REPLY

We thank Dr. Tobias for pointing out a very important caveat to our recent case report:[1] not all calcium channel antagonists are equal. Certainly, the cardiovascular effects of diltiazem (a benzothiazepine) cannot be equated with other calcium channel antagonists from a completely different chemical group, like verapamil (a phenylalkylamine) or nifedipine (a dihydropyridine).[2] While diltiazem has its primary effect on phase four depolarization (reduced automaticity), nifedipine has its primary effect on coronary and systemic vasorelaxation.[3] With such a different pharmacological effect, it is reasonable to expect a different quality of cardiovascular control with different calcium channel antagonists under the conditions described in our report. The issue remains, under the circumstances described, if tumour devascularization cannot be obtained, an increased level of awareness is required, and the use of monotherapy with calcium channel blockers should be questioned. We do not argue or disagree with Dr. Tobias that another class of calcium channel antagonist may be more effective (perhaps one with more systemic vasorelaxation) in control of cardiovascular variables than a benzothiazepine (as reported in our case report). Is it reasonable (or wise) in a clinical circumstance with elevated plasma catecholamine concentrations not to use more specific and readily available alpha-one (prazocin) or beta-one (metoprolol or esmolol) antagonists? We think not.

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Peribulbar anaesthesia

To the Editor:

We read with interest the findings of Dr. J.E. Roberts *et al.*, "Improved peribulbar anaesthesia with alkalinization and hyaluronidase"[1] We have recently completed a similar prospective randomised double-blind study on patients undergoing peribulbar anaesthesia for cataract extraction and lens implantation.

We compared two groups given a standard local anaesthetic mixture of 0.5% bupivacaine and 2% lidocaine (without adrenaline) in a 1:1 ratio, with or without hyaluronidase 25 ml^{-1}. One anaesthetic operator experienced in the technique of peribulbar anaesthesia described by R.A. Fry *et al.*[2] performed the procedure, noting the time taken to establish satisfactory anaesthesia – measured as the degree of globe akinesia. Control group ranged 2 to 15 min (median 4 min). Hyaluronidase group range 2 to 12 min (median 4 min). Using a Mann-Whitney test to compare onset times to globe akinesia between groups, P value = 0.6, 95% confidence interval (−1 to 2 min). We concluded that the efficacy of our pH unadjusted hyaluronidase-bupivacaine-lidocaine mixture (pH 5.24) was equal to that of our control solution containing only local anaesthetic (pH 5.16).

Our failure to demonstrate a difference between the two groups in this study supports the findings of J.E. Roberts *et al.*, in that the use of hyaluronidase is inappropriate in a setting outside of its pH limits for activity of 6.4 to 7.4, despite our having used two and a half times the concentration used in their study.

We question the need for the use of adrenaline containing local anaesthetic solutions and propose that it serves only to acidify further the local anaesthetic mixture, with little to be gained clinically (0.5% bupivacaine-2% lidocaine with adrenaline 1/200,000. 1:1 ratio pH = 4.67 and 0.5% bupivacaine-2% lidocaine with adrenaline 1/200,000. 1:1 ratio + hyaluronidase 25 units/ml of mixture pH = 4.81).

Alkalinization with bicarbonate would appear to optimize the activity of hyaluronidase, but we feel that with our current practice a rapid and effective block can be reliably achieved without the need to either alkalinize the mixture or add hyaluronidase.

There are inherent risks in using adrenaline and hyaluronidase,[3] as there are in the preparation of drug mixtures, such as the alkalinization of local anaesthetic mixtures with bicarbonate (wrong drugs and or wrong dose).

We advocate a simplistic approach to peribulbar anaesthesia to safeguard its popularity and good safety record.

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