[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.

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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.

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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

that the anti free light chain antiserum we purchased from Behringwerke is raised in the goat by suitable mixtures of isolated Bence-Jones protein. The goat is a species that recognises only hidden determinants; consequently, as was clearly stated in the leaflets of Behringwerke, this antiserum precipitates only with free light chains and no bound chains.

We clearly stated the source of our antisera in our papers.

(3) The references concerning our papers are not complete; missing are

(a) Biochemical findings in MS. III. Immunoglobulins of restricted heterogeneity and light chain distribution in CSF of patients with MS. F Bollengier, P Delmotte, A Lowenthal, *J Neurol* 1976;**212**:151–8.

(b) Oligoclonal immunoglobulins, light chain ratios and free light chains in CSF and serum from patients affected with various neurological diseases. F Bollengier, N Rabinivitch, A Lowenthal. *J Clin Chem Clin Biochem* 1978; **16**:165–73.

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Thompson replies:

Dr Bollengier's first two points are essentially the same: what is the preferred method to detect light chains which are free (versus bound to heavy chains)—either some ostensibly "specific" antiserum or some electrophoretic separation of free from bound light chains which is then independently confirmed using other antisera against heavy chains (IgG and IgA). Experience has taught us to be wary of suppliers' claims about antibody specificity, mainly because their test systems are often rather different from those being used in one's own methods. Our previous work with the technique which relies on the suppliers' claims, have shown that "specific" antisera against free light chains, can in fact also react with bound light chains.[1] She has also noted a similar aspect of the same general problem, and hence we referred to her 1979 paper.[2] She says "Discrepancies in results according to different techniques used have been frequently noticed." She states this because she had noted "much higher" levels of free light chains in normals than had previous workers. With our electrophoretic separation and post immunofixation technique we find, however, no free light chains in normals.

Concerning her third point, the two "missing" references are clearly listed in her 1979 paper,[2] which we referenced as her latest paper on the topic.

References

[1] Walker RWH, Keir G, Thompson EJ. Assessment of cerebrospinal fluid immunoglobulin patterns after isoelectric focusing: Use of kappa and lambda light chain immunoperoxidase staining. *J Neurol Sci* 1983; **58**:123–34.

[2] Bollengier F. Bound and free light chains in serum from patients affected with various neurological diseases. *J Clin Chem Clin Biochem* 1979;**17**:45–9.

Book reviews

Biochemistry and the Central Nervous System (5th Ed.) By Henry McIlwain and Herman S Bachelard. (Pp 660; £40·00.) Edinburgh: Churchill Livingstone, 1985.

It is 30 years since the first edition of this volume appeared. During the ensuing period, major advances have been made in our understanding of the biochemistry of the central nervous system. However, the new editions have, in turn, successfully kept the reader abreast of the most recent developments. Indeed, this book has become a classic reference volume of this era.

The 5th edition of *Biochemistry and the Central Nervous System* is no exception to the standard set by the previous editions. It is, without doubt, a reference work which all libraries and many individuals will value. The contents have been substantially reorganised and this edition sees the addition of four new chapters. Particular attention is paid to brain neurotransmitters, modulation of their actions and the consequences in terms of pharmacological effect, drug action and disease process. The volume as a whole is well produced and presented. The mammoth effort put into the new edition by the authors will be well appreciated for many years to come by clinicians and basic scientists involved in a variety of neuroscience disciplines. I congratulate Professors McIlwain and Bachelard on the achievement of making brain biochemistry understandable to such a wide audience.

P JENNER

Cerebral Vascular Disease 5 *(World Federation of Neurology 12th Salzburg Conference) Excerpta Medica International Congress Series 687.* Edited by JS Meyer, H Lechner, M Reivich & EO Ott. (Pp 361; $74.00, £53.) Amsterdam: Elsevier Biomedical Press B.V. 1985.

This book catalogues the proceedings of the 12th Meeting of the International Salzburg Conference on Cerebral Vascular Disease held in September, 1984. The volume is, therefore, published in reasonably good time. It is well printed with 360 pages with good reproduction of diagrams, and illustrations but only moderately good reproduction of radiographs. The proceedings included a half-day symposium on cost-effectiveness of stroke investigation and, as usual, consisted of a collection of papers