ABNORMAL LUNG COLLAGEN RATIOS IN VENTILATED HUMAN †1852 NEONATES. Craig Shoemaker, Jerold Last, Karen Reiser, Boyd Goetzman. Department of Pediatrics and California Primate Research Center, University of California, Davis

California, Davis We assessed lung collagen in human infants at risk for chronic lung disease (CLD) because Type I collagen is increased in adult RDS, and idiopathic or experimental pulmonary fibrosis. We determined collagen Types I and III in the lungs from 15 human infants who died while receiving mechanical ventilation, and in 3 stillborn infants. We analyzed lung tissue by CNBr digestion, column chromatography and index polypeptide separation by poly-acrylamide gel electrophoresis. To decrease the influence of measurement error of Type I collagen, we calculated ratios of Type I to III collagen. We then compared infants with clinical and/or pathological CLD to those without CLD using pooled variance techniques and one tailed t-tests. variance techniques and one tailed t-tests. MEAN BW(GMS±SD) MEAN RATIO I/III±SD VENT. DAYS(RANGE) NO CLD-9 2004±953

collagen [/III ratios existed, but bacterial preumonia may have been contributory to rapidly developing CLD. All infants with pathologic CLD had increased Type I collagen. Two infants dying early with very high I/III ratios had evidence of prenatal brain damage, suggesting that some increased collagen synthesis may be the result of prenatal lung injury.

FACTORS AFFECTING THE EFFICACY OF AMINOPHYLLINE FOR **O1853** APNEA OF PREMATURITY. <u>Maureen Sims</u>, Savitri <u>Rambhatla</u>, <u>Gloria Yau</u>, <u>Luis Cabal</u>, <u>Paul Y.K. Wu</u>. sch. of Med., LAC-USC Med. Ctr., Dept of Ped., Los Angeles. USC Aminophylline (A) is often used to treat apnea of prematurity To determine response times, optimal length of therapy, effectiveness of A in relation to gestational age (GA) and postnatal age (PA), we conducted a randomized, blinded controlled study in 45 premature infants with recurrent apnea. Infants were divided into 2 groups. Group I (n=23, BW=1395±359g, GA=31±2wks., PA=3.2± 2d) received A (mean serum level 8.9mcg/ml) and Group II (n=22, BW=1306±336g, GA=30±2wks., PA=2.3±1.8d). Results: In infants not requiring assisted ventilation (AV): Group I (18/22) had less apnea 24 hours after institution of therapy (p<.025) and apnea ended in 67% of the infants by the 7th day. In 3 infants, apnea was minimally reduced (<25%) during the first 7 d and persisted for 4 weeks despite continued therapy. Group II (14/23) did not show a significant decrease in apnea until 72 hours and only 35% were free of apnea by 7th day. Apnea persisted in 3 in-Only 35% were free of apnea by 7th day. Apnea persisted in 3 in-fants for 4 weeks. In infants requiring AV: similar numbers in both groups developed respiratory failure. Twelve of the 13 in-fants needing AV were \leq 31 GA and had \geq 4 apneic episodes during the first 24 hours PA. Conclusion: A does not prevent respira-tory failure and is not effective in infants \leq 31 wks. GA with early onset repetitive apnea. Twenty percent of premature infants are refractory to A and can be identified by evaluating their response within the first 7 days of therapy. Prolonged treatment with A is not an effective therapy for apnea of prematurity.

EFFECT OF THEOPHYLLINE ON VENTILATORY PARAMETERS 1854 DURING INSPIRATORY RESISTIVE LOADING. E.M. Sivieri 1004 M.R. Wolfson, V.K. Bhutani, W.W. Fox, T.H. Shaffer and <u>S. Abbasi</u>, Univ. Pa. Sch. Med., Pennsylvania Hosp, Dept. Pediatr, Temple University Sch. Med., Dept. Physiology, Philadelphia. Pa. Previous studies suggest that preterm infants lack the venti-betown encourses of the preterm infants lack the ventilatory reserve necessary to compensate for an additional inspira-tory load. Theophylline (TH) is often prescribed for apnea of

prematurity and bronchopulmonary dysplasia. To evaluate the effect of TH on ventilatory parameters, seven preterm infants ($\overline{x} \pm$ SEM: 29.6 ± .92 weeks gestation; 1.24 ± .14 kg birthweight) were challenged with an externally applied inspiratory resistive load (300 cm H_2O/L/sec). The infants were studied at a mean age of 46.3 \pm 9.3 days, weight of 1.75 \pm .19 kg, and TH level of 7.6 \pm .9 mg%. Tidal volume (V_T), frequency (f), minute ventilation (MV) inspiratory/total breath time (T_i/T_{tot}) and work of breathing (WOB) were assessed during spontaneous unloaded (UL) and loaded (L) breathing. Heart rate, transcutaneous 0_2 and $C0_2$ tensions were continuously monitored. Mean \pm SEM values are shown below:

	VT	MV	f	T_i/T_{tot}	WOB
	m1/kg	m1/M/kg	b/M		kg cm/kg
UL	8.8 ± 1.3	551 ± 82	64 ± 4	.46 ± .02	.028 ± .001
L	10.3 ± 1.5	566 ± 19	56 ± 5	.49 ± .03	.053 ± .018
These an ind resist apy in and en of obs	data indicate creased ventil tive challenge mproves the ver nhances their structed breat	that theop atory effor . This stuntilatory r ability to	whylline the t in respondy suggest reserve cap maintain v	nerapy is ass onse to an in ts that theop bacity of pre ventilation d	sociated with aspiratory whylline ther- eterm infants luring periods

MECHANISM OF GAS EXCHANGE ABNORMALITIES DURING GROUP 1855 B STREPTOCOCCAL SEPSIS IN PICLETS. <u>G.K. Sorensen</u>, <u>G.J. Redding</u>, and W.E. Truog, Dept. of Pediatrics, Univ. of Washington, Seattle, WA. Spon. by D.E. Woodróm.

