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CORRELATION OF PNEUMOGRAM FINDINGS WITH CLINICAL APNEA/BRADYCARDIA. Harold R. Bigger, Howard W. Needelman, Laura S. Bradford (Spon. by Joseph R. Christian) Rush Medical College, Rush-Presbyterian-St. Luke's Medical Center, Department of Pediatrics, Chicago.

The pneumogram (PCG) has become a common test to evaluate clinical apnea/bradycardia (A/B) in infants. We compared 12 hour PCG results with evidence of A/B. Over an 11 month period, 305 PCGs were evaluated at RPSLMC, representing a heterogeneous group of NICU patients, Near-Miss SIDS, and SIDS Sibs, both on and off theophylline. Individuals were often represented more than once. Patient charts were reviewed for evidence of A/B within 2-3 days of the PCG. Insufficient clinical data resulted in deletion of 88 PCGs, leaving 217 PCGs for analysis. Of the 65 PCGs coincident with A/B, 56 were abnormal. Of the 152 coincident with no A/B, 65 were abnormal. Individual parameters correlated as follows:

Clinical Apnea	A _D		PB		Apnea ≥15 Second		Bradycardia	
	<0.5	≥0.5	<3.5	≥3.5	Present	Absent	Present	Absent
No	100	52	133	19	9	143	19	133
Yes	30	35	52	13	22	43	38	27
	p<0.05		N.S.		p<0.05		p<0.05	

Mean PB in those with A/B was 1.8% total sleep time and in those without was 1.7% (N.S.). Mean A_D in those with A/B was 0.92% and in those without was 0.65% (p=0.05). In this population, we find: 1) most patients with clinical A/B have an abnormal PCG; 2) an abnormal PCG does not predict clinical A/B; 3) PB did not discriminate infants with clinical A/B.

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VASOPRESSIN DECREASES LUNG WATER IN FETAL LAMBS. Richard D Bland, Candice D Fike, William G Teague, David Braun, and Lanny C Keil. Cardiovasc Res Inst, Dept Pediatr, Univ California, San Francisco, CA

Lung liquid volume in fetal lambs decreases before birth, as systemic arterial pressure and plasma protein concentration increase (Bland et al, J Appl Physiol 53:992, 1982). These developments may be the result of hormonal changes that occur late in gestation. Plasma concentrations of vasopressin increase several hours before birth in fetal lambs (Stark et al, Biol Neonate 35: 235, 1979) and intravenous infusion of arginine vasopressin causes reabsorption of fetal lung liquid (Perks & Cassin, Chest 81:63S, 1982). In chronic studies of 9 fetal lambs we found that plasma vasopressin, measured by radioimmunoassay, increased from $2.3 \pm .4$ pg/ml before labor to 37.2 ± 9.2 pg/ml late in labor. In lambs killed during labor, there was an inverse relationship between lung water content and plasma vasopressin concentration. To see if this hormone might contribute to the prenatal decrease in fetal lung liquid, we intravenously infused either arginine vasopressin (treated) or isotonic saline (control) for 8 h in 4 sets of twin fetal lambs at 90-95% of term gestation. Plasma vasopressin concentrations in treated lambs were similar to those measured in lambs during labor. Infusion of vasopressin increased systemic arterial pressure by 9 ± 3 torr and plasma protein concentration by 3 ± 1 mg/ml. Extravascular lung water, measured gravimetrically, was significantly less in treated twins than in controls (11.9 ± 1.0 vs 13.8 ± 1.0 g/g dry lung). Thus, vasopressin may contribute to the hemodynamic changes and reduction in lung liquid that occur in lambs before birth.

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XANTHINE ACTIONS IN GUINEA PIG TRACHEA. NJ Braden and PD Walson, Clin Pharm/Tox, Dept. Peds., Children's Hosp., Col., OH

Theophylline (T) is a widely used antiasthmatic agent with significant toxicities: its mechanisms are not well understood. Therapeutic concentrations of T do not inhibit phosphodiesterase effectively but do antagonize adenosine (A). Antagonism of A correlates to T toxicity but does not seem to be required for the more potent antiasthmatic actions of the related xanthine enprofylline (E). Guinea pig tracheal rings were used to study xanthine actions while eliminating the effects of A on vascular smooth muscle. The data shown are mean $\pm$ changes $\pm$ 1SD, where a)=response to 10μ M carbachol, b)=the baseline itself, n=number of rings and positive values represent contraction.

	150 μ M T (n)	30 μ M E (n)	100 μ M A (n)
a)	-16.6 ± 3.0 (11)	-11.3 ± 1.9 (8)	-1.7 ± 1.0 (4)
b)	-19.6 ± 5.0 (12)	-28.3 ± 3.8 (4)	$+4.5 \pm 1.5$ (4)

With controlled pH (7.20 to 7.25 in Tyrodes), resting tension (2g) and equilibration (1.5-2.0 h), T and E but not A effects were significant (p=.01).

These data in the isolated airway are different than previously demonstrated in the perfused lung but still suggest that antiasthmatic actions of xanthines may be independent of A. Therefore the use of E may avoid A antagonism and perhaps xanthine toxicity.

