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TITLE: THE EFFECTS OF FENTANYL AND SUFENTANIL ON ICP AND CBF IN RABBITS WITH AN ACUTE CRYOGENIC BRAIN INJURY

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Previous work has shown that sufentanil may increase cerebral blood flow (CBF) in dogs.[1] Clinical studies have demonstrated that sufentanil may also increase cerebrospinal fluid pressure in some patients with brain tumors.[2] However, a randomized clinical trial comparing sufentanil and fentanyl in neurosurgical patients could find little difference between these two narcotics.[3] The present study was undertaken to compare the effects of fentanyl and sufentanil on cerebral hemodynamics and CBF in a model of acute brain injury.

Following institutional review and approval, 19 New Zealand white rabbits were studied. Animals were anesthetized in a plexiglass box with 4% halothane and their tracheas intubated. Anesthesia was maintained with 1% halothane and muscle paralysis with pancuronium. Normocapnia was maintained at all times. Animals were surgically prepared with femoral arterial and central venous catheters, and a ventriculostomy for measurement of intracranial pressure (ICP). Monitored variables included heart rate, mean arterial pressure (MAP), ICP, temperature (servocontrolled to 37° C), arterial blood gases, end-tidal CO_2 and ET halothane. Global CBF was measured utilizing the hydrogen clearance technique. After recording baseline variables, a cryogenic injury was produced over the left hemisphere with liquid nitrogen. Sixty minutes following brain injury, animals were randomized to receive one of the following five minute infusions: FENTANYL (200 µg/kg) in 20 cc of normal saline (NS), SUFENTANIL (20 µg/kg) in 20 cc of NS, or PLACEBO - 20 cc of NS. Physiologic variables, ICP and CBF were compared between groups with ANOVA. Within groups, paired t-tests were used to test for differences in MAP, ICP and CBF.

Arterial blood gas values were not different between groups at any time point. Cryogenic injury increased ICP significantly in all animals from 5±2 mm Hg to 16±8 mm Hg (pre-infusion), with no differences between groups. ICP increased significantly following infusion of both narcotics and NS. However, there were no significant differences in ICP between the three groups at any time. CBF decreased significantly following the administration of sufentanil (paired t-test), but there were no differences in CBF between groups. At no time were any significant differences noted in MAP between groups (Table).

Our data indicate that equipotent doses of sufentanil or fentanyl in this model of brain injury do not cause an increase in ICP compared to control animals receiving normal saline. The decrease in CBF following sufentanil, though statistically significant, was modest and probably of little clinical significance. These data agree with other animal and human studies that have found few or no important differences between fentanyl and sufentanil with regards to intracranial or systemic effects.

REFERENCES: 1.Milde LN *et al*: Anesth Analg 70:138-146, 1990 2. Marx W *et al*: J Neuros Anes 1:3-7,1989 3. From RP *et al*: Anesthesiology 73:896-904, 1990.

	MAP (mmHg)		ICP (mmHg)		CBF (cc/100 gm/min)	
	PRE	POST	PRE	POST	PRE	POST
FENTANYL	73±5	63±8	16±5	21±7*	65±10	58±18
SUFENTANIL	76±6	65±7*	18±10	21±12*	71±12	50±13*
PLACEBO	75±5	73±5	14±8	16±9*	72±21	67±17

* Significant change from pre-infusion value, p<0.05

A619

TITLE:COMPARISON OF SODIUM NITROPRUSSIDE AND LABETALOL ON THE INTRACRANIAL PRESSURE OF CATS

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INTRODUCTION: The commonly used antihypertensive agent sodium nitroprusside (SNP) increases intracranial pressure and causes tachycardia. Labetalol (La) is also an antihypertensive agent with α and β advenergic blocking activity;(1) but how it affects intracranial pressure in comparison to SNP is yet to be determined. We, therefore, compared the effect of La and SNP on the intracranial pressure and heart rate of cats.

METHODS: Ten cats (2.5-3.5 kg) were anaesthetized the trachea intubated and lungs ventilated with a respirator. A 7-French gauge catheter was inserted via a trephined hole in the left-parietal area to the subarachnoid space for ICP monitoring. In one group; (five cats) normal ICP (N-ICP) was maintained. In the other group, a 5-mm hole was trephined in the right parietal area and a 10-French gauge Foley catheter was placed in the extradural space. The balloon of Foley catheter was inflated until the ICP reached 27±2 mm Hg and remained constant (AI-ICP). Mean arterial pressure, heart rate (HR), ICP, ECG and endtidal carbon dioxide concentration were monitored continously. Rectal and brain temperatures were maintained at 37±0.5C. La or SNP was infused to decrease the blood pressure of each cat by approximately 30%. La was administered to five of the normal intracranial pressure cats and to five with artificially increased intracranial pressure. An equal number of cats with normal intracranial pressure and artificially increased intracranial pressure were infused with SNP. Before and during infusion of the vasodilators, blood pressure, electrocardiogram and highest intracranial pressure were recorded. Intragroup differences were analyzed by the student's paired t-test and intergroup difference by analysis of variance. P values less than 0.05 were considered signifcant.

RESULTS: The results are summarized in the table.

TABLE

Normal ICP (n=10)

	Before - Labetalol	Before - SNP
BP	105±7 *70±8	110±5 *75±5
ICP	6±0.3 6.2±0.6	6±.2 7±.2
HR	162±18 *147±20	159±20 *178±12

Increased ICP (n=10)

	Before - Labetalol	Before - SNP
BP	112±6 *77±8	108±5 *74±8
ICP	29±.5 29.5±.6	28±.6 *40±.8
HR	165±16 *146±18	150±12 *170±20

Blood pressure (BP) & intracranial pressure ICP in mm Hg and heart rate HR in beats per minute. * ($P < 0.05$) significant.

DISCUSSION: In our experiment, there was no significant increase in intracranial pressure during La infusion for cats with normal or increased intracranial pressure, whereas, sodium nitroprusside increased the ICP of cats with artificially increased ICP. We therefore conclude that if an antihypertensive agent is required in patients with intracranial hypertension, La may be an appropriate drug to select.

References: The Pharmacological Basis of Therapeutics. Eds. Pg. 933.