†1525 GASTROESOPHAGEAL REFLUX (GER) TO THE PROXIMAL ESOPHAGUS IN NEONATES WITH BRONCHOPULMONARY DYSPLASIA. Bruce D. Sindel, M. Jeffrey Maisels, Thomas V. N. Ballantine. Penn St Univ Coll of Med, M.S. Hershey Med Ctr, Dept of Pediatrics and Surgery, Hershey, PA.

Recurrent aspiration following GER may contribute to the severity of chronic lung disease. If so, it should be possible to document acid reflux to the proximal esophagus. Using an esophageal pH probe placed at the level of the 1st or 2nd thoracic vertebra, we evaluated GER in 11 infants with BPD and 9 infants with no BPD. Results (mean±SD):

	BPD	Controls	P Value
% Time pH <4	3.83± 7.92	16.16±17.33	0.04
Episodes GER/h	0.49± 0.76	1.33± 0.88	0.03
Episodes GER <5 min/hr	0.12± 0.26	0.37± 0.33	0.07
Longest episode (mins)	7.40±11.57	34.85±44.10	0.06
Post-conceptional age at study (wks)	41.07± 9.41	39.47± 4.69	NS
Gestational age (wks)	28.45± 4.50	34.11± 5.95	0.03
Birthweight (g)	1173±688	2146±1033	0.02
Mechanical ventilation	6	0	
Nasogastric feeding	10	0	

Control infants have frequent episodes of GER to the upper esophagus but either do not aspirate or are not embarrassed by small aspirations. Infants with BPD have significantly less GER to the proximal esophagus than non BPD babies. The reason for this is not apparent. Either GER in BPD infants is unimportant or aspiration may follow occasional episodes of GER causing severe bronchospasm as well as aggravating existing lung disease.

1526 THE EFFECT OF A NISSEN FUNDOPLICATION ON INFANTS WITH CHRONIC LUNG DISEASE (CLD). Bruce D. Sindel M. Jeffrey Maisels, Thomas V. N. Ballantine, Stephen R. Karl. Penn St Univ Coll of Med, M.S. Hershey Med Ctr, Dept Pediatrics (Newborn Medicine) and Surgery, Hershey, PA.

Chronic intermittent aspiration may be a cause of ongoing pulmonary insult in infants with the severe form of CLD. Over a 2 yr. period, 10 infants with CLD received a Nissen fundoplication procedure and a gastrostomy because of failure to improve or recurrent episodes of deterioration. The following results are for 2 wks. pre/postop unless otherwise stated (mean± SD):

	Preop	Postop	P Value
# on mechanical ventilation (n=10)	5	1	0.070
# on oxygen therapy (n=10)	10	6	0.043
Days without enteral feeds (n=10)	4.60± 4.72	0.80± 1.40	0.025
Weight gain g (in hospital, n=8)	304±181	141±131	0.058
PCO_2 (1 wk., n=6)	58.93±20.66	48.50± 9.13	0.003
Respiratory rate (no mechanical ventilation - 1 wk., n=3)	64.81±12.67	61.29±12.29	0.002

Decreased weight gain following surgery may reflect the caloric cost of postoperative healing. The majority of these patients had negative diagnostic studies for GER (Barium swallow and pH probe). These data suggest that GER contributes to CLD in some infants but is not readily diagnosed by current techniques. A randomized controlled trial of anti-reflux surgery in infants with severe CLD can be justified.

†1527 PROSTAGLANDIN D2 DOES NOT LOWER PULMONARY ARTERIAL PRESSURE OR IMPROVE OXYGENATION IN INFANTS WITH PERSISTENT PULMONARY HYPERTENSION SYNDROME (PPHN). Scott J. Solfer, Ronald I. Clyman and Michael A. Heymann. Univ of Calif and Mt Zion Hosp, Dept of Ped and CVRI, San Francisco.

Prostaglandin D2 (PGD2) decreases pulmonary arterial pressure (PAP) and increases pulmonary blood flow (PBF) without changing systemic arterial pressure (SAP) in fetal and newborn lambs. PGD2 increases PAP and decreases PBF in older animals. This suggested a role for PGD2 in the decrease in PAP at birth and in the treatment of infants with PPHN. The effects of PGD2 were studied in 6 infants (age 1-2d; BW 3277±345g) with a descending aortic PaO2<100mmHg during mechanical hyperventilation with an FiO2 of 1.0. Tolazoline and dopamine were used in four of the patients. No patient had congenital heart disease or sepsis. Informed consent was obtained. Catheters were placed to measure PAP and SAP. PGD2 was infused intravenously at doses of 1-25 mcg/kg/min. PAP, SAP, heart rate and arterial blood gases were measured prior to each dose change.

	N	PAP	SAP	HR	PaO2	pH
Pre	6	63±9	58±10	168±26	59±23	7.52±.09
PGD2 1-4	4	58±10	62±11	181±17	44±6	7.50±.03
5-9	5	53±7	61±4	188±10	55±19	7.48±.08
10-25	4	60±13	62±8	183±8	40±42	7.47±.08

Mean±SD; PGD2:mcg/kg/min; PAP,SAP,PaO2:mmHg

Two patients had a transient increase in PaO2. All had HR increases. Two had an increase in PAP. No deleterious effects occurred during the infusion. Four patients subsequently died. Though PGD2 is a specific pulmonary vasodilator in fetal and newborn animals, it does not lower PAP in infants with PPHN.

