Title : LOCAL CEREBRAL BLOOD FLOW AND METABOLISM DURING HALOTHANE AND ENFLURANE

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Introduction. Heretofore, studies describing the relationship between cerebral blood flow (CBF) and metabolism (CMR) during anesthesia have been limited to considering the entire brain. This technical limitation may be considerable as the brain is known to be a heterogeneous organ insofar as structure and function are concerned. In the present study, we employed quantitative autoradiographic techniques to measure local CBF (1-CBF) and local glucose metabolism (1-CMRg) during anesthesia with 2 different volatile agents.

Methods. 1-CBF and 1-CMRg were respectively determined in rats with the ^{14}C-iodoantipyrine and ^{14}C-deoxyglucose methods in separate groups of rats (n=5-8). Arterial blood gas status was the same in the awake controls and anesthetized rats. 1-CBF and 1-CMRg was determined in 25 structures; nine representative areas are reported herein.

Results. Table 1 summarizes our results. In anesthetized rats the arterial pressure fell between 33% to 55% while CBF remained unchanged. Therefore, the changes in 1-CMRg are the major cause of the 1-CBF/1-CMRg ratio changes shown in Figure 1.

Discussion. The average awake 1-CBF/1-CMRg ratio (CBF equivalent=CBF_{EQ}) reported in humans is about 10 and our value in awake rats ranges between 10 to 20. When anesthetic related hypotension remains uncorrected, marked differences in the CBF_{EQ} are apparent among rats anesthetized with halothane and enflurane. Thus, our study provides the first evidence that different volatile anesthetics cause differential relationships between CBF and CMR in various brain structures. While the general tendency appears to be an increase in the CBF_{EQ} during anesthesia with volatile agents (i.e., a "superflow state"), with enflurane the hippocampus CBF_{EQ} remains uniquely unchanged. This is apparent when contrasted with other structures during enflurane anesthesia as well as at 2 MAC of halothane. The low CBF_{EQ} in the hippocampus during enflurane is due to its elevated 1-CMRg which we have previously shown to be secondary to seizure activity localized in that structure. These findings suggest that substrate demand could potentially outstrip supply within the hippocampus during enflurane induced seizures. Further studies are required to answer this postulation.

TABLE 1

STRUCTURE #		AWAKE	1 MAC HALO.	1 MAC ENFL.	2 MAC HALO.	2 MAC ENFL.
1. Frontal Cortex	CBF	256	209	165	237	214
	CMRG	13.2	13.3	8.5*	3.2*	8.1
2. Parietal Cortex	CBF	256	191	178	218	179
	CMRG	13.5	12.6	9.8	3.9*	7.3*
3. Visual Cortex	CBF	243	187	146	209	162
	CMRG	12.3	13.2	8.2	3.0*	6.4
4. Basal Ganglia	CBF	183	191	181	244	169
	CMRG	13.0	15.7	10.2	4.3*	7.7
5. Medial Thalamus	CBF	207	139	140	202	203
	CMRG	13.8	12.2	7.7*	4.6*	8.8
6. Hypothalamus	CBF	130	126	149	148	187
	CMRG	9.0	9.8	6.3	2.4*	5.8
7. Hippocampus	CBF	135	103	143	145	140
	CMRG	10.1	11.6	12.4	2.9*	12.0
8. Reticular Form	CBF	182	133	207	210	242
	CMRG	10.1 *	10.5*	7.1	2.3*	5.4
9. Inferior Collic	CBF	502	253	394	391	636
	CMRG	26.2	15.2	10.1*	5.5*	8.0*

L-CBF (ml/100g/min) and 2-CMRG (mg/100g/min)in nine selected structures.
Mean values are reported with significance (p <.01) by an asterisk.