

●194 NEURAL REGULATION OF THE PULMONARY CIRCULATION FOLLOWING SYSTEMIC HYPOTENSION IN CONSCIOUS DOGS. Paul A. Murray, Patrick W. Clougherty, Daniel P. Nyhan, Bessie B. Chen, Harold M. Goll (Spon. Mark C. Rogers). The Johns Hopkins Medical Institutions, Department of Anesthesiology and Critical Care Medicine, Baltimore, MD 21205.

To investigate autonomic nervous system (ANS) regulation of the pulmonary vascular response to increasing cardiac index (CI:ml/min/kg) following systemic hypotension (H), the pulmonary vascular pressure gradient (pulmonary arterial-pulmonary capillary wedge pressure:  $\Delta P$ ) was measured at multiple levels of CI during stepwise inflation and deflation of an inferior vena cava (IVC) occluder. In intact dogs, maximum IVC constriction decreased ( $p < 0.01$ ) CI from  $139 \pm 9$  to  $46 \pm 3$ , and systemic arterial pressure from  $108 \pm 2$  to  $55 \pm 3$  mmHg. Following 15 minutes of H, CI was gradually increased by deflation of the IVC occluder. Surprisingly,  $\Delta P$  was not significantly changed at any level of CI following H in intact dogs or after cholinergic block (atropine 0.1 mg/kg). In contrast,  $\beta$  adrenergic block (propranolol 1 mg/kg) increased  $\Delta P$  at every level of CI following H, e.g. at CI = 100,  $\Delta P$  was increased ( $p < 0.01$ )  $16 \pm 2\%$  from  $10.2 \pm 0.5$  mmHg. Pulmonary vasoconstriction following H was not observed during total autonomic ganglionic block (hexamethonium 30 mg/kg), i.e.  $\Delta P$  at CI = 100 was slightly decreased ( $p < 0.05$ )  $6 \pm 2\%$  from  $9.7 \pm 0.8$  mmHg following H. These results suggest that the pulmonary circulation is actively modulated by the ANS following H. ANS-mediated vasodilator and vasoconstrictor influences appear to offset one another in the intact and cholinergic blocked conscious dog.

●195 RELATIONSHIP OF DIAPHRAGMATIC CONTRACTILITY TO DIAPHRAGMATIC BLOOD FLOW IN A NEWBORN MODEL. David G. Nichols, Sandra Howell, Jody Massik, Raymond C. Koehler, Christine A. Gleason, James R. Buck, Richard J. Traystman, James L. Robotham. Johns Hopkins Medical Institutions, Department of Anesthesiology/Critical Care Medicine, Baltimore, MD.

We determined the relationship of diaphragmatic contraction rate to diaphragmatic blood flow ( $Q_{di}$ ), metabolism, and contractility in 6 newborn lambs. The diaphragm was paced for 15 min. at slow (SC, 20/min) and fast (FC, 100/min) contraction rates each followed by a 30 min. recovery period in open-chested, mechanically ventilated lambs.  $Q_{di}$  was measured with radiolabeled microspheres. Transdiaphragmatic pressure ( $P_{di}$ ) was measured with the abdomen tightly casted to preserve length and geometry of the diaphragm. Diaphragmatic fatigue was defined as a reduction in  $P_{di}$  at stimulation frequencies of 10-100 Hz. With SC there was a mild reduction in  $P_{di}$  at all stimulation frequencies.  $P_{di}$  was profoundly reduced at all stimulation frequencies during FC with only partial recovery. During SC there was a significant increase compared to control in  $Q_{di}$  (273%), diaphragmatic  $O_2$  delivery ( $OD_{di}$ , 273%),  $O_2$  consumption ( $VO_{2di}$ , 593%), and fractional  $O_2$  extraction ( $E_{di}$ , 188%). During FC there was a further increase in  $Q_{di}$  (332%),  $OD_{di}$  (331%), and  $VO_{2di}$  (729%). These increases were also significantly different from SC ( $p < 0.05$ ).  $E_{di}$  was increased compared to control (188%) but similar to SC. Lactate gradient (lac v-a) remained unchanged. We conclude that diaphragmatic fatigue at fast contraction rates is unrelated to a limitation in aerobic metabolism, as the muscle is able to increase  $OD_{di}$  and  $VO_{2di}$  while lac v-a remains unchanged.