Hypoxemia is common in newborns with Group B Streptococcal (GBS) sepsis, yet the mechanism of abnormal gas exchange in this disease is unknown. We studied ventilation-perfusion matching /Q) and shunt fraction using the multiple inert gas elimi-May of an sinit fraction down for a strong the metric as the sourcements were made in anesthetized animals ventilated with room air before, during, and after a 30 min intravenous infusion of 2x10⁹ colony forming units/kg of GBS. Shunt fraction was measured by sulfur hexafluoride retention and standard deviation of distribution of pulmonary blood flow (sdPBF) was used as an index of V_A/Q betergeneity. Changes are depicted below: *denotes $p_{A}^2,05$

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	Base-	During	Post Infusion			
	line	Infusion	20 min	l hr	2 hrs	
PaO _o (mmHg)	99±6	54±9*	75±24	85±14	87±12	
CO(m1/min/kg)	264±37	153±27*	218±33*	215±52	183±45*	
PvO ₂ (mmHg)	37±2	26±8*	23±7	33±4	30±4	
Ppa(mmHg)	15±3	39±4*	21±9	18±5	22±5	
Shunt(%)	1.9±1.4	6.4±4.9	7.2±12.3	2.1±1.6	1.7±1.0	
sdPBF(Units)	.44±.30	1.0±.19*	.61±.29	.69±.25	.78±.21	
PaO, CO and P	v0, fell	while Ppa	and sdPBF in	creased si	gnifi-	
cantly during	GBS infus	ion. Shur	nt increased	to >6% in	only 2 of	
the animals.	We conclu	de that an	terial hypox	emia durin	g GBS se	
sis in newborn	piglets	results pr	imarily from	diminishe	d cardiad	
output with re	sultant f	all in mix	ed venous ox	ygen tensi	on along	
with an increa	se in V./	Q heteroge	eneity within	the lungs		

NON-INVASIVE MONITORING OF BLOOD GAS PARAMETERS IN 1856 PEDIATRIC AND YOUNG ADULT PATTENTS WITH CHRONIC PULMONARY DISEASE. <u>Marilyn E. Alley and Alexander</u> <u>Spock</u>, Duke University Medical Center, Department of Pediatrics, Durham, N. C.

Evaluation of respiratory function in patients with chronic lung disease is usually performed by clinical evaluation, chest x-ray, pulmonary function tests and blood gas analyses. Noninvasive techniques have been utilized for inpatient but not out-patient monitoring of blood gas parameters in chronic lung disease. In this study we prospectively compared the results of non-invasive techniques, as measured by an IL capnograph for PECO2 and a BLOX oximeter for oxygen saturation (SoxO2), with arterialized blood gas values (SaO2, PaO2, PaCO2) in 45 consec-utive patients in the age range of 6 to 31 years. The following results were obtained:

	Pa02	Sa02	Sox02	PaC02	PECO2	
range	48-109	84-98	88-98	30-53	30-52	
mean	68.0	91.8	92.4	38.8	39.0	
	Sox02	2 v Sa02*	Pa02 v Sox	02 * PEC	02 v PaCO2*	
correlat	ion	*	*		*	
coeffici	ent 0.	764	0.716		0.943	
*signifi	cance < 0.0	001				

From these data, the SaO2 and PaO2 are both predictable from the SoxO2, as is the PaCO2 from the PECO2. This indicates that the capnograph and oximeter can be used effectively to noninvasively monitor blood gas parameters in patients with chronic pulmonary disease, particularly in pediatric patients. Any abnormalities or variation can be confirmed by blood gas levels.

HEMODYNAMIC EFFECTS OF HIGH FREQUENCY JET VENTILATION 1857 (HFJV) AND CONVENTIONAL VENTILATION (CV). W.A. Spohn, <u>S.E. Courtney</u>, D.S. Miles, R.W. Gotshall, W.J. Yike, <u>S.M. Ciarlariello</u>. Wright State University, The Children's Medical Center, Depts. of Pediatrics and Physiology, Dayton, Chio

(Spon. by M. Kogut).

The purpose of this study was to evaluate the effects on the cardiovascular system of HFJV compared to CV. Seven dogs (x=19.6 Kg) were monitored for 1 hour on CV, followed by 1 hour of HFJV, and were then returned to CV. Dogs were anaesthetized with pentobarbital and the skeletal musculature blocked with pavulon. Thermal dilution cardiac output, (CO), systemic and pulmonary arterial pressure were obtained at 20 min intervals. A Harvard volume respirator was set at $V_{\rm T}$ of 200 and rate of 24. A Healthdyne model 300 high frequency ventilator was set at a mean rate of 120 and a drive pressure of 30, to achieve a similar peak pressure (\bar{x} =13) and PEEP (\bar{x} =2.5) as on CV. Blood gases remained within the normal range with both forms of ventilation. (x values) CO SV MAP SVR PVR (x values)

	(1/min)	(m1)	(mm Hg)	(mm Hg/1/min)) (mm Hg/l/min)	
V1	3.6	19	132	42	4.1	
FJV	2.8	16	138	54	5.5	
V2	2.4	13	142	67	6.4	
There was a	signific	cant	(p <0.01),	progressive	fall in CO, due	1
-			2000 C 2000		date and the second second	

a fall in stroke volume (SV). Mean arterial pressure (MAP) was maintained as both systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) significantly increased (p <0.01). These results most likely reflect compensation to a temporal fall in CO and not differences between CV and HFJV.