was found to be highly effective in inducing changes in the intraocular pressure (IOP) and vasodilatation in the uvea in the rabbit; the reaction seems to be in common with an axon reflex mediated by the peripheral branches of the nerve, at the endings of which some active histamine-like substance is liberated, causing pupillary ejection reaction and intraocular vasodilatation.

Can mechanical activation of iris trigeminal nerve terminals develop naturally and contribute to miosis seen during and between attacks? Acute elevations in the IOP have been shown to discharge impulses in iris fibres (whole nerve and corneo-scleral fibres) probably due to mechanical distortion of the iris and the chamber angle which suggests the production of painful impulses described in experimental animals.1 An association between migraine and low-tension glaucoma (LTG) has been suggested recently; the differential diagnosis of LTG should include wide diurnal fluctuations in which high pressures are occurring at times when they are not being recorded. Given the central importance of autonomic nerves in mechanism of action of drugs, autonomic hyperfunction in those with migraine during headache-free intervals1 allows development of a relatively higher IOP in response to a variety of stimuli and situations, thereby resulting in exaggerated fluctuations in the pressure that possibly contributes both to visual field loss and mechanical activation of iris nerve fibres.

The results of studies of autonomic nervous system dysfunction in migraine have been contradictory.4 Besides wide normal inter- and intra-individual variations in the reactions of the autonomic nervous system, it may be useful (and not necessarily simplistic) to consider autonomic hyperfunction during migraine attacks as an adaptive (secondary stress) response liable to "fatigue" variably in the later stages of severe headache, one function of which may serve to limit the effects of vasodilatation (of intraocular and cranial blood vessels) resulting from antidromic discharge from trigeminal nerve fibres.

Increased risk of multiple sclerosis among nurses and doctors

A recent study concluded that the multiple sclerosis (MS) death rates in British nurses and qualified medical practitioners was not greater than expected. However, as part of a population-based prevalence study of MS in North East Scotland,2 the occupation of all economically-active females over 15 years of age was classified at the time of onset of the disease and compared with the distribution of economically-active males and females in North East Scotland based on the 1961 Census.3 Fifteen female nurses (occupational group 282) had MS (expected 6.2) and four male medical practitioners (occupational group 280) were affected whilst 0.8 were expected (both p < 0.001).3

Whilst the actual numbers involved were small, particularly for medical practitioners, an analysis of occupation at the time of onset of MS will nevertheless produce a less biased assessment than analysis of occupation at the time of death, given the well-recognised downward occupational drift in chronic disabling diseases such as MS and accepted by Dean and Gray.1 If we conclude that, at least in North East Scotland in 1970, there was an excess risk of MS among female nurses and possibly among male doctors.

[References]

1 Dean D, Gray R. Do nurses or doctors have an increased risk of developing multiple sclerosis? J Neurol Neurosurg Psychiatry 1990;53:899-902.


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