought to be due to "alcoholic neuritis". His left lower extremity was analgesic; the right lower extremity was very weak. Beevor's sign was upward and the right plantar reflex was extensor. No calcification of the interspace was noted on the plain radiographs; myelography disclosed a complete block opposite the body of T10. The cerebrospinal fluid contained 0·42 g/l protein. Laminectomy at T9-10 disclosed large distended veins on the dorsal surface of the cord with a "very angry" bluish discoloration (fig). No lesion was found in the gutters of the spine extra—or intradurally. The lesion was thought to be an angioma; the dura was left open and the wound closed. After operation he could move his toes only. Reoperation 48 hours later with radical rotation of the cord at the site of the bluish discoloration disclosed a sequestered hardened disk embedded in the anterior substance of the cord, one inch below the interspace, with no visible tear in the posterior longitudinal ligament. The disk was removed. His flaccid paraplegia postoperatively became spastic. There was no return of bowel or bladder function.

Mr Hulme's case[1]

This 40-year-old woman had a weak numb right leg and urgency of micturation for 3 months. She was spastic in her right leg and there was impaired joint and vibration sense both legs. The weakness and spasticity in her left leg got worse although her right leg improved slightly. Calcium was noted at the 9th interspace and "not appreciated". Repeated lumbar punctures showed no block but an elevation in the protein level of the cerebrospinal fluid. Later there was severe burning in the region of the left iliac spine; a repeat myelogram showed a "rounded bony density opposite the T9-10 interspace". The patient underwent a standard laminectomy. The operative note reads, "Exploration through a standard laminectomy showed the spinal cord tightly stretched over an anterior protrusion and a slight bluish discoloration at the predicted level. . . . . Further bone was removed laterally so as to enable a careful exploration to be made both intrathecally and extrathecally anterior to the cord where a projecting knob was palpable. An attempt was made to remove this. . . . . it was found, however, that the summit of the protrusion was adherent to the anterior surface of the theca and could not be removed without producing serious disturbance and traction on the cord. This portion had to be left in situ. . . . ."

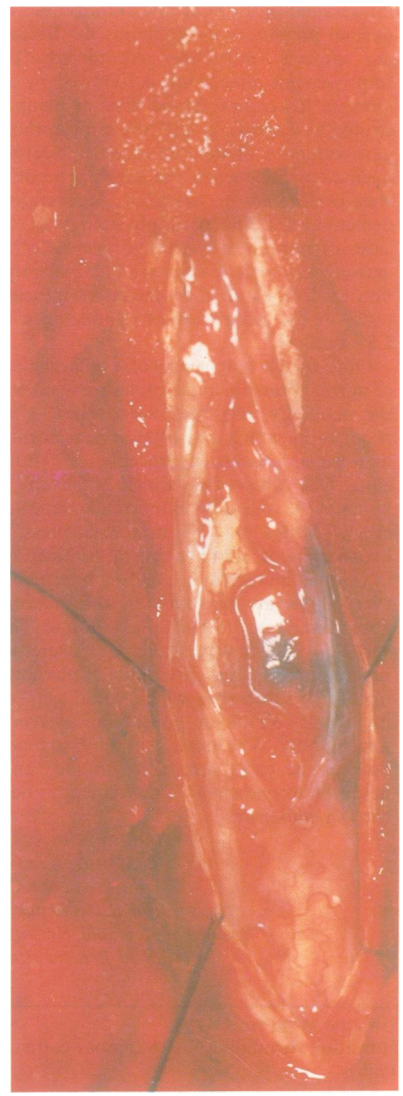

Fig *Laminectomy at T8, 9, 10 showing the bluish discoloration of the cord due to an intradural rupture of a thoracic disk.*

A flaccid paraplegia resulted once a later costotransversectomy[8] suggested by an associate resulted in the bony protrusion being removed without cord disturbance. The patient remained paraplegia. Mr Hulme documented six other cases removed successfully by costotransversectomy without neurologic deficit.

