

Drops and falls

SIR—Congratulations on the fascinating case report by Carey highlighting falls caused by β -blocker eyedrops for glaucoma [1].

We recently saw an 87-year-old man who collapsed in the eye clinic waiting room. He was using twice-daily β -blocker eyedrops (levobunolol 0.5%) to treat glaucoma complicating corneal graft surgery. An ECG in 2006, before his operation, showed first-degree heart block and a pulse rate of 57 bpm. Several weeks later, he collapsed in the eye outpatient clinic and was unresponsive with a pulse rate of <30 bpm and blood pressure of 100/60 mmHg. ECG showed no acute changes. He recovered after basic resuscitation, and his β -blocker eyedrops were stopped.

Ten weeks later, his pulse rate had improved to 63 bpm, but he volunteered his satisfaction at not falling over since his change of eyedrops, whereas previously he had daily falls!

Numerous cardiovascular side-effects are attributed to the use of β -blocker eyedrops including falls, arrhythmia, syncope and myocardial infarction [2]. Glynn *et al.* found that the commonest cause of falls in elderly glaucoma patients was β -blocker eyedrops [3].

Systemic absorption of topical β -blocker eyedrops occurs via the nasal mucosa after passage through the nasolacrimal canal and via pulmonary absorption of inhaled drug particles. These routes avoid hepatic first-pass metabolism giving a high plasma β -blocker concentration that correlates well with haemodynamic impairment [4]. Vuori and Kaila [5] reported persisting high levels of systemic β -receptor occupancy 12 h after a single β -blocker eyedrop; they also noted a slower rate of decline of β -receptor occupancy in older patients.

Topical β -blockers need to be considered in any case of falls, and we would like to draw attention to the newer fixed combination eyedrops whose names give no suggestion as to their β -blocker (0.5% timolol) component (Cosopt™, Combigan®, Xalacom®, DuoTrav™ and Ganfort®). Physicians need to specifically ask their patients whether they use eyedrops as many simply fail to recognise them as ‘medication’.

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References

1. Carey B. Atishoo! Atishoo! We all fall down! *Age Ageing* 2006; 35: 446–7.
2. Nelson WL, Fraunfelder FT, Sills JM, Arrowsmith JB, Kitisky JN. Adverse respiratory and cardiovascular events attributed to timolol ophthalmic solution, 1978–85. *Am J Ophthalmol* 1986; 102: 606–11.
3. Glynn RJ, Seddon JM, Krug JH Jr *et al.* Falls in elderly patients with glaucoma. *Arch Ophthalmol* 1991; 109: 205–10.
4. Nieminen T, Uusitalo H, Turjanmaa V *et al.* Association between low plasma levels of ophthalmic timolol and haemodynamics in glaucoma patients. *Eur J Clin Pharmacol* 2005; 61: 369–74.
5. Vuori M, Kaila T. Plasma kinetics and antagonist activity of topical ocular timolol in elderly patients. *Graefes Arch Clin Exp Ophthalmol* 1995; 223: 131–4.

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