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 have much 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 contraction and intense intraocular vasodilatation.³

Can mechanical activation of iris trigeminal nerve terminals develop naturally and contribute to meiosis seen during and between migraine attacks? Acute elevations in the IOP have been shown to discharge impulses in iris nerve fibres (and whole nerve and corneo-scleral fibres) probably due to mechanical distortion of the iris and the chamber angle⁴ which suggests the production of painful impulses described in experimental animals.^{3,4} An association between migraine and low-tension glaucoma (LTG) has been suggested recently;⁵ 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 effects on aqueous dynamics, autonomic hypofunction in those with migraine during headache-free intervals⁶ allow 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.⁶ 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 view 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.

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Drummond replies:

I thank Dr Gupta for his comments and for the opportunity to clarify a point which may not be clear in my paper.¹ In 62 patients with headache on the usually-affected side the pupil was smaller, on average, and eyelid separation greater, on the symptomatic side.

These autonomic disturbances, however, were not normally observed in the same

patients. In fact, miosis was usually associated with ptosis on the symptomatic side during attacks of migraine (in darkness, $r = 0.38$, $p < 0.001$, see table 4). Since miosis persisted during the headache-free interval^{1,2} it seems likely that, in some cases, permanent ocular sympathetic deficit prevented greater eyelid separation on the affected side during attacks of migraine.

Discharge of the trigeminal nerve could have contributed to miosis during migraine, but this is unlikely to be the mechanism of miosis during the headache-free interval.^{1,2} On the other hand, thermoregulatory flushing is reduced on the usual side of migraine headache,³ consistent with a decrease in cervical sympathetic outflow.^{4,5} Intraocular pressure does not increase on the symptomatic side during attacks of migraine,⁶ which argues against the idea that autonomic disturbances are due solely to trigeminal discharge.

Thirty years ago, Walsh and O'Doherty⁷ suggested that swelling of the internal carotid artery during migraine could cause ophthalmoplegia, either by direct pressure on nerves in the cavernous sinus, or by interfering with the local circulation of the involved nerves. Narrowing of the internal carotid artery, presumably due to oedema, was demonstrated by arteriography in two of three selected cases. This same process could cause cervical sympathetic deficit in migraine, because sympathetic fibres supplying the eye and skin of the forehead form a plexus around the internal carotid artery.

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- 1 Drummond PD. Disturbances in ocular sympathetic function and facial blood flow in unilateral migraine headache. *J Neurol Neurosurg Psychiatry* 1990;53:121-5.
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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.¹ However, as part of a population-based prevalence study of MS in North East Scotland,² the occupation of all economically-active patients over 15 years of age was classified at the time of onset of the disease³ and compared with the distribution of economically-active males and females in North East Scotland based on the 1961 Census.⁴ 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 $p < 0.001$).⁵

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 I 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.

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- 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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Gray and Dean reply:

We are grateful for Dr Shepherd's comments on our study of mortality from multiple sclerosis (MS) among doctors and nurses¹ and for his interesting data suggesting an excess incidence of MS among doctors and nurses in North East Scotland.

Dr Shepherd suggests that downward occupational drift by the time of death could explain the lack of any excess of MS deaths among doctors and nurses in our study. However, we believe this to be unlikely—particularly for medical practitioners. The British doctors study was a prospective study and the occupation of medical practitioner was (by definition!) the necessary prerequisite to be included. Occupation was determined from the 1951 British Medical Register and not from the death certificates.

There may be three explanations for the excess incidence seen among doctors and nurses in Dr Shepherd's study. First, the methods of identifying patients² are likely to have resulted in over-representation of doctors and nurses. To supplement the original register of patients with MS further patients were identified by visits to local hospitals and extensive surveillance of their records. A further 73 cases in this study were then identified by asking the region's GPs to note any additional cases that they knew of. As a result, nurses and doctors with MS seem more likely to be identified than those in non-medical professions.

Second, using the 1961 census data to estimate the proportion of economically active males and females who are doctors or nurses in 1970 may have introduced some bias if the proportion who were doctors and nurses had increased over the intervening period.

Finally, doctors and nurses may have MS diagnosed earlier in the disease than other MS sufferers. Again, this "lead time" bias is likely to suggest—artefactually—that there is