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 Title : ENFLURANE ALTERS REGIONAL GLUCOSE UTILIZATION
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INTRODUCTION Low (1%) concentrations of enflurane increase glucose stores in mice¹, while high (2.5-5%) concentrations cause regional variation of glucose utilization in rats.² The following study was designed to determine if regional glucose utilization (rCMRg) is altered by low concentrations of enflurane anesthesia.

METHODS The [¹⁴C] deoxyglucose (2-DOG) method of Sokoloff³ was used to measure rCMRg. A preliminary experiment established that the published rate constants for the method are unaffected by enflurane anesthesia. 200 gm female Sprague-Dawley rats were anesthetized with enflurane and a femoral artery and vein were cannulated. Control rats were awakened 5-24 hr before the experiment. Anesthetized rats received 0.75%, 1.48%, or 2.13% enflurane for 45 min prior to rCMRg measurement. Enflurane concentrations were verified by gas chromatography. The anesthetized animals were intubated and ventilated with 100% O₂ to maintain a PaCO₂ comparable to that of control animals. For all rats, arterial blood pressure was recorded and kept at normal levels with Ringers lactate infusion; temperature was maintained with a heat lamp, and hematocrit was measured periodically. After the intravenous injection of 50 µci of 2-DOG, serial blood glucose and 2-DOG levels were measured and the rat then decapitated. Twenty-micron serial frozen brain sections were cut at -20°C and autoradiographs prepared for every fifth slice. Optical density of 6 to 16 sections per rat was measured for 18 brain regions, and rCMRg calculated.

RESULTS Control animals exhibited considerable regional variation in rCMRg; this became less apparent following enflurane exposure (Table 1). Depression of rCMRg was invariably present in gray matter structures, while rCMRg for white matter structures was increased or decreased depending on the specific structure and the anesthetic dose. When the data for white matter were pooled, there was no significant change in rCMRg regardless of enflurane concentration, while pooled data for gray matter structures demonstrated a depression of glucose uptake (Table 2).

DISCUSSION Our data suggest that clinical concentrations of enflurane cause regional variation in rCMRg. It is apparent that the neuronal structures are more sensitive to this effect than are glia. The possibility exists that there is a relationship between the phylogenetic age of an area and its sensitivity to enflurane induced metabolic depression.

REFERENCES

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metabolic rate in brain. J Neurochem 18: 2301-2316, 1971

2. Myers RR, Shapiro HM: Local cerebral metabolism during enflurane anesthesia. Electroencephalogr Clin Neurophysiol 47:153-162, 1971

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Table 1. rCMRg (% CHANGE FROM CONTROL)*

	Enflurane		
	0.75%	1.48%	2.13%
Sensory motor cortex	-15.9	-15.1	-19.1
Auditory cortex	-11.1	-21.9	-21.8
Visual cortex	-10.1	-19.0	-28.4
Parietal cortex	-34.9	-40.5	-45.4
Amygdala	-38.3	-41.1	-39.9
Hippocampus	-34.4	-35.8	-29.7
Hypothalamus	-26.2	-42.8	-24.8
Caudate-putamen	-23.5	-26.9	-43.1
Thalamus	-22.4	-32.5	-37.4
Medial geniculate	-65.3	-71.3	-71.6
Lateral geniculate	-23.0	-42.9	-34.8
Superior colliculus	-22.3	-39.3	- 8.6
Pontine gray matter	-23.9	-22.8	-24.3
Cerebellar cortex	- 1.0	-28.5	- 4.5
Cerebellar nucleus	-12.1	- 9.5	- 5.3
Truncus corpus callosum	+13.6	+21.1	+37.2
Genu corpus callosum	+ 5.2	+10.2	-13.3
Internal capsule	+ 1.7	+ 5.4	-23.9

* Mean values for 2 rats compared to mean values for 4 control rats

Table 2. GLUCOSE UPTAKE
(mg/100 mg tissue/min, means ± SE)

	CONTROL	ENFLURANE		
		0.75%	1.48%	2.13%
Gray N=90	15.9 ±1.13	12.4* ±.825	10.3* ±.662	10.7* ±.558
White N=18	7.39 ±.866	7.82 ±.758	7.61 ±.566	7.00 ±.269

* p<.0005 vs control using Student's t-test