GASTROESOPHAGEAL REFLUX (GER) TO THE PROXIMAL ESOPHA-

†1525 GASTROESOPHAGEAL REFLUX (GER) TO THE PROXIMAL ESOPHAGUS IN NEONATES WITH BRONCHOPULMONARY DYSPLASIA. Bruce D. Sindel, M. Jeffrey Maisels, Thomas V. N. Ballantine. Tenn 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 thora-cic vertebra, we evaluated GER in 11 infants with BPD and 9 infants with no BPD. Results (meantSD): BPD Controls P. Value

	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	41.07± 9.41	39.47± 4.69	NS
at study (wks)			
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	
		C OTD	

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 bables. 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.

THE EFFECT OF A NISSEN FUNDOPLICATION ON INFANTS

THE EFFECT OF A NISSEN FUNDOPLICATION ON INFANTS WITH CHRONIC LUNG DISEASE (CLD). <u>Bruce D. Sindel</u> 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 fundo-plication procedure and a gastrostomy because of failure to im-prove or recurrent episodes of deterioration. The following results are for 2 wks. pre/postop unless otherwise stated (meant SD). P V SD):

	Preop	Postop	Value
# on mechanical ventilation (n=10)		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	64.81±12.67	61.29±12.29	0.002
ventilation - 1 wk., n=3)			

Decreased weight gain following surgery may reflect the caloric cost of postoperative healing. The majority of these patients had <u>negative</u> diagnostic studies for GER (Barium swallow and pH probe). These data suggest that GER contributes to CLD in some probe). probe). These data suggest that Gen contributes to the 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.

PROSTAGLANDIN D2 DOES NOT LOWER PULMONARY ARTERIAL PRESSURE OR IMPROVE OXYGENATION IN INFANTS WITH †1527 PERSISTENT PULMONARY HYPERTENSION SYNDROME (PPHN).

LJ21 PERSISTENT PULMONARY HYPERTENSION SYNDROME (PPHN). Scott J. Solfer. Ronaid L. 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 Pa02<10mmHg during mechanical hyperventila-tion with an FiO2 of 1.0. Tolazoline and dopamine were used in tion 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.

-		Ν	PAP	SAP	HR	Pa02	рН
Pre		6	63 <u>+</u> 9	58 <u>+</u> 10	168 <u>+</u> 26	59 <u>+</u> 23	7.52 <u>+</u> .09
PGD2	1-4	4	58 <u>+</u> 10	62 <u>+</u> 11	181 <u>+</u> 17	44 <u>+</u> 6	7.50 <u>+</u> .03
	5-9	5	53 <u>+</u> 7	61 <u>+</u> 4	188 <u>+</u> 10	55±19	7.48 <u>+</u> .08
	10-25	4	60 <u>+</u> 13	62 <u>+</u> 8	183 <u>+</u> 8	40 <u>+</u> 42	7.47 <u>+</u> .08
	Mean+SD:	PGD	2:mca/ka/	min: PA	SAP, PaO	2:mmHg	

Two patients had a transient increase in PaO2. All had HR increases. Two had an increase in PAO2. 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.

TRANSCUTANEOUS PULSE OXYGEN SATURATION (tcSaO₂) MONITORS ARE SUPERIOR TO tcPO₂ MONITORS IN INFANTS WITH BRONCHOPULMONARY †**1528** DYSPLASIA (BPD). Alfonso J. Solimano, John A. Smyth, Tejinder K. Mann, Susan G. Albersheim, Gillian Lock-itch. (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 then 20 days. toSaO measured by two monitors (Nelldiagnosis of BPD who were oxygen dependant and older than 30 days. tcSaO, measured by two monitors (Nell-cor 100, BTI Biox III) and tcPO, (Transend) were cor-related with arterial SaO, (Radiometer OSM2 Hemoxi-meter) and PaO, (Corning 178) measured on blood from an indwelling čatheter. For each infant, the FiO, was adjusted to obtain 3-5 sets of data in the range of 70-95% SaO. of 70-95% SaO₂. <u>Subjects</u> (average, range): B. Wt. 885, 540-1880 g.; <u>G.A. 25.8</u>, 23-34 wks.; Postnatal age 75, 30-241 days; Study wt. 1983, 850-5420 g. 2 <u>MONITOR</u> <u>VARIABLE</u> n r SLOPE <u>AVERAGE ERROR</u> <u>Nellcor</u> 100 SaO₂ 53 0.91 0.86 +2.5% BTI Biox III SaO₂ 53 0.79 0.91 +1.0% Transend PO₂ 53 0.65 0.55 -29.0% AVERAGE ERROR P02

Our data confirms reports by others that tcPO, does not accurately represent PaO, in older infants with BPD. Based on accuracy, self-calibration and an unheated sensor that will not cause skin burns, moni-toring tcSaO, has major advantages over tcPO₂ in the management of BPD.

FACTORS INFLUENCING TRANSCUTANEOUS CARBON DIOXIDE **1529** FACTORS INFLUENCING TRANSCUTATEOUS CARBON BIORIDE 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 wind the factor work ofter hit busing an irridium/irridium

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_cC0_2 =1.30 PgC0_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.

A MODEL OF CHANGING BODY COMPOSITION OF THE PREMATURE **†1530** 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 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 rela-ting 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/1 ICF. Using the above incre-mental changes and data derived from the reference fetus, we can estimate the BC of the growing premature. The new BC is shown below mental 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 2000q 675cc 789cc 89mEq 236g 241g 50.4 mEq 0.43 0.0103 59g