Date : May 1, 1980

Title : LACK OF CEREBRAL BLOOD FLOW CHANGE DURING KETAMINE ANESTHESIA IN VENTILATED GOATS

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Introduction. The prevailing clinical view is that ketamine administration causes an increase in cerebral blood flow (CBF) and cerebral spinal fluid (CSF) pressure, while causing an increase in cerebral metabolic rate (CMR_{O2}). However, critical examination of the literature does not support this prevailing view. Ketamine has been variously reported to cause a decrease in CBF and CMR_{O2},[1] an increase in CBF and CMR_{O2},[2] and to have little effect on CBF and CMR_{O2}.[3] Likewise, CSF pressure has been reported both to be elevated or unchanged following ketamine treatment. It was the intent of this study, therefore, to clarify these ambiguities.

Methods. A previously developed method for measuring CBF and CMR_{O2} in both awake and anesthetized goats was employed. In 8 adult, female goats an electromagnetic flow probe was placed around the internal maxillary artery. Next, a small hole was made in the top of the skull and a catheter was inserted into the superior saggital sinus. The hole was then sealed and the catheter anchored with dental cement. Catheters were also implanted in the femoral artery and vein. Following surgery the animals were permitted to recover 7-10 days. On the day of experimentation the goats were studied in one of two ways: paralyzed with 0.05 mg/kg of pancuronium, or unparalyzed. In either situation control values for CBF and CMR_{O2} were first established while the animal was awake and unrestrained. Following this, ketamine (5 mg/kg) was administered intravenously and CBF and CMR_{O2} measurements were made at various intervals following injection.

Results. In paralyzed animals ketamine had (by 5 minutes) no significant effect on CBF and produced a 12% decrease in CMR_{O2} (Table 1). In non-paralyzed subjects ketamine produced a 41% mean increase in CBF and a 10% reduction in CMR_{O2}. However, in the non-paralyzed group not all animals responded in this fashion i.e., some animals on occasion showed no change or a slight decrease in CBF (in the aggregate the increase in CBF was statistically significant). Examination of mean arterial blood pressures (MABP) and blood $PaCO_2$ levels revealed significant elevations in these parameters in non-paralyzed goats as compared to paralyzed.

Discussion. It was concluded from these studies that ketamine has no significant effect on CBF and produced only a slight decrease in CMR_{O2} in situations where ventilation was controlled. The ambiguity of previous reports apparently stems from the fact that non-paralyzed experimental subjects were employed and no provisions were made for changes in blood $PaCO_2$ and MABP. In addition, the data suggests that increases in CSF pressure during ketamine anesthesia could be minimized with adequate ventilation.

TABLE 1

	CBF ml/100g/min	CMR_{O2} ml_{O2}/100g/min	PCO_2 torr
Paralyzed			
Awake	58±14*	4.34±0.81	29±3
5 min.	65±17	3.81±0.77	30±4
10 min.	65±15	4.05±0.84	30±3
Recovery	61±20	4.31±0.85	29±5
Non-Paralyzed			
Awake	68±19	4.28±0.66	32±3
5 min.	105±41+	3.84±0.73	38±5+
10 min.	90±31+	4.02±0.81	38±5+
Recovery	74±22+	4.04±0.78	35±3

*All values mean ±SD
+Significantly different from paralyzed group, $P < 0.05$, n=8

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