1813 Factors Associated with Fatal Outcome in Premature Infants with Prolonged Bronchopulmonary Dysplasia (BPD). J. Perlman, V. Moore, M. Siegel, J. Dawson, (Spon. by J.J. Volpe), Wash. Univ. Sch. Med., St. Louis Child. Hosp., Depts. Ped., Radiol., St. Louis, MO

BPD remains a significant problem in the premature infant. Mortality associated with Stage IV BPD is 39% (J. Pediat. 816, 1979). The objective of this retrospective study was to determine factors associated with fatal outcome in infants with Stage IV BPD (radiographic) and a prolonged hospital course >100 days.

The study comprised 24 premature infants; 11 died and 13 survived (controls). No differences between the groups were noted as regards birthweight, gestational age, ventilator requirement, radiographic changes and medications (Digoxin, aldactazide), except for Lasix which was used twice as frequently in the group who died vs controls (P<.001). Four striking differences were noted between the groups (Table).

	Died n=11	Control n=13	Mean Duration (days) Died	Control	
Hypertension (syst.>113 mmHg)	11	1*	65.8		
Hypochlorydia (Cl.<80 mEq/l)	11	6*	25	6	
Met. Alkalosis (>pH 7.45)	9	3*	25	3.4	
↓ Head Growth	10	1*	-		*P<.001

The data define significant alterations in blood pressure, serum chloride, acid base status, and head growth. The findings are important for two reasons. First, they define factors that may be important in the genesis of a poor outcome in such infants, and second, they suggest that close monitoring of these parameters and correction when possible, may improve prognosis.

†1814 MEDIATORS AFFECTING NEUTROPHIL (PMN) FUNCTION ARE RELEASED AFTER PULMONARY INTRAVASCULAR COAGULATION. Marc Perlman, Arnold Johnson, Asrar Malik (Spon. Bernard Pollara). Albany Medical College, Albany Medical Center, Departments of Pediatrics and Physiology, Albany, N.Y.

Awake sheep (n=4) with lung lymph fistulas were infused with thrombin (T)(80U/kg). Lung lymph was collected before and at 5 to 60 min. after T. PMN were isolated from donor sheep. Lymph chemotaxis was measured by chemotaxis under agarose. Superoxide anion (O_2-) release by PMN incubated with lymph was measured by difference in ferricytochrome C reduction with and without superoxide dismutase. PMN aggregation was measured on an aggregometer. Data are shown as mean ± SEM

		LYMPH SAMPLE (MIN)				
	Baseline	5	15	20	30	60
(O_2-)nmoles	0.07	0.05	1.42	0.58	0.57	0.12
10^6cell/min	±0.02	±0.02	±0.03	±0.19	±0.16	±0.09
Chemotaxis	0.6	0.6	2.7	1.4	1.2	0.4
(mm)	±0.4	±0.3	±0.6	±0.2	±0.2	±0.4
Aggregation	2.2	47.8	43.7	34.2	41.6	-----
(%)	±1.3	±14.2	± 4.2	± 3.3	± 3.6	-----

Addition of hirudin (a thrombin inactivator) to lymph did not reduce chemotaxis indicating that responses were not due to T. Conclusion. Thrombin-induced intravascular coagulation causes time-dependent intrapulmonary generation of mediators of PMN chemotaxis, aggregation and O_2- production. These may be important in PMN-dependent acute lung injury in the patient with disseminated intravascular coagulation. (Supported by HL-17355, HL-26551,HL-31359 and HL-07529).

†1815 PULMONARY VASCULAR PERMEABILITY TO PROTEINS INCREASES AFTER THROMBIN-INDUCED INTRAVASCULAR COAGULATION. Marc B. Perlman, Siu K. Lo, Asrar B. Malik (Spon. Bernard Pollara), Dept. of Pediatrics and Physiology, Albany Medical College, Albany, N.Y. 12208.

Since intravascular coagulation is a proposed etiologic factor in acute lung injury, we examined whether thrombin-induced intravascular coagulation results in increased lung vascular permeability to proteins. Sheep (n=5) were prepared with lung lymph fistulas and balloon-tipped left atrial catheters. The sheep were studied in the unanesthetized state. The left atrial balloon was inflated to produce step increases in left atrial pressure (P_{la}) to a maximum of 25 Torr. Pulmonary lymph flow ($\dot{Q}$lym) and lymph-to-plasma protein concentration ratio (L/P) were measured. The protein reflection coefficient (σ_d), a measure of pulmonary vascular permeability, was determined by increasing P_{la} until $\dot{Q}$lym approached the filtration-independent state; i.e. further increases in $\dot{Q}$lym did not further decrease L/P and σ_d approached (1-L/P). α -thrombin (80 U/kg) was then infused and a new steady-state $\dot{Q}$lym was reached to allow σ_d to be determined after thrombin. (σ_d=1 indicates complete impermeability to proteins and σ_d=0 indicates complete permeability). In the present study, σ_d before thrombin was 0.70±0.03 (mean±SE) and decreased to 0.59±0.01 after thrombin ($p<0.05$). We conclude that pulmonary intravascular coagulation increases pulmonary vascular permeability to proteins and that this may be a factor in mediating acute lung injury. (Supported by HL17355, HL26551 and GM07033).

