†1819 ANTIDIURETIC HORMONE (ADH) RESPONSE IN SEVERE STATUS ASTHMATICUS IN CHILDREN. Madu Rao, Millicent Mitchell, Stanley Cohen, Richard Kravath, and Phillip Steiner, (Spon. by L. Finberg). State University of New York, Downstate Medical Center, Department of Pediatrics, Brooklyn, New York.

Twenty two children (age range:3-14 years, Sex:m:f::12:10) admitted to Kings County Hospital of Brooklyn with status asthmaticus were studied to determine their plasma ADH levels in relation to the severity of the status asthmaticus. Plasma ADH levels were measured by RIA at the time of admission and again at 1-2 weeks following recovery from status. The results are shown in the table.

	GROUP I n=14	GROUP II n=8
1. clinical sev.	severe	mild
2. PaO2(mmHg)	66-71	74-89
3. PaCO2(mmHg)	41-68	31-38
4. serum osm/kg	252-272	266-279
5. Urine osm/kg	676-278	610-700
6. Mean ADH (mU/ml)	12.6	6.3*
7. hyponatremia	2/14	none

*p< 0.001

We conclude from the study that 1. ADH response increases with severity of status asthmaticus 2. With high sp. gr. of urine during status, in addition to dehydration increased ADH secretion should be kept in mind and 3. In addition to other known factors, increased ADH may play a role in pulmonary edema with status asthmaticus.

1820 THE VENTILATORY RESPONSE TO HYPOXIA IN THE CHRONIC UNANESTHETIZED NEWBORN KITTEN. Henrique Rigatto, Carl Weibe, Claudio Rigatto, David Lee and Don Cates. Dept. of Pediatrics, University of Manitoba, Winnipeg, Canada.

We studied the ventilatory response to hypoxia in 7 newborn kittens (n=12), 2 to 28 days old. Kittens were surgically instrumented to record diaphragmatic EMG (EMG_{DI}), eye movements (EOG), electrocortical activity (ECoG), and neck EMG. Ventilation ($\dot{V}_E$) was measured with a mask and a flow-through system. Kittens inhaled 21% O_2 for a control period of at least 2 min, 10% O_2 for 10 min, and 21% O_2 for 2 min again. Measurements were made in quiet sleep. With a decrease in FiO_2 to 10%, there was an immediate increase in $\dot{V}_E$ (0.210±0.017 to 0.300±0.025 L/min/kg; p<0.005) which was not sustained, ventilation decreasing to 0.224 ±0.028 by 10 min of hypoxia. V_T increased from 3.8±0.2 ml/kg to 5.0±0.4 (p<0.005) and then decreased to 3.9±0.2. Frequency increased from 56±4.0 breaths/min to 63±3 (p<0.02) and then decreased to 55±5. The changes in $\int EMG_{DI}xf$ followed those in $\dot{V}_E$. Kittens studied before 7 days of life had a more pronounced decrease in ventilatory output at 10 min of hypoxia than older kittens. Breathing pattern became irregular or periodic during hypoxia. On return to 21% O_2 ventilatory output decreased abruptly due to apnea. These results suggest; 1) the hyperventilatory response to hypoxia in unanesthetized kittens is not well sustained with values at end of hypoxia close to control levels; 2) the biphasic response to hypoxia is primarily a function of frequency; 3) younger kittens are less able to sustain hyperventilation than older kittens. We speculate that the late fall in ventilation is due to a central mechanism affecting frequency.

●1821 ELEVATED CATECHOLAMINES AND ABNORMAL HYPOXIC AROUSAL IN APNEA OF INFANCY. Antonio M. Rodriguez, David Warburton, and Thomas G. Keens, USC School of Medicine, Childrens Hospital of Los Angeles, Division of Neonatology and Pediatric Pulmonology, Los Angeles.

Arousal from quiet sleep (QS) in response to a hypoxic challenge (HC) does not occur in many apnea of infancy (AOI) patients [Ped. Res., 16:363A, 1982]. Since catecholamines (C) stimulate respiration, abnormal C metabolism might be involved in the depressed arousal response to HC in AOI. We studied 14 AOI infants [age 12.4±3.3 (SE) months; with a history of apnea, cyanosis and limpness, requiring at least vigorous shaking for resuscitation; no identified treatable cause of apnea; and still having apnea]. When in QS by EOG and behavioral criteria, HC (PIO2 80 mmHg) was given for 3 minutes or until arousal (eye opening, agitation, movement, crying) occurred. Through an indwelling venous line, blood samples were obtained for epinephrine (E), norepinephrine (NE), and dopamine (D) levels [by REA] while awake, after 10-min of QS, and 1 min after the start and completion of each HC. Of the 14 AOI patients, 11 (79%) did not arouse to 2 consecutive HC 10 min apart. In infants who did not arouse, E was 2.5x greater during QS (P< 0.025), NE was 2.7x greater while awake (P< 0.02), 6.4x greater in QS (P< 0.001), and 12x greater post-HC (P< 0.001) than in those who did arouse. There were no differences in D. We conclude that E and NE are increased in AOI patients with abnormal hypoxic arousal responses: decreased C responsiveness might contribute to the abnormal arousal response to HC.

