TITLE: PRERETINAL OXYGEN TENSION DURING ENFLURANE ANESTHESIA IN CATS

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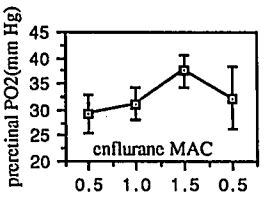
We have reported dose-dependent increases in retinal blood flow during enflurane anesthesia.[1] In this study we tested the hypothesis that this increase in blood flow results in preservation of oxygen availability in the retina during enflurane anesthesia. The measurement of preretinal oxygen tension with a microelectrode placed near the retina has been shown to reflect retinal blood flow and oxygen availability.[2]

Seven adult cats weighing 2.5-3.5 kg were studied after approval of our Animal Care Committee. The animals were anesthetized with enflurane and air-oxygen. Ventilation was controlled to maintain arterial PCO_2 at 25-30 mm Hg and PO_2 at 90-100 mm Hg. Arterial (MAP), central venous and intraocular (IOP) pressures were recorded continuously. End-tidal enflurane and CO_2 were monitored. A polarographic oxygen microelectrode was inserted through the pars plana into the vitreous and positioned within 100 μ of the retinal surface as we have described previously.[3] Multiple measurements of preretinal oxygen tension were made at 0.5, 1.0, 1.5 and upon return to 0.5 MAC enflurane concentration. Results were analyzed using repeated measures ANOVA with a correction for multiple comparisons.

There was a significant decrease in retinal perfusion pressure (MAP-IOP) between 0.5 and 1.5 MAC enflurane (98 ± 6 to 54 ± 4 mm Hg, mean ± SEM). This was accompanied by a significant increase in preretinal oxygen tension (29.1 ± 3.6 to 37.5 ± 3.2 mm Hg, $p < .02$) which returned to near baseline upon restoration of enflurane concentration to 0.5 MAC (figure).



These results indicate that increasing anesthetic depth results in a preservation of retinal oxygen availability, either as a consequence of increased blood flow or decreased oxygen utilization. Enflurane may safely be used during surgical procedures where retinal ischemia is of concern.

Supported by NEI 004071 and the Brain Research Foundation.

References

1. ARVO Abstracts, 3282, 1990
2. Exp Eye Res 32:369-379, 1981
3. Invest Ophthal Vis Sci 25:1129-34, 1984

TITLE: AGE DOES NOT CHANGE THE DYNAMICS OF ATRACURIUM REVERSAL

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The clinical dynamics of pharmacologic antagonism ("reversal," REV) of neuromuscular blockade (NMB) in elderly patients remain poorly understood. Aging impairs mobilization of acetylcholine, causes end-plate proliferation, and may hinder access of drugs to extracellular fluid.[1,2] Clinical studies are inconsistent, showing either no change or a marked delay of REV.[3] To isolate the process of REV in vivo from drug-specific effects or age-related changes in their pharmacokinetics, we studied REV with edrophonium (EDR) and neostigmine (NST) in young and elderly patients during infusion of atracurium (ATR).

Methods: With institutional approval, 12 young (17-34 yrs) and 13 older (64-85) surgical patients anesthetized with isoflurane/N2O/O2 received ATR 0.25 mg/kg by bolus. Subsequently ATR infusion (4-32 mg/hr) maintained integrated electromyographic (IEMG) evoked responses to q 20 sec ulnar nerve stimuli at 10% of unparalyzed control values. When infusion rates and NMB were stable for at least 20 min, REV was randomly accomplished with EDR 1 mg/kg or NST 0.07 mg/kg. Continuous recording provided times to onset and peak NMB with ATR bolus,times to onset and peak of REV, and IEMG response magnitude for t-test analysis.

Results: Age was not associated with a statistically significant delay in NMB or a delay in onset or peak times of subsequent REV. In both young and elderly, EDR produced the same degree of REV as NST, but in a much shorter interval (Table). Speculation regarding clinically important hindered distribution of these drugs to extracellular fluid or altered REV dynamics in the elderly is not supported. For any given degree NMB, choice of reversal drug, not patient age, determines the time required for antagonism of NMB.

References: 1. J Neurocytol (1983)12:13-25
2. Anesthesiology (1990)72:403-411
3. Anesthesiology (1984)61:A303

Table:
Mean [SE]

	young [n=12]	older [n=13]
NMB onset [sec]	70(34)	88(35)
Max NMB [% ctrl IEMG]	11(8)	13(16)
NMB peak [sec]	447(141)	517(188)

	NST		EDR	
	young	older	young	older
n=	7	6	5	7
REV onset[sec]	46(6)	43(8)	32(5)	43(3)
Max IEMG [% ctrl]	75(4)	70(6)	80(7)	67(7)
REV peak [sec]	*429(79)	*417(51)	*152(35)	*180(22)

*NST different than EDR, $p < 0.02$