TITLE:

THE COMPARATIVE CARDIOVASCULAR DEPRESSION OF HALOTHANE IN NEONATES AND

**INFANTS** 

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Cardiovascular depression during halothane anesthesia is believed to be more profound in neonates than in older infants. The purpose of this study was to measure and compare cardiovascular changes at equal MAC halothane levels in neonates (n=10) and two groups of infants 1-6 mo (n=14) and 6-12 mo (n=14) of age and to predict differences in cardiovascular depression at equal end-expired halothane levels.

Using two-dimensional and pulsed Doppler echocardiography, left ventricular dimensions and cardiac output were measured in 38 unmedicated infants prior to anesthesia induction. A second cardiovascular measurement was recorded with end-expired halothane concentrations maintained at 1.0 MAC (adjusted for age) for ten minutes. After inspired halothane concentrations had been increased to achieve 1.5 MAC end-expired levels cardiovascular measurements were repeated. Analysis of variance was used to compare cardiovascular variables at equal MAC levels. In addition, an analysis of covariance was used to assess cardiovascular differences at equal end-expired halothane concentrations.

Cardiac index (CI), stroke volume index (SVI) and ejection fraction (EF) declined similarly and significantly from awake values at 1.0 MAC and 1.5 MAC in all three groups. The decreases in CI, SVI and EF were significantly greater at

1.5 MAC (approximately 35% from awake levels) than at 1.0 MAC in all groups of neonates and infants. Heart rate (HR) decreased significantly at 1.0 MAC in infants but not in neonates.

Based on analysis of covariance, the slope of decline in CI and SVI were significantly greater in neonates than infants when compared at equal end-expired halothane concentration. Heart rate were similar when estimated at equal end-expired halothane concentrations.

When halothane concentrations are compared at equal MAC levels, the cardiovascular depression produced by halothane is similar in neonates and in infants; however, at equal end-expired halothane levels neonates experience greater cardiovascular depression.

		AWAKE	1.0 MAC	1.5 MAC
NEONATE	HEART RATE	1444 ±4.9	143.1 ± 4.1	134.9*† ±5.3
	CARDIAC INDEX	3.70 ±0.37	2.71** ±0.26	2.16*† ±0.24
INFANTS 1-6 MONTHS	HEART RATE	152.1 ±3.9	138.4 * ±3.8	130.4*† ±4.0
	CARDIAC INDEX	3.60 ±0.29	2.94* ±0.24	2.49*† ±0.20
INFANTS 6-12 MONTHS	HEART RATE	128.3 ±3.3	119.0* <u>+</u> 4.2	110.9 * † ±4.2
	CARDIAC INDEX	3.14 ±0.14	2.46 * ±0.21	2.28*† ±0.16

Results are expressed as mean ± SEM. \* p < 0.05 from awake + p < 0.05 from 1.0 MAC

## A1133

TITLE:

PEDIATRIC AIRWAY PRESSURE RELEASE

VENTILATION

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Airway pressure release ventilation (APRV) augments alveolar ventilation intermittently decreasing lung volume while maintaining oxygenation. Infants are at particular risk of airway closure and hypoxemia. Conventional mechanical ventilation (CMV) in neonates is associated with both acute barotrauma and chronic lung disease. APRV ventilates without high peak inspiratory pressures (Pip), thus it may be well suited for infants. This study determined the feasibility of APRV in neonatal lambs with acute lung injury (ALI) and assessed release durations.

Seven lambs (<10 days old) were anesthetized and instrumented. ALI was induced with oleic acid (165 mcg/kg IV) over 2 hours. Mean Paw, Pip, mean arterial pressure (MAP), cardiac output (CO) and arterial pH, pCO2, pO2 were measured during continuous positive airway pressure (CPAP), APRV, and CMV at equal mean Paw. Three lambs were studied with varying durations of airway pressure release (0.2 to 1 sec). Data were analyzed by analysis of variance.

In this pediatric animal model of ALI, APRV maintained oxygenation and augmented alveolar ventilation as well as CMV, but at lower Pip and without compromising cardiovascular function (Table 1). In this model, PaO<sub>2</sub> and PaCO<sub>2</sub> were optimal with airway pressure release times of 0.4 to 0.6 seconds (Table 2).

Table 1	<u>CPAP</u>	APRV	<u>CMV</u>
PaO <sub>2</sub> (torr)	73 <u>+</u> 6	77 <u>+</u> 7	89 <u>+</u> 8
PaCO,(torr)	41 <u>+</u> 2*	29 <u>+</u> 2	29 <u>+</u> 1
рH	7.34 <u>+</u> 0.01	7.46 <u>+</u> 0.03	7.45±0.02
mean Paw	13.4 <u>+</u> 1.5	13.5 <u>+</u> 1.4	$13.9 \pm 1.4$
Pip(cm H <sub>2</sub> O)	13.4 <u>+</u> 1.5*	19.7 <u>+</u> 1.7	36.4±3.2*
CO(L/min)	1.2 <u>+</u> 0.2	1.2±0.2	0.9 <u>+</u> 0.1*
MAP(mm Hg)	94 <u>+</u> 7	96 <u>+</u> 6	91 <u>+</u> 7*
(mean + SEM)	* <b>-</b> p <	.05 compared	to APRV

Table 2 Release time (seconds)

	<u>0</u>	0.2	0.4	0.6	0.8	1.0	
PaO,(torr)	84 <u>+</u> 5	89 <u>+</u> 10	97±10	93 <u>+</u> 11	93 <u>+</u> 13	88 <u>+</u> 14	
PaCO, (torr)	) 39 <u>+</u> 4	36 <u>+</u> 4	35 <u>+</u> 2	33 <u>+</u> 4	34 <u>+</u> 5	36 <u>+</u> 7	
pН	7.30	7.33	7.37	7.36	7.36	7.33	
	±.03	±.02	±.01	±.03	±.04	±.05	
mean Paw	14 <u>+</u> 3	12 <u>+</u> 3	11 <u>+</u> 3*	10 <u>+</u> 3*	9 <u>+</u> 2*	8 <u>+</u> 2*	
Pip(cm H <sub>2</sub> O)	)14 <u>+</u> 3	14 <u>+</u> 3					
(mean ± SE			< .05	compa	red to	CPAP	