†196 PROSPECTIVE STUDY OF POST INTUBATION LARYNGEAL EDEMA (LE) TREATED WITH AEROSOLIZED EPINEPHRINE (AE). Jacob N. Nutman, Kathleen M. Deakins, Karen K. Baldesare, Madolin K. Witte (Spons. J. L. Blumer) Case Western Reserve University School of Medicine, Rainbow Babies & Childrens Hospital, Department of Pediatrics, Cleveland, Ohio.

Although AE is commonly used in treating post intubation LE, the efficacy and side effects of this therapy in pediatric patients (pts) have not been studied. We, therefore, undertook this study to assess AE therapy in post-intubation stridor in a Pediatric Intensive Care Unit (PICU). Pts with a previous history of stridor were excluded. Following extubation, pts demonstrating clinically significant stridor and a croup score (CS)  $> 4$  points received AE. Heart rate (HR), mean arterial pressure (MAP), respiratory rate (RR), and CS were recorded at 20, 40 and 60 min and 4 hrs. Additional AE was administered as needed. Pts were followed until discharged from the PICU. The diagnoses, duration of intubation, admission TISS score and outcome were also recorded. We studied 27 pts, aged 1 to 36 mths. Diagnosis was airway disease in 26% and lung disease in 22%. Mean duration of intubation was  $5.8 \pm 5$  days and mean TISS  $38 \pm 19$ . Post extubation stridor developed within  $38 \pm 51$  min. Initial CS was  $6.8 \pm 2.9$ , and best score achieved with AE within 60 min was  $2.9 \pm 2.3$  ( $p < 0.01$ ). CS at 4 hrs was  $2.0 \pm 2.5$ . CS decreased by  $\geq 2$  points after AE in 23 pts (85%). Those that did not improve had initial CS of  $7.5 \pm 5.7$  (not different from the overall group). Four pts needed reintubation, but in only one was this felt to be due to LE. 14 pts (54%) required 2 or more additional AE. There were no significant differences in CS ( $7.2 \pm 6$  vs  $6.1 \pm 2.2$ ), TISS ( $38 \pm 17$  vs  $34 \pm 23$ ), or duration of intubation ( $6.6 \pm 5.9$  vs  $4.1 \pm 2.7$ ) in pts requiring multiple AE vs those requiring only one additional AE. No significant change in HR or MAP following AE was seen. We conclude that AE is effective in treating symptomatic post-intubation LE. Duration of intubation, initial TISS and CS did not predict severity of LE, response to AE or need for additional AE. The efficacy and safety of AE in post-intubation LE make it a useful therapy in patients with this disorder.

†197 PULMONARY VASCULAR RESPONSE TO INCREASING CARDIAC INDEX FOLLOWING SYSTEMIC HYPOTENSION IS MODIFIED BY PENTOBARBITAL ANESTHESIA. Daniel P. Nyhan, Bessie B. Chen, Harold M. Goll, Patrick W. Clougherty, and Paul A. Murray (Spon. Mark C. Rogers) The Johns Hopkins Medical Institutions, Department of Anesthesiology and Critical Care Medicine, Baltimore, MD 21205.

We examined the effect of sodium pentobarbital (PB) anesthesia on the pulmonary vascular response to increasing cardiac index (CI) following systemic hypotension (H). The pulmonary vascular pressure gradient (pulmonary arterial-pulmonary capillary wedge pressure:  $\Delta P$ ) was measured at multiple levels of CI (ml/min/kg) during stepwise inflation and deflation of an inferior vena cava (IVC) occluder in 10 conscious dogs, and again during PB anesthesia (30 mg/kg, iv). Maximum IVC constriction decreased ( $p < 0.01$ ) CI ( $131 \pm 9$  to  $46 \pm 4$  vs  $120 \pm 8$  to  $39 \pm 4$ ) and systemic arterial pressure ( $113 \pm 2$  to  $51 \pm 2$  mmHg vs  $94 \pm 4$  to  $47 \pm 2$  mmHg) in conscious and PB dogs, respectively. Following 15 min of H, CI was gradually increased by deflation of the IVC occluder. Compared to values obtained during inflation of the IVC occluder,  $\Delta P$  was not significantly changed at any level of CI following H in conscious dogs. For example,  $\Delta P$  was  $6.0 \pm 0.5$  mmHg before and  $6.0 \pm 0.6$  mmHg after H at CI = 60, and  $11.7 \pm 0.7$  mmHg before and  $11.8 \pm 0.8$  mmHg after H at CI = 120. In contrast, pulmonary vasoconstriction was observed following H during PB, i.e.  $\Delta P$  was increased ( $p < 0.01$ )  $24 \pm 5\%$  from  $6.1 \pm 0.4$  mmHg at CI = 60, and  $30 \pm 7\%$  from  $10.5 \pm 0.6$  mmHg at CI = 120. Thus, pulmonary vascular regulation following H is altered by PB anesthesia.

