Sir,

Salomon *et al.*[1] cite a publication of ours[2] as advocating 'administration of aspirin for prevention of a second event of nonarteritic anterior ischemic optic neuropathy (NAION).' This is a miscitation. On the contrary, we did not conclude, based on our data, that aspirin was beneficial. Rather we concluded that 'our findings suggest a possible short-term (1 to 2 years) benefit but little or no long-term (5 years) benefit in using aspirin to reduce the risk of NAION in the fellow eye.' We also advised the reader that our results be viewed with caution because our study was not conducted to prospectively assess the benefit of aspirin with a standard controlled protocol.

The authors' finding of a lower incidence of second eye NAION in aspirin-treated compared with non-treated patients is almost certainly due to chance. This apparent association is being driven by an implausibly high second-eye rate (50%) in their small number of non-treated patients ($n = 16$). We believe, based on our data from 431 patients with NAION, that the incidence of second-eye NAION within 5 years, without aspirin therapy, is likely to be only about 10–15%. In fact, the authors' life-table estimate of 20% for the 5-year incidence of second eye NAION in the aspirin-treated patients (325 mg/day) actually exceeds our estimate for non-aspirin-treated patients.

Although we share the authors' opinion that a prospective study is warranted, we noted in our article that the expected low incidence of second-eye NAION limits the feasibility of such a study.

References

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2. Beck RW, Hayreh SS, Podhajsky PA, Tan ES, Moke PS. Aspirin therapy in nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol 1997;123:212–7.

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Sir,

In their letter Beck and Hayreh pointed out that the incidence of second eye NAION in our study[1] was higher than in their study[2] (50% versus 20%, respectively). However, another study by Hayreh's group[3] reported a cumulative incidence of second eye NAION of 34.6% at 5 years and 56.7% at 10 years. Thus, the incidences of second eye involvement seem to vary from one study to another.

In our study the lower incidence of second eye involvement among those patients taking 325 mg/day aspirin was not statistically significant as we carefully pointed out ($p = 0.358$). Nonetheless, in our view the estimated 250% increase in mean time to second eye involvement in patients on aspirin (325 mg/day) appeared to be of clinical significance. An interesting contrast between our results and those of Beck *et al.*[2] is that we found that aspirin provided the greatest reduction in risk of second eye NAION from > 2 years after the first event, whereas they found the greatest benefit of aspirin treatment during the first 2 years after the first event. Obviously, only a large multicentre prospective study of patients with NAION with and without aspirin may resolve this important issue.

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3. Beri M, Klugman MR, Kohler JA, Hayreh SS. Anterior ischemic optic neuropathy. VII. Incidence of bilaterality and various influencing factors. Ophthalmology 1987;94:1020–8.

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Sir,

The article by Murray *et al.* on biometry of the silicone oil-filled eye[1] both complicates and oversimplifies the issue of axial length estimation. The length of an object measured ultrasonically is the speed of sound through it multiplied by the time the sound wave takes to traverse it. Before biometry became as critical as it is now many machines used an average sound speed through an 'average' eye, usually 1550 metres per second (not 1532 m/s as the authors state). But this immediately fails once one is measuring other than an average eye. Thus in a long or a short eye, or in an eye with a thick or a thin lens, using that speed could not give a correct measurement. For example, in a long eye approximately 84% of the time the sound is travelling at 1532 m/s (sound speed in aqueous and vitreous) while in a short eye it is travelling at this speed for only 72% of the time. Therefore the 'average' speed in these two types of eye is very different. To calculate any distance where the speed varies we need to know for how *long* sound travelled at each speed and then calculate how *far* it travelled at each speed. The sum of all those little distances is the total distance travelled.

Many recently manufactured A-scan machines calculate axial length in this way. If one looks at Fig. 1 one can see that the axial length is the sum of the anterior chamber depth (ACD) plus the lens thickness (LT) plus the distance from the back of the lens to the retina. The machine has measured the time taken for the echo to travel to and from the front of the lens, divided it by 2 and multiplied it by 1532 giving a length of 2.76 mm. Similarly the same calculation for the lens at a sound speed of 1641 m/s gives a thickness of 4.75 mm. Finally the vitreous is measured at 14.36 mm in the same way. The axial length is therefore the sum of the three: 21.67 mm. However if the vitreous cavity is filled with silicone oil then the sound wave slows down to 987 m/s (in this

