# CORRESPONDENCE

## Fluoxetine oral administration increases intraocular pressure

EDITOR,—Fluoxetine is a widely prescribed antidepressant which acts as a selective inhibitor of neuronal serotonin uptake.[1] Recently, Ahmad[2] reported on a case of acute narrow angle glaucoma due to fluoxetine administration. For this purpose, we verified the intraocular pressure (IOP) variations in 20 consecutive depressed outpatients (five males and 15 females, age range 33–47 years) to whom fluoxetine therapy was first prescribed. All patients received a complete ophthalmic examination to exclude the pre-existence of both acute and chronic glaucoma. Patients with systemic diseases other than affective disorders were excluded from the study.

After at least 12 hours of fasting, in early morning (8 am) subjects were assigned to either 20 mg of oral fluoxetine or placebo in a randomised crossover double blind fashion. The alternative treatment was given 1 week later, a time lag considered sufficient for a single dose fluoxetine washout.[3] Intraocular pressure was recorded (with a Goldmann tonometer mounted on a Haag-Streit slit-lamp) at baseline and hourly up to 12 hours, both when patients were given fluoxetine and when they were given placebo.

All patients showed a significant increase in IOP 2 hours after oral administration of fluoxetine and 8 hours later some patients still exhibited higher IOP values (Fig 1); however, when patients received placebo there were no significant changes in IOP.

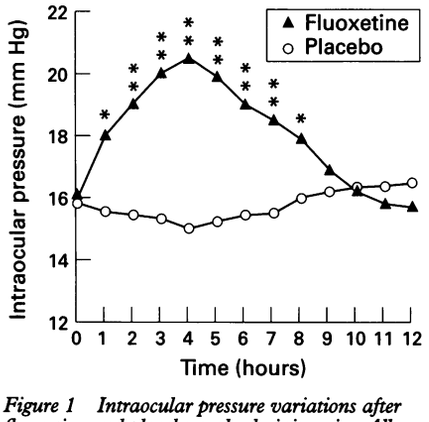


*Figure 1 Intraocular pressure variations after fluoxetine and placebo oral administration. All values are mean plus or minus SD. Paired Student's t test was used for statistical analysis. *p<0.05; **p<0.01 (treated versus untreated).*

The mechanism by which fluoxetine increases IOP might be due to the inhibitory effect on serotonin uptake. In fact serotonin, when injected into the anterior chamber, produces a significant increase in IOP[4] and ketanserin, an agent with serotonergic blocking properties, reduces IOP both in animals and in humans.[5] These data suggest that ophthalmic examination might be included in the protocol of depressed patients given fluoxetine therapy because this drug might raise intraocular pressure.

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1 Wong T, Bymaster FP, Horng JS, Molloy BB. A new selective inhibitor of uptake of serotonin into synaptosomes of rat brain: 3-(p-trifluoromethy-phenoxy)-N-methyl 3-phenyl-propylamine. *J Pharmacol Exp Ther* 1992;**193**:804–11.
2 Ahmad S. Fluoxetine and glaucoma. *Ann Pharmacother* 1991;**25**:436.
3 Malcolm L. Fluoxetine efficacy vs comparative drugs: an overview. *Br J Psychol* 1988;**153**:51–8.
4 Osborne NN, Tobin AB. Serotonin accumulating cells in the iris-ciliary body and cornea of various species. *Exp Eye Res* 1987;**44**:731–46.
5 Costagliola C, Iuliano G, Rinaldi M, Russo V, Scibelli G, Mastropasqua L. Effect of topical ketanserin administration on intraocular pressure. *Br J Ophthalmol* 1993;**77**:344–8.

## Visual screening programme for preschool children

EDITOR,—I welcome Williamson *et al*'s recent article[1] and your editorial[2] suggesting that school vision screening may be the optimum method of detecting amblyopia in children in some circumstances. I would support the view that a larger number of children could be screened. In Glasgow, in the north west area, covering approximately the same geographical area as that quoted in Williamson *et al*'s study of preschool orthoptic screening, 95% of children enrolled in mainstream school in primary one were screened during the session 1994–5.

However, in the same year there was a significant difference between the number of new visual defects detected (unilateral visual acuity 6/9 or poorer using a single letter test) in schools in the sector of the city where preschool orthoptic screening was being carried out—131/3235 (4%)—compared with that where there was no preschool screening (398/3644 (11%)).

The quality of school vision screening in Glasgow is being studied at present and improvement is actively being sought. Only once satisfactory standards can be demonstrated, in addition to the ability to screen a large proportion of the population, can it be argued that this should stand alone as the sole method of detecting amblyopia while it is still amenable to treatment.

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1 Williamson TH, Andrews R, Dutton GN, Murray G, Graham N. Assessment of an inner city visual screening programme for preschool children. *Br J Ophthalmol* 1995;**79**:1068–73.
2 Elston J. Preschool visual screening. [Editorial] *Br J Ophthalmol* 1995;**79**:1063–4.

## Reply

EDITOR,—The communication from our colleague, Dr Spowart, raises a statistic of interest—that is, an apparent but unsubstantiated reduction in the proportion of children with poor vision in areas of the north west of Glasgow with preschool screening. The 11% prevalence of children at school with 6/9 vision or less (using singles testing) in the unscreened population is similar to the proportion of preschool children in our survey with this level of visual acuity at screening (9.2%, also with singles testing). Although the screening programme may have reduced the amblyopia somewhat we feel unable to take the credit for reducing the rate to 4%. Refraction of the patients at the first visit improved the visual acuity in a proportion (78.3% remained with 6/9 or worse, Snellen acuity, after spectacles) but the proportion remained similar after the first year of hospital treatment—that is, when they were at school (77.8%). We therefore cannot explain such a difference in the screened and unscreened populations of Dr Spowart especially when only 57% of the total population of children attended our screening services. Was the demographic constitution of her two populations different? Did the advertising of visual disability via preschool screening cause parents to seek advice from others—for example, optometrists? The questions remain. We advocate school screening by personnel specifically trained because of the increased attendance. Perhaps school nurses could be employed thereby allowing redeployment of orthoptists to refract and treat the increased numbers of children detected.

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# BOOK REVIEW

**Colour Atlas of Scleritis.** By P Watson, J M Ortiz. Pp 122. £55. London: Mosby Wolfe, 1995.

The authors have complied an exceptional illustrated documentation of scleral inflammation. The book is a great pleasure to read and, with the authors' selection and great variety of cases, gives the reader the full spectrum of scleral inflammation. The authors have provided a thorough description of the diagnosis and management of scleritis which includes understanding the normal anatomy and vasculature of both the conjunctival and episcleral vessels. Throughout the book they describe examples of scleritis and associated scleral inflammation using fluorescein angiography which beautifully illustrates the vascular changes that occur in the various forms of scleritis where examples of the diagnostic power of ultrasonography in these conditions are shown.

It is certainly a very useful and educational book to have in any department. If there is a criticism it is perhaps in the discussion of the management of scleritis, which would have benefited from an appraisal of the underlying immunopathology and greater details of why immunosuppressive regimes are used. Although I am sure it was not in their remit, documentation of case series with this form of treatment or reference to it would be of use to readers so that they can gain understanding of the success of such treatment.

Overall this is one of the clearest examples of presenting scleritis in an illustrated form available today.

ANDREW DICK