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RESPIRATORY DYSRHYTHMIA: A NEW CAUSE OF CENTRAL ALVEOLAR HYPOVENTILATION (CAH). Robert T. Brouillette, Carl E. Hunt, and Gail E. Gallemore. Northwestern University and East Tennessee State University, Children's Memorial Hospital and Johnson City Medical Center Hospital, Department of Pediatrics, Chicago, IL and Johnson City, TN.

Normal mammalian breathing is characterized by the rhythmic, alternating activation of inspiratory and expiratory muscles. In previously described patients with CAH, neural output to respiratory muscles during sleep has been characterized by an inadequate rate or depth of breathing. In the present case, CAH appeared to result from a disorganization of neural output to inspiratory and expiratory muscles. SB was a normal infant until he developed aseptic meningitis at age five months. Persistent brainstem dysfunction at 16 months was indicated by inability to suck and swallow, diminished gag reflex and CAH requiring continuous mechanical ventilation. Polygraphic recordings during sleep demonstrated severe central hypoventilation ($P_{aCO_2} > 76$ mmHg), writhing truncal movements, and marked though disorganized respiratory muscle activation with a tonic or right-left pattern rather than the normal inspiratory-expiratory phase switching. Marked respiratory muscle electromyographic activity indicated quantitatively adequate central neural output. Fluoroscopy confirmed dyssynchronous diaphragmatic movements; only rarely did the right and left hemidiaphragms contract simultaneously. Phrenic nerve stimulation ruled out diaphragmatic paralysis. We conclude that injury to the brainstem respiratory pattern generator can result in CAH when normal inspiratory-expiratory switching mechanisms are damaged.

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THEOPHYLLINE REDUCES HYPOXIC VENTILATORY DEPRESSION WITHOUT INCREASING CATECHOLAMINES. Roger D. Bruce, Robert A. Darnall, John S. Althaus (Spon. by John Kaffwinkell, Dept. of Pediatrics and Anesthesiology, University of Virginia School of Medicine, Charlottesville).

Theophylline (T) reduces hypoxic ventilatory depression in newborn piglets and can enhance the release of catecholamines (CATs) which in turn may stimulate ventilation. To determine if the effect of T on ventilation was due to the release of CATs, we measured plasma CATs and ventilation in two groups of spontaneously breathing newborn piglets <4 days old treated with either T (n=7) or normal saline (NS) (n=6) both during normoxia and hypoxia. The piglets were anesthetized with ketamine and xylazine, intubated and the femoral artery catheterized. Epinephrine (EPI) and norepinephrine (NE) were measured before and 30 minutes after treatment with T (15 mg/kg) or NS. The animals were exposed to 10% oxygen and the CATs remeasured after 5 minutes of hypoxia. Respiratory rate (RR), expiratory flow integrated to minute ventilation (Ve), heart rate and blood pressure were continuously recorded. CATs were assayed by high pressure liquid chromatography with electrochemical detection.

Treatment with T during normoxia was associated with an increase in tidal volume. During hypoxia treatment with T prevented the fall in Ve and RR seen in the NS group. EPI and NE increased during hypoxia but there was no difference between the groups at 5 minutes. In our model the increase in CATs observed during hypoxia was not enhanced by T. This is consistent with the hypothesis that some mechanism other than catecholamine release is responsible for the effect of T in reducing neonatal hypoxic respiratory depression.

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DETECTION OF GASTRIC CONTENTS IN TRACHEAL FLUID USING AN AUTOMATED LACTOSE ANALYZER. David J. Burchfield, Josef Neu, (Spon. by Donald V. Ettzman) U. of Fla. College of Medicine, Dept. of Peds., Gainesville

Aspiration of feedings in chronically ventilated patients may contribute to chronic lung disease and prolong their ventilator course. Lactose detected in the tracheal aspirate of an infant being fed a lactose containing diet would infer aspiration of gastric contents. Using a Yellow Springs Instrument Model 27 (YSI27) lactose analyzer serial dilutions of formula with lactose concentrations ([LAC]) of 900-1.0 mg/dl, known solutions with [LAC] of 800-1.5 mg/dl and 9 tracheal aspirates from 2 neonates being fed a lactose-containing formula were analyzed for lactose. 12 tracheal aspirates from 4 neonates never fed served as controls. All tracheal aspirates were further analyzed using a previously described method which employs enzymatic hydrolysis by B-galactosidase (sensitivity 0.9 mg/dl).

Results of in vitro samples (mg lactose/dl)							
[LAC] formula (known)	900	225	64	16	4	1	
*[LAC] formula (YSI)	784	191	47	11	1	0	
[LAC] Standard dilution	800	200	50	12.5	3	1.5	
+ [LAC] Stand. dil. (YSI)	775	191	47	10	1	0	

* $y = -3.41 + 0.870x$, $r = 0.99$, $p < .01$

+ $y = -1.65 + 0.973x$, $r = 0.99$, $p < .01$

Results of in vivo samples (mg lactose/dl)							
[LAC] YSI	6	0	2	12	4	4	1
[LAC] Enzymatic	0	0	0	0	0	0	0
Control YSI	6	0	3	3	7	8	4
Control Enzyme	0	0	0	0	0	0	0

Sample analyzed by HPLC and found to have no galactosides

The YSI 27 appears to be a sensitive instrument in vitro. Due to background interference in tracheal secretions, further work will be needed to use it clinically to detect aspiration.