●1816 ANTIDIURETIC HORMONE (ADH) SECRETION DURING RESPIRATORY DISTRESS IN CHILDREN WITH BRONCHOPULMONARY DYSPLASIA (BPD). M. Rao, N. Eid, M. Mitchell, L. Herod, A. Parekh and P. Steiner, (Spon. by L. Finberg) State University Hospital, Downstate Medical Center, Department of Pediatrics, Brooklyn, New York.

Twelve children (age:3 mos to 7 mos, sex:m:f::8:4) known to have bronchopulmonary dysplasia were studied for antidiuretic hormone response during hospitalization with respiratory distress, hypoxemia and hyperinflation on chest x-ray. 7/12 had wheezing at the time of admission. None had received any medications before admission. None of the children had clinical evidence of dehydration (normal plasma renin activity of 2.4-4.7 ng/ml). Plasma ADH levels were measured by radioimmune assay (N=up to 1.7 mU/ml) and were elevated 6-9 fold (2.4 mU/ml-3.1 mU/ml) within 7-10 days. 3/12 children developed hyponatremia (129-131) 24 hours after admission. Further, 2/3 children with hyponatremia developed diastolic hypertension for up to 2 days. 7/12 were given furosemide at the time of admission for the presence of wheezing. Though the wheezing diminished considerably following furosemide, there was no reduction in the plasma ADH levels. We conclude that 1. ADH secretion is common in children with BPD during acute respiratory distress 2. The mechanism for the production of ADH is either hypoxemia and/or hyperinflation 3. Hyponatremia occurs with increased ADH secretion and therefore care should be exercised in the administration of fluids 4. Though furosemide may have a beneficial effect on the respiratory distress in children with BPD, the plasma ADH levels are not influenced by its administration and 5. The continued ADH secretion in children with BPD feeds a vicious cylcle.

1817 HYPERINFLATION (AIRTRAPPING) AS A STIMULANT OF ADH RESPONSE IN CHILDREN WITH CHRONIC ASTHMA. Madu Rao, Nemir Eid, Millicent Mitchell, and Phillip Steiner, (Spon. by L. Finberg) State University of New York, Downstate Medical Center, Department of Pediatrics, Brooklyn, New York.

Elevation of plasma antidiuretic hormone (ADH) levels during status asthmaticus is well documented. The physiologic mechanisms responsible for the hormone release include hypovolemia, diminished left atrial filling due to hypoxemia and vasoconstriction from bronchospasm, stress and the role of adrenergic agents. We postulated that hyperinflation (airtrapping) in a chronic asthmatic child could reduce left atrial filling by mechanical forces and so increase the ADH response. The study included 12 chronic asthmatic children (age range:10-15 years, Sex:m:f::8:4). All bronchodilators were discontinued at least 24 hours before the study which consisted of measurement of lung functions including airway resistance and total lung capacities by body plethysmography. In addition, blood was collected for plasma ADH levels, serum and urine osmolalities. 9/12 children had 6-8 fold increase in plasma ADH levels (N-up to 1.7 mU/ml) with normal airway resistance and >120% of predicted normals for total lung capacities. 4/12 had normal lung functions with normal levels of plasma ADH levels. 4/9 children with elevated ADH levels were given furosemide to induce diuresis and this had no effect on either the plasma ADH levels nor the lung functions. In conclusion, 1. hyperinflation in itself produces ADH response 2. whether the hormone causes a negative effect on the lungs by shift in the water balance remains to be studied and 3. diuresis appears to have no effect on either the hormonal response or improving the lung functions.

1818 PULMONARY MANIFESTATIONS IN CHILDREN WITH "AIDS" Madu Rao, Senih Fikrig, R. Menez, Millicent Mitchell, Nemir Eid, and Phillip Steiner, (Spon. by L. Finberg) State University of New York, Downstate Medical Center, Department of Pediatrics, Brooklyn, New York.

Acquired immunodeficiency syndrome (MMWR 32:688, 1984) was diagnosed in 7 children (Age:5 months-2.5 years, Sex:m:f::5:2, Ethnicity:5 Haitians, 1 Hispanic and 1 black American) at Downstate Medical Center from 1981 to 1983. Respiratory problems (cough and pneumonia) developed within 1-3 months of the diagnosis of AIDS in all children. Radiographic features consisted of bilateral diffuse interstitial infiltrates and nodular densities mimicking miliary Tbc. (A-a) O_2 gradients were very wide (53.1mm Hg), particularly in children with pneumocystis carinii infection (biopsy proven in 3/7). Histology of the lung consisted of lymphocytic infiltration with scattered bronchiectasis, fibrosis and thickening of vessels. 3/7 had pneumocystic carinii infection with one child having in addition cryptococcal and staphylococcal infections respectively. 3/7 expired within 1-3 months of the onset of respiratory manifestations. We conclude that 1. Respiratory manifestations are common in AIDS 2. Clinical and radiographic features are nonspecific 3. Wide (A-a) O_2 gradients should arouse suspicion of pneumocystis carinii infection and should suggest doing a lung biopsy 4. An early recognition of the problem will lead to a more effective therapy for the infectious complications of AIDS.