These two cases describe the ominous problems of a thoracic disk protrusion projecting into the dura or cord with secondary cord discoloration. The disk was not removed at the first operation and in both cases the patient became paraplegic. We firmly believe that every effort should be made to differentiate a cord tumour from thoracic disk with meticulous myelography and computed tomography at the involved level. The approach to the disk then must be made by one of several approaches described since 1967.[9–13] The results since this breakthrough have been most encouraging with no repeated results of the tragedies of the standard laminectomy approach.

ROBERT G FISHER
Department of Surgery, Section of Neurosurgery, University of Medicine and Dentistry of New Jersey, Rutgers Medical School, Academic Health Science Center, CN 19 New Brunswick, New Jersey 08903, USA

References

[1] Hulme A. The surgical approach to thoracic intervertebral disc protrusions. *J Neurol Neurosurg Psychiatry* 1960;**23**:133–37.
[2] Love JG, Kiefer EJ. Root pain and paraplegia due to protrusions of thoracic intervertebral disks. *J Neurosurg* 1950;**7**:62–69.

[3] Love JG, Schorn VG. Thoracic disc protrusions. *JAMA* 1965;**191**:627–31.
[4] Logue V. Thoracic intervertebral disc prolapse with spinal cord compression. *J Neurol Neurosurg Psychiatry* 1952;**15**:227–41.
[5] Arseni C, Nash F. Thoracic intervertebral disc protrusion. A clinical study. *J Neurosurg* 1960;**17**:418–30.
[6] Fisher RG. Protrusions of thoracic disc. The factor of herniation through the dura mater. *J Neurosurg* 1965;**6**:591–3.
[7] Tovi D, Strang RR. Thoracic intervertebral disk protrusions. *Acta Chir Scand* 1960;**267**:41.
[8] Crafoodi C, Hiertonn T, Lindblom K, Olsson S-E. Spinal cord compression caused by a protruded thoracic disk. Report of a case treated with antero-lateral fenestration of the disc. *Acta Orthop Scand* 1958;**28**:103–7.
[9] Patterson RHJ, Arbit E. A surgical approach through the pedicle to protruded thoracic disks. *J Neurosurg* 1978;**48**:768–72.
[10] Perot P, Munro DD. Transthoracic removal of midline thoracic disc protrusions causing spinal cord compression. *J Neurosurg* 1969;**31**:452–8.
[11] Ranshoff J, Spencer F, Siew F, Gage LJ. Transthoracic removal of thoracic disc. Report of three cases. *J Neurosurg* 1969;**31**:459–61.
[12] Sebhar LN, Jannetta PJ. Thoracic disc herniation, operative approaches and results. *Neurosurgery* 1983;**12**:303–5.
[13] Simeone FA. The modern treatment of thoracic disc disease. *Orthop Clin North Am* 1971;**2**:453–62.

The relationship of peripheral trauma and pain to dystonia

Sir: Schott[1] has recently reported four subjects in whom minor peripheral injury was responsible for the development of segmental dystonia. Although the mechanisms underlying trauma-induced dystonia are not known, activation of endogenous endorphins and adrenocorticotropin hormone (ACTH) might be involved. All patients experienced severe pain, sufficient to have activated central endogenous endorphins and ACTH.[2] The endogenous endorphins and ACTH[2] have been shown to be involved not only with the regulation of or reaction to pain, but also a wide range of motor and behavioural responses in laboratory animals.[3–6]

However, we are not aware of any report that endogenous administration of endorphins or other opioids produced dystonic movements. In fact, in one study[5] morphine or beta-endorphin injected directly into the brainstem of rats caused catalepsy and rigidity but not dystonic movements. However, ACTH N-terminal fragments, but not ACTH itself, adminstered in the same manner produced postural asymmetry and dystonic movements resembling human dystonia.[5 6] Jacquet and Abrams[5 6] have suggested that some forms of human dystonia may be related to a genetic abnormality of the ACTH molecule. In the cases reported by Schott[1] it is possible that the patients may have had an underlying mutation in the structure of the ACTH molecule. Thus, it is conceivable that the pain and associated stress of the patients activated the cerebral production and/or release of this abnormal compound leading to the development of the segmental dystonia. Although this mechanism is a conjecture, it is of interest that exogenously administered ACTH was reported to ameliorate symptoms of torsion dystonia in one patient.[7]

Taken together it appears that abnormalities of the molecular structure of ACTH may be implicated, at least in part, in the pathophysiology of human dystonia. We are currently studying this issue.