†1528 TRANSCUTANEOUS PULSE OXYGEN SATURATION ($tcSaO_2$) MONITORS ARE SUPERIOR TO $tcPO_2$ MONITORS IN INFANTS WITH BRONCHOPULMONARY DYSPLASIA (BPD). Alfonso J. Solimano, John A. Smyth, Tejinder K. Mann, Susan G. Albersheim, Gillian Lockitch. (Spon: J.G. Hall). U.B.C., Children's Hospital, Div. Neonatology, Dept. Pediatrics, Vancouver, Canada.

We studied 12 infants with a clinical and radiologic diagnosis of BPD who were oxygen dependant and older than 30 days. $tcSaO_2$ measured by two monitors (Nellcor 100, BTI Biox III) and $tcPO_2$ (Transend) were correlated with arterial SaO_2 (Radiometer OSM2 Hemoximeter) and PaO_2 (Corning 178) measured on blood from an indwelling catheter. For each infant, the FiO_2 was adjusted to obtain 3-5 sets of data in the range of 70-95% SaO_2.

Subjects (average, range): B. Wt. 885, 540-1880 g.; G.A. 25.8, 23-34 wks.; Postnatal age 75, 30-241 days; Study wt. 1983, 850-5420 g.

MONITOR	VARIABLE	n	r^2	SLOPE	AVERAGE ERROR
Nellcor 100	SaO_2	53	0.91	0.86	+2.5%
BTI Biox III	SaO_2	53	0.79	0.91	+1.0%
Transend	PO_2	53	0.65	0.55	-29.0%

Our data confirms reports by others that $tcPO_2$ does not accurately represent PaO_2 in older infants with BPD. Based on accuracy, self-calibration and an unheated sensor that will not cause skin burns, monitoring $tcSaO_2$ has major advantages over $tcPO_2$ in the management of BPD.

1529 FACTORS INFLUENCING TRANSCUTANEOUS CARBON DIOXIDE MEASUREMENT (t_cCO_2) IN NEONATES. Roger F. Soll, Jeffrey D. Horbar and Jerold F. Lucey, University of Vermont College of Medicine, Dept. of Pediatrics, Burlington.

The relationship between t_cCO_2 and P_aCO_2 was studied in 20 neonates and the factors effecting this relationship determined.

Infants ranging from 28 to 36 weeks gestation were studied during the first week after birth using an irridium/irridium oxide electrode (Hellige Kapnomonitor) which was maintained at a temperature of 42°C. Four to eight readings were obtained on each neonate and compared to simultaneous umbilical arterial samples. There is a linear relationship between P_aCO_2 and t_cCO_2 for the entire population (t_cCO_2=1.30 P_aCO_2+16.65, r=0.80). Variation among individuals accounts for 42% of the variance about the regression line.

Stepwise multiple regression revealed that increases in base deficit and mean arterial blood pressure were associated with increases in t_cCO_2. Arterial pH did not significantly effect the t_cCO_2 measurement.

We conclude that, 1) for a population of premature infants, there is a linear relationship between t_cCO_2 and P_aCO_2 using the Hellige Kapnomonitor, 2) there is considerable individual variation among infants making precise prediction of P_aCO_2 from t_cCO_2 difficult, and 3) base deficit and mean arterial blood pressure have a significant effect on t_cCO_2 measurements.

†1530 A MODEL OF CHANGING BODY COMPOSITION OF THE PREMATURE INFANT. Donald W. Spady, W. Szymanski, David Schiff, University of Alberta, Faculty of Medicine, Dept. of Pediatrics, Edmonton, Alberta, Canada.

Serial measurements of total body water (TBW), extracellular fluid (ECF), nitrogen (N) balance, and total body potassium(TBK) were made in healthy, growing premature infants weighing between 1100 and 2000 gm. In 30 infants a total of 49 estimates of TBW and ECF were made using antipyrine and bromide; in 23 of these, weekly N balances were made. Mothers milk was the diet in 13 cases formula in 7 and a mixture of the two in 10. TBK was measured 84 times by whole body counting in 40 other premature infants with characteristics similar to the above and aged between 2 and 90 days and weighing between 1100 and 3600 g. Power equations relating weight to TBW,ECF,ICF and TBK have been derived and estimates made of changes in these parameters over a weight gain of 900 g. N and fat retention was also estimated. Data presented are not subdivided by type of diet, although we feel this may markedly affect body composition (BC). Of the 900 g gained, 309cc was ICF, 333 cc ECF, 116 g protein, and 123 g fat. Extracellular solids accounted for 26 g and TBK increased by 37.8 mEq. Body cell mass (BCM) increased by 425 g. TBK expressed relative to fat free mass (FFM) was 49.7 mEq/kg; the K:N ratio was 0.49 and the TBK: BCM ratio 0.0113. TBK was 135 mEq/l ICF. Using the above incremental changes and data derived from the reference fetus, we can estimate the BC of the growing premature. The new BC is shown below

WT	ICF	ECF	TBK	Fat	Prot	TBK/KGFFM	K:N	TBK:BCM	Ash
1100g	374cc	457cc	51mEq	112g	125g	51.7 mEq	0.39	0.0098	33g
2000g	675cc	789cc	89mEq	236g	241g	50.4 mEq	0.43	0.0103	59g