●1822 ALPHA 1 ANTITRYPSIN IN THE PREDICTION OF BRONCHOPULMONARY DYSPLASIA (BPD). Warren Rosenfeld, Luz Concepcion, Ignacio Zabaleta, Ramesh Jhaveri, & Hugh Evans. Department of Pediatrics, Interfaith Medical Center, SUNY/Downstate Medical Center, Brooklyn, New York.

BPD may be due to lung connective tissue damage caused by oxidants and proteolytic enzymes in prematures with RDS who are treated with ventilators and oxygen. Antiproteases, such as AAT, may protect the lungs against this destruction. To study its potential role we measured plasma trypsin inhibitory capacity (TIC) (Erlanger method) in 27 prematures who required ventilator therapy for treatment of RDS. Day 1 TIC values were significantly lower (p<0.0001) in the 13 patients who developed BPD when compared to the 14 who did not. Other significant variables included birthweight (p<0.005), severity of RDS (p<0.03) and gestational age (p<0.05). One way analysis of variance demonstrated day 1 TIC to be the most significant variable(p<0.00001) followed by birth weight (p<0.0175), severity of RDS (p<0.058) and gestational age (p<0.0739). A composite of these 4 variables (TIC, birth weight, severity RDS and gestational age), were 100% sensitive and 86% specific in the prediction of BPD. Decreased TIC on day 1 may be of pathogenetic and prognostic significance in the development of BPD.

	Wt (Gms)	Ga (Wks)	TIC DAY 1	3	7	Wk2	3	4	5	6	7	8	9	10
NO BPD #14	1221	29.4	.97	.72	.67	.78	.70	.65	.63	.60	.76	.81	.61	.71
BPD #13	971	28.0	.34	.55	.82	.72	.62	.61	.57	.63	.76	.76	.60	.74

1823 CERULOPLASMIN (CER) LEVELS IN PREMATURES AT RISK FOR BRONCHOPULMONARY DYSPLASIA (BPD). Warren Rosenfeld, Ignacio Zabaleta, Luz Concepcion, Ramesh Jhaveri, & Hugh Evans. Department of Pediatrics, Interfaith Medical Center, SUNY/Downstate Medical Center, Brooklyn, New York.

Oxidant injury during ventilation therapy in prematures with RDS has been postulated as a possible cause of BPD. Antioxidants, such as ceruloplasmin (CER), may provide protection against the occurrence of BPD. CER serum levels have been correlated with the degree of severity of RDS and are capable of scavenging superoxide radicals in vitro. We have evaluated the potential role of serum ceruloplasmin levels in predicting the subsequent development of BPD in 30 prematures with RDS who required ventilator therapy.

CER was not significantly lower in those patients who developed BPD when compared to those who did not.

	BWT	DAY1	3	7	Wk2	3	4	5	6	7	8	9	10
NO BPD#15	1251	11.4	110	115	124	119	112	135	120	126	148	146	135
BPD#15	986*	9.1	9.4	9.3	8.5	134	121	121	153	141	196	125	198

*p < 0.005

Although patients who developed BPD had lower birth weights (p<0.005), lower gestational age (p<0.03) and had more severe RDS (p<0.03), there were no significant differences in CER levels. In both groups CER levels ranged from 33-50% of pooled adult plasma. One way analysis of variance did not demonstrate that the CER levels were a significant predictor for the development of BPD.

1824 THE EMPTY THORAX: AN AVOIDABLE LETHAL FACTOR IN CONGENITAL DIAPHRAGMATIC HERNIA. Rita L. Saldanha, Arthur E. Kopelman and Walter J. Pories (Spon. by Jean F. Kenny). ECU School of Medicine, Pitt County Memorial Hospital, Department of Pediatrics, Greenville, NC.

In spite of sophistication in neonatal care, congenital diaphragmatic hernia (CDH) which presents within the first 24 hours of life has a high mortality (generally around 50%).

The observation that neonates with CDH deteriorate several hours after surgery, frequently with a mediastinal shift, suggest that they may be dying of the empty thorax syndrome. When a patient is on positive pressure ventilation, insertion of a chest tube connected to a suction or a waterseal into the thoracic cavity, when there is no expandable lung, creates a vacuum and the mediastinum is shifted to that side.

In addition to using currently accepted management, we used a modification of an infant chest bottle. A chest tube was connected to a bottle with a vent, but without a waterseal. This permitted evacuation of fluid but prevented mediastinal shifts.

We treated a consecutive series of 11 infants with this approach, ten of which were symptomatic soon after birth. Three of these were moribund at the time of surgery and died. Seven of the remaining eight salvageable infants required surgery within 24 hours of life.

Seven out of eight (87.5%) of the infants survived if they were considered potentially salvageable prior to surgery. The use of thoracic drainage with avoidance of the empty thorax syndrome appeared to play a significant role in obtaining these improved results.