ventilation, alveolar PO_2 and PCO_2, arterial pH, PCO_2 and PO_2, Co_2 respiratory response curves, and sensitivity to O_2. We measured ventilation with a nose piece and a screen flowmeter, using a constant flow-through to eliminate dead space and valves. Analyses were made from 3 to 5 min after the baby began breathing air, 2% or 4% Co_2 in air or 100% O_2. Sensitivity to O_2 was assesed by the change in ventilation when 100% O_2 was substituted for air.

Results:

	$\dot{V}_E$ L/min/kg	$PaCO_2$	$PACO_2$	PaO_2
P[1]	0.230±0.008	41±2	39±1	57±3
R[1]	0.273±0.011	38±2	35±2	66±4
	p<0.001	p<0.025	p<0.005	p<0.001

	PAO_2	pH
P[1]	102±2	7.34±0.01
R[1]	108±2	7.33±0.01
	p<0.001	p>0.5

	CO_2[2]		%$\Delta\dot{V}E$
	Slope	Position[3]	Air .. O_2
P[1]	0.030±0.007	42.7±2.2	−37±11
R[1]	0.035±0.007	36.0±2.7	−20±9
	p>0.2	p<0.001	p<0.025

All values: mean±0.2 S.E.
[1] P = periodic; R = regular.
[2] Response curve in L/min/kg/mm Hg.
[3] $PACO_2$ at 0.300 L/min/kg.

These findings show that periodic breathing in low birth weight infants is associated with hypoventilation, shift of the Co_2 response curve to the right and marked sensitivity to O_2. We suggest that low birth weight infants have increased O_2 chemoreceptor activity, but breathe periodically because of relative central depression.

170 *Ventilatory Disturbance and Arterial-Alveolar N_2 Differences During Recovery from Hyaline Membrane Disease.* A. PATHAK, L. MORRISON, L. M. PRUDENT, R. B. CHERRY and N. M. NELSON, Harvard Med. Sch. and Boston Hosp. for Women, Boston, Mass.

In infants recovering from hyaline membrane disease (HMD) persistent hypoxemia has been identified which seems not to result from venoarterial shunting [ADAMSON *et al.*, Pediatrics *44:* 168, 1969] but which could be due either to pulmonary diffusing defect or to ventilation/perfusion imbalance. Since the arterial-alveolar N_2 difference ($aADN_2$) and venous-alveolar N_2 difference ($vADN_2$) are unaffected by diffusion defect or by venoarterial shunting but are increase by impairment of ventilation with respect to perfusion, we have measured $vADN_2$ in a group of 10 normal low birth weight infants and in 4 infants convalescing from HMD. (Simultaneous comparison of $vADN_2$ and $aADN_2$ in seven infants revealed no significant difference.) In another 4 infants convalenscing from HMD the alveolar-arterial O_2 difference ($AaDO_2$) and arterial-alveolar CO_2 difference ($aADCO_2$) were also examined.

Number of infants	Age (days)	Birth weight (g)	Dx
10	18.5±11.2	1713.6±331.5	normal
4	7 to 16	1644 to 2892	HMD
4	10 to 33	1276 to 2395	HMD

Number of infants	$AaDO_2$ mm Hg	$aADCO_2$ mm Hg	$aADN_2$ mm Hg	$vADN_2$ mm Hg
				14.1±18.6
				77.0±36.8
	59±7.5	9.5±5.5	44.8±4.9	42.8±7.4

Unlike the hypoxemia seen in infants with early and developing HMD (which is due to an inequality of perfusion and a persistence of venoarterial shunting), the present findings suggest that it is inequality of ventilation which is mainly responsible for persistent hypoxemia during convalescence from HMD.

171 *Adjustment of Ventilation in the Newborn.* PETER A. M. AULD, ALFRED N. KRAUSS and JANE SOODALTER, Dept. of Ped., Cornell Univ. Med. Center, New York, NY.

In adapting to extrauterine life, ventilation and perfusion must soon become well-matched. The time course of this process can be assessed by measurement of the urinary-alveolar nitrogen gradient ($uADN_2$). An elevated $uADN_2$ is an indication of the maldistribution of $\dot{V}A/\dot{Q}$, specifically the presence of areas of low $\dot{V}A/\dot{Q}$. Seventeen healthy full-term neonates were studied serially from the first day of life. Values of $uADN_2$ within the range for normal adults were found (less than 10 mm Hg). Serial studies on 3 infants with evidence of aspiration pneumonia revealed $uADN_2$ consistently elevated above the normal. Children and adults with obstructive pulmonary disease were tested as a validation of the method and the expected elevation of $uADN_2$ was found. The studies indicate that (1) ventilation is uniformly distributed in the first days of life in normal infants; (2) maldistribution of ventilation is an important cause of arterial unsaturation in aspiration pneumonia. These results will be considered in the context of other studies of extrauterine pulmonary adaptation.

172 *Immaturity and Enhanced Susceptibility to Acute Hemodynamic Pulmonary Edema.* ELIHU P. REES, DORA A. STINSON, PAUL M. TAYLOR and DORIS W. WATSON, Dept. of Ped., Univ. of Pittsburgh Sch. of Med. and Magee-Women's Hosp.

The hypothesis that the immature lung is more susceptible than the mature lung to the development of acute pulmonary edema (PE), was tested by determining the transmural hydrostatic pressure in excess of whole blood colloid osmotic pressure (COP) necessary to produce PE in pups and adult dogs. Ten pups and ten adults were anesthetized with pentobarbital, parpalyzed with succinylcholine, and artificially ventilated. Transmural pulmonary artery wedge pressure (TPAWP) (as a reflection of vascular filtration pressure) was monitored continuously and whole blood COP intermittently. A balloon catheter in the thoracic aorta was inflated to produce an elevation of TPAWP in relation to COP that was maintained for 30 min by infusion of isosmotic dog blood to maintain a constant differential pressure (TPAWP-COP). Lungs were then removed and the degree of PE assessed by gross and microscopic appearance and determination of water content.

Interstitial edema first appeared with the following range of values:

	TPAWP mm Hg	Total protein g%	COP mm Hg	TPAWP-COP mm Hg
Adult dogs	24–30	5.6–7.0	19–22	6 to 8
Pups	10–15	4.2–5.6	12–17	−2 to +1