198 URINARY VASOPRESSIN IN CENTRAL NERVOUS SYSTEM INSULTS. Cuadalupe Padilla\*, John A. Leake\*, Robert Castro\*, M. Gore Ervin\*, Michael G. Ross\*, Rosemary D. Leake, Delbert A. Fisher, UCLA School of Medicine, Harbor/UCLA Medical Center, Departments of Pediatrics and Obstetrics, Torrance, CA.

Insults to the central nervous system (CNS) are frequently associated with the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). There are few reports of serum vasopressin (VP) values in meningitis but none in head trauma (HT) in pediatric patients. We measured urine VP by RIA, urine (U) and serum (S) osmolality (Osm) and sodium in 18 nonhypoxic pediatric patients with CNS insults within 24 hours of hospitalization and daily x2. 7 patients experienced HT, 7 bacterial meningitis (BM), and 4 aseptic meningitis (AS). Mean ( $\pm$  SEM) urine VP (pg/ml) results were as follows:

	Hosp. Day 1	Day 2	Day 3
HT (n=7) range 2-15 y	$369 \pm 196$	$78 \pm 24$	$58 \pm 21$
BM (n=7) range 0-15 mo	$84 \pm 73$	$36 \pm 13$	$38 \pm 20$
AS (n=4) range 1-10 mo	$21 \pm 15$	$15 \pm 6$	$6 \pm 1$
Controls (n=12) range 0-13 y	$14 \pm 3$		

Pediatric patients with CNS insults demonstrated elevated urine AVP levels. Defining SIADH as (S)Na $<135$ , (U)Na $>25$ , S(Osm) $<280$ , S(Osm) $<U(Osm)$ , 43% of HT patients, 29% of BM and none of AS exhibited the syndrome sometime during the 3 hospital days. Conclusions: 1) the prevalence of SIADH is high in HT and BM but not in AS, 2) urine VP is a reliable indicator of SIADH in hyponatremic patients.

†199 AIRWAY PATHOLOGY AFTER PROLONGED HIGH-FREQUENCY JET VENTILATION. Mark J. Polak, Richard L. Bucciarelli, and William H. Donnelly. (Spon by W.H. Drummond) University of Florida School of Medicine, Shands Hospital, Depts. of Pediatrics and Pathology, Gainesville.

High-frequency ventilation has become a valuable tool in the management of neonates with severe pulmonary disease. Along with reports of clinical successes, have come reports of serious complications, primarily mucous obstruction of the airways and necrotizing tracheitis. Airway histopathology from four infants who died after prolonged (ave., 8 days), high-frequency jet ventilation (HFJV) with the Mallinkrodt Bunnell Life-Pulse Jet Ventilator were compared to 12 matched control infants who died after conventional mechanical ventilation (CMV). Each of the four HFJV treated infants was matched to three CMV treated infants for birth weight, gestational age, date of birth and duration of mechanical ventilation. The 4-point, 9-variable, histologic scoring system of Ophoven et al. was used to score histologic changes at the level of the larynx, mid-trachea, carina, and right and left main bronchi. Total injury scores and patterns of acute and chronic injuries were compared using the Wilcoxon rank-sum test for nonparametric measurements. We found no statistically significant differences in total injury scores, or in patterns of acute or chronic injury at the levels of the larynx, trachea, carina, and right and left bronchi. Our results are in contrast with previously published data. The differences may be related to improved humidification systems provided by the most recent generation of jet ventilators.