(Supported in part by NINCDS grant NS15441 and the Muscular Dystrophy Association).

REUVEN SANDYK*
PAUL CONSROE†
Departments of Internal Medicine (Neurology) and Pharmacology/Toxicology*†*
University of Arizona, Tucson, AZ 85724, USA

References

[1] Schott GD. The relationship of peripheral trauma and pain to dystonia. *J Neurol Neurosurg Psychiatry* 1985;**48**:698–701.
[2] Haynes RC, Murad F. Adrenocorticotropic hormone: adrenocorticosteroids and their synthetic analogs: inhibitors of adrenocortical biosynthesis. In: Gilman AG, Goodman LS, Rall TW, Murad F, eds. *The Pharmacological Basis of Therapeutics*. 7th ed. New York: Macmillan Publishing Co, 1985:1459–89.
[3] Sandyk R. The endogenous opioid system in neurological disorders of the basal ganglia. *Life Sci* 1985;**37**:1655–63.
[4] Grillner S, Zangger P. How detailed is the central pattern generation for locomotion. *Brain Res* 1975;**88**:367–71.
[5] Jacquet YF, Abrams GM. Postural asymmetry and movement disorder after unilateral microinjection of adrenocorticotropin 1–24 in rat brainstem. *Science* 1982;**218**:175–7.
[6] Jacquet YF. "Dystonia"-like postural asymmetry after microinjection of ACTH N-terminal fragments but not after $ACTH_{1-39}$ in rat brainstem suggests role of neuropeptide mutation in genetic movement disorder. *Brain Res* 1984;**294**:144–7.
[7] Baumann L, Baumann J. Betrachtungen zu einem Fall von Torsiondystonie. *Acta Neurol Scand* 1963;**39:Suppl 14**:221–5.

Transient global amnesia after whiplash trauma

Sir: We are pleased to see the letter from Hofstad and Gjerde on whiplash amnesia.[1] Their note strengthens our opinion that whiplash amnesia is probably a particular form of transient loss of short-term memory,[2] Whiplash amnesia seems to present characteristic features as the reported cases[1 3] show. For instance, selective pain in the neck and dizziness are not found in transient global amnesia and definite retrograde amnesia is rarely absent in amnesia by concussion. We thus appreciate the work of Hofstad and Gjerde and we think that the description of clinical features in newly diagnosed cases of whiplash amnesia confirm our opinion.

I BUENAVENTURA
J MATIAS-GUIU
The Neurology Service, Hospital del Valle de Hebrón, Universidad Autonoma Barcelona, Spain

References

[1] Hofstad H, Gjerde IO. Transient global amnesia after whiplash trauma. *J Neurol Neurosurg Psychiatry* 1985;**48**:956–7.
[2] Matias-Guiu J, Buenaventura I, Cervera C, Codina A. Whiplash amnesia. *Neurology (Cleveland)* 1985;**35**:1259.
[3] Fisher CM. Whiplash amnesia. *Neurology (NY)* 1982;**32**:667–8.

Free light chains in the cerebrospinal fluid

Sir: My letter concerns the article "Free light chains in the cerebrospinal fluid: an indicator of recent immunological stimulation", published in your journal 1985;**48**:995–8.

I do not agree with the following statements of the authors since they misinterpret the findings of our laboratory:

(1) "... Bollengier *et al* suggested that the free light chains occurred in CSF..." Nowhere in our papers did we suggest the presence of free light chains; the word "suggest" refers to a mere hypothesis and in fact we clearly demonstrated and quantified those free light chains in radial immunodiffusion by using a specific anti free light chain antiserum (ref. 9 in the article by Vakaet and R. Thompson)

(2) "... antiserum directed against Bence-Jones protein which had been previously adsorbed with heavy chains..." We did no such thing for the very reason