Ocular responses after intravenous lidocaine

To the Editor:
We read with interest the review of the physiological and anaesthetic implications of increased intraocular pressure (IOP).[1]

We wish to further emphasize the role of intravenous lidocaine in mitigating or preventing the systemic and ocular reactions and especially the acute increase in IOP associated with laryngoscopy and tracheal intubation.[2] The mechanisms of elevation of IOP after laryngoscopy and after succinylcholine administration are different, so pretreatment with a small dose of gallamine or d-tubocurarine will not attenuate the increase in IOP caused by tracheal intubation itself. The rise in IOP is, in fact, caused by sympathetically mediated vasoconstriction generating increased venous return and a sudden rise in central venous pressure.

The administration of 2 $mg \cdot kg^{-1}$ lidocaine IV two minutes prior to intubation has attenuated significantly the rise in heart rate and lowered the incidence of such local laryngeal reflexes as cough and breath-holding, and of ocular responses such as pupillary dilatation or lacrimation. IV lidocaine caused a significant reduction of IOP prior to tracheal intubation, compared to saline given in a control group, as well as significant suppression of the increase in IOP following laryngoscopy and intubation.

We advocate the routine use of intravenous lidocaine prior to tracheal intubation in patients suffering from glaucomatous or lacerated eye, and in other urgent ophthalmic operations.

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1 *Cunningham AJ, Barry P.* Intraocular pressure – physiology and implications for anaesthetic management. Can Anaesth Soc J 1986; 33: 195–208.
2 *Drenger B, Pe'er J, BenEzra D, Katznelson R, Davidson JT.* The influence of intravenous lidocaine on the increased intraocular pressure induced by tracheal intubation. Anesth Analg 1985; 64: 1211–3.

Erratum – Cardiovascular and respiratory effects of ketamine in the neonatal lamb

To the Editor:
I am writing to you to inform the readers of an error which appeared in our article[1] in the January 1986 issue.

The doses of ketamine used in this study were reported incorrectly as 1 $mg \cdot kg^{-1}$ and 2 $mg \cdot kg^{-1}$. These were equivalent doses and the actual doses of ketamine used in the lambs were 15 $mg \cdot kg^{-1}$, to be equivalent to 1 $mg \cdot kg^{-1}$ in humans and 30 $mg \cdot kg^{-1}$, to be equivalent to 2 $mg \cdot kg^{-1}$. These doses were used because the dose requirements for ketamine in animals are higher than in man.[2] We apologize for this confusion.

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1 *Burrows FA, Norton JB, Fewel J.* Cardiovascular and respiratory effects of ketamine in the neonatal lamb. Can Anaesth Soc J 1986; 33: 10–5.
2 *Booth NH.* Intravenous and other parenteral anesthetics. *In*: Booth NH, McDonald LE (eds.) Veterinary Pharmacology and Therapeutics, 5th Edition. Iowa State University Press, Ames, Iowa, 1982.

Plasma cholinesterase activity following administration of glycopyrrolate or atropine

To the Editor:
An *in vitro* study of the effects of glycopyrrolate and atropine on human plasma cholinesterase by Zsigmond *et al.*[1] in this journal showed a moderate inhibitory effect on this enzyme with high concentraitons of both agents, with a theoretical possibility of an interaction with succinylcholine or ester-type local anaesthetics. It has since been pointed out that on pharmacological grounds only extremely large doses of glycopyrrolate, which are unlikely ever to be used in clinical practice, would produce such an inhibition of plasma cholinesterase activity in man.[2] We now report actual measurements of plasma cholinesterase