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TITLE: DIFFERENTIAL SENSITIVITY TO HALOTHANE ANESTHESIA OF THE GENIOGLOSSUS, INTERCOSTALS, AND DIAPHRAGM: A COMPARISON BETWEEN KITTENS AND ADULT CATS

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Recent studies in humans and animals have indicated that different inspiratory muscles have different sensitivities to respiratory depressants. The sensitivity of inspiratory muscles during early growth and development compared with that in adults of the same species, however, has not been studied. We studied the activity of the diaphragm, the external intercostals, and the genioglossus by means of electromyography (EMG) and its moving time average (MTA) with different concentrations of halothane in seven 2-month-old kittens, and compared these data with similar data from cats.[1] The kittens spontaneously breathed 1.0%-2.0% halothane in O_2 (adult cats, 1.0%-3.0%), while arterial PCO_2 was maintained at about 60 mm Hg at all concentrations of halothane by adding CO_2 to the inspired gas as needed. Muscle activity was evaluated in terms of the peak height of MTA, with activity at 1% halothane used as the control measurement. In kittens, as in adult cats, halothane anesthesia significantly decreased inspiratory muscle activity in a dose-dependent fashion: most in the genioglossus (GG), less in the intercostals (IC), and least in the diaphragm (Table). Genioglossal activity was abolished at 1.5% and 2.0% halothane in kittens, but was depressed but preserved in cats. The average decrease in diaphragmatic and intercostal activity at the same anesthetic concentration was more pronounced in kittens than in adult cats, but the difference was not significant. Considering the fact that the minimum alveolar concentration for halothane determined in kittens (1.39±0.03%) was significantly greater than that in cats (1.21±0.05%), it is probable that at equal depths of anesthesia these muscles as well may be more depressed in kittens than in cats. In both age groups, respiratory frequency, inspiratory time (T_I), and inspiratory duty cycle (T_I/T_{TOT}) did not change significantly with increasing concentration of halothane.

Table. Effect of Halothane Anesthesia on Inspiratory Muscle Activity° in Kittens (n=7)

Conc. (%)	Diaphragm (costal)	Diaphragm (crural)	IC	GG
1.5	61.8 ± 7.3	71.8 ± 5.8	68.9 ± 9.6	0.0 ± 0.0*
2.0	54.5 ± 6.2	66.6 ± 4.5	35.4 ± 8.8*	0.0 ± 0.0*

°Expressed as percent of peak height of moving time average from that at 1% (control). *p < 0.01

Reference

1. Ochiai R, Guthrie RD, Motoyama EK: Anesthesiology 70:812-816, 1989.

A926

TITLE: **Direct Depression of Myocardial Function by Halothane in the Isolated, Perfused Infant Rabbit Heart**

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Most information about the effects of anesthesia in newborns has been obtained from intact animals and humans in which multiple factors (autonomic reflexes, alterations in preload and afterload, and direct anesthetic effects) interact to produce observed effects. In this study we used an isolated, perfused heart to examine direct myocardial effects of halothane in infant rabbits.

This study was approved by the institutional Animal Care Committee. Eight rabbits, 8-14 days old, were anesthetized with thiamylal, a thoracotomy was performed, and the aorta was cannulated. The heart was isolated and perfused retrogradely (Langendorff preparation) with oxygenated Krebs' solution at 36°C. Stimulating and recording electrodes were placed in the right atrial appendage and the right ventricle. A saline-filled latex balloon was inserted through the left atrium and mitral valve into the left ventricle to record left ventricular pressure (LVP) isovolumetrically. Pressure was electronically differentiated to determine maximal rate of contraction (+dP/dt) and relaxation (-dP/dt). Coronary flow (Q) was measured by timed collections of coronary effluent. Halothane was equilibrated with the Krebs coronary perfusate. Three doses of halothane were used and control measurements were made before and after each dose. For each anesthetic dose and control, 20 min was allowed for equilibration. Heart rate was recorded and then the atrium was paced at a rate slightly higher than the intrinsic atrial rate. During pacing we determined: A-V time, LVP, (+)dP/dT, (-)dP/dT, and coronary Q.

Halothane directly depressed myocardial function and increased coronary flow in a dose-dependent manner in infant rabbit hearts (Fig. 1). At the highest dose, contractility (LVP and dP/dt) was profoundly depressed while electrophysiological indices (heart rate and A-V time) and coronary Q were only mildly altered.

Further studies are necessary to elucidate the mechanisms of halothane depression and to compare these effects to older animals.

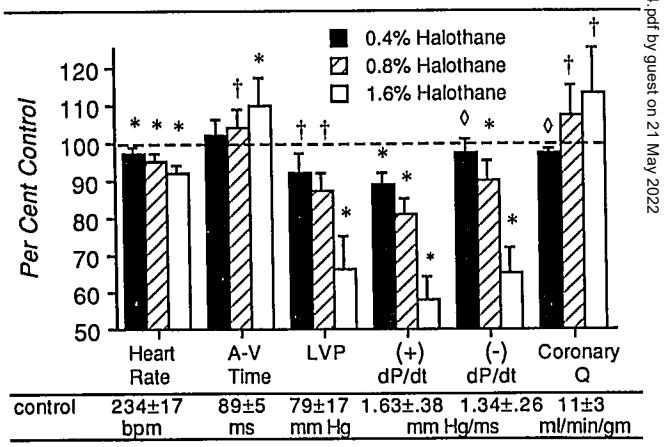

Figure 1. Effects of halothane on myocardial function. Values are mean ± sd. * p<0.05 *vs* control (paired t-test) and *vs* all other doses (repeated measures ANOVA). † different from control only. ◊ different from other doses only.