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.

B. Drenger MD
M. Gertel MD FRCPC
J. Pe'er MD
Department of Ophthalmology
Hadassah University Hospital
P.O.B. 12000
11-91 120 Jerusalem
Israel

REFERENCES

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.

Frederick A. Burrows MD FRCPC
Department of Anaesthesia
University of Toronto and
The Hospital for Sick Children
555 University Avenue
Toronto, Ontario
M5G 1X8

REFERENCES

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

activity in man following administration of glycopyrrolate or atropine.

Twenty-six adult patients of both sexes on no medication and without any medical disorder likely to affect plasma cholinesterase activity were studied after obtaining their consent and the Hospital Ethical Committee's approval. No premedication was administered. The patients were divided into four groups, two receiving glycopyrrolate 7.5 $\mu g \cdot kg^{-1}$ IV (Group A, n = 7) or atropine 15 $\mu g \cdot kg^{-1}$ (Group B, n = 7) prior to induction of anaesthesia and two receiving glycopyrrolate 10 $\mu g \cdot kg^{-1}$ (Group C, n = 6) or atropine 20 $\mu g \cdot kg^{-1}$ IV (Group D, n = 6) prior to neostigmine administration for antagonism of nondepolarising neuromuscular block. Venous blood samples were taken before the anticholinergic drug administration and 1, 2, 5, 10, 20 and 30 minutes later in groups A and B and 1, 2, 5, 10 and 15 minutes later but prior to the administration of neostigmine in groups C and D. Plasma was separated within 30 minutes and cholinesterase activity was estimated using a colorimetric method[3] with butyrylthiocholine as the substrate. A 2 µl sample was diluted with 300 µl of the reagent and was incubated for 120 seconds. Statistical analysis of the data was carried out using analysis of variance.

Plasma cholinesterase activity in the four groups is given in the Table. Analysis of variance showed that there was no significant difference in plasma cholinesterase activity in any group over the period of study.

Our results make it unlikely that routinely used doses of glycopyrrolate or atropine have no clinically important effect on plasma cholinesterase activity. Interpolation made from the *in vitro* low anticholinesterase effect and minimal changes in plasma cholinesterase activity measured in man leads to the conclusion that either anticholinergic drug is unlikely to interfere with the hydrolysis of succinylcholine or ester-type local anaesthetics in patients.

TABLE Plasma cholinesterase activity ($I.U. \cdot ml^{-1}$) following glycopyrrolate and atropine administration (mean ± SD)

Minutes after drug admin.	*0*	*1*	*2*	*5*
Group A	5.65	5.49	5.41	5.51
n = 7	±1.81	±1.86	±1.85	±1.78
Group B	5.49	5.34	5.43	5.51
n = 7	±1.09	±1.00	±0.96	±1.01
Group C	5.44	5.38	5.57	5.82
n = 6	±1.43	±1.11	±1.37	±1.45
Group D	5.16	5.07	5.05	5.04
n = 6	±1.03	±1.20	±1.28	±1.20

Minutes after drug admin.	*10*	*15*	*20*	*30*
Group A	5.42		5.31	5.34
n = 7	±1.78		±1.77	±1.80
Group B	5.43		5.40	5.34
n = 7	±1.08		±1.05	±1.00
Group C	5.62	5.79		
n = 6	±1.41	±1.39		
Group D	5.11	5.41		
n = 6	±1.21	±0.88		

R.K. Mirakhur MD PHD FFARCS
Department of Clinical Anaesthesia
Royal Victoria Hospital
Belfast
Northern Ireland
C.J. Jones B PHARM MPS
A.H. Robins Co. Ltd.
Horsham, England

REFERENCES

1 *Zsigmond EK, Winnie AP, Barabas E, Wang XY.* The inhibitory effect of glycopyrrolate on human plasma-cholinesterase. Can Anaesth Soc J 1985; 32: 20–2.
2 *Mirakhur RK.* Glycopyrrolate and human plasma cholinesterase. Can Anaesth Soc J 1985; 32: 683.
3 *Ellman GL, Courtney KD, Andres VJ, Featherstone RM.* A new and rapid colorimetric determination of acetylcholinesterase activity. Biochem Pharmacol 1961; 7: 88–95.

Percutaneous sheath introducer shaft-hub disconnection during pulmonary artery catheterization

To the Editor:

We wish to report a recent problem which occurred during the insertion of a pulmonary artery catheter using an Arrow® Percutaneous Sheath Introducer Set, SI-09800.

Our patient was a 29-year-old man weighing 90 kg, scheduled for an elective aorto-coronary bypass graft. The right internal jugular vein was used for percutaneous cannulation by the Seldinger technique, and the catheter sheath inserted without difficulty and connected to the haemostasis valve with side port adapter. The balloon-tipped pulmonary artery catheter passed easily down the catheter sheath to approximately 25 cm at which time, while watching the monitor screen, the anaesthetist felt the separation of the shaft of the catheter sheath from the hub (Figure 1). As the anaesthetist was holding the shaft-hub junction, he was able to prevent the shaft from advancing into the neck. The hub and pulmonary artery