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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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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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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