

Title : LUNG FUNCTION IN CHRONIC RESPIRATORY FAILURE IN INFANCY

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Introduction: To determine the sequential changes in pulmonary gas exchange and mechanics that contribute to chronic respiratory failure in infancy, we studied 7 infants who had been mechanically ventilated (MV) from age 1-3 days until 4-15 mos. Informed consent was obtained from the parents and permission granted by the Committee for the Protection of Human Subjects.

Methods: All infants were born prematurely (≤ 36 wk gestation) and had tracheostomies. Six infants had bronchopulmonary dysplasia (BPD), one aspiration. Every infant was severely growth retarded to \leq the 3rd percentile for weight; age range was 4-15 months; weight 2.1-7.5 kg. The IMV rates prior to the studies ranged from 0-55, F_{I,O_2} 0.21-0.45, inflation pressures (IP) 22-38 cmH₂O, and PEEP 0-8 cmH₂O. For the study of respiratory mechanics, the 5 infants on mechanical ventilation received an F_{I,O_2} of 0.50, IMV rate of 6, IP 25-30 cmH₂O, and PEEP 1-2 cmH₂O. The other two infants were studied with the same conditions without IMV. All infants were supine and tolerated the 3-5 minutes of the study well. No audible tracheal air leak occurred in any patient. End tidal PCO₂ (PECO₂) and airway pressure were determined at the tracheostomy; an esophageal balloon was placed in the distal third of the esophagus to measure intrathoracic pressure. A pressure transducer, pneumotachometer, integrator, and recorder were used to measure the patient's spontaneous minute volume (V_E), respiratory rate (f), tidal volume (V_T) and flow during the 10 seconds between IMV breaths. Calculations of inspiratory and expiratory lower airway resistance (R_{A-I} , R_{A-E}) were done at iso-volume points (50% of V_T). The dynamic lung compliance (C_L) was measured in the conventional manner at points of no flow.

Results. Patient data and the range of published normal values¹⁻⁵ are summarized in the Table. Patients 1 and 2 with the most severe BPD had a marked reduction in V_T , hypercapnia (PECO₂ ≥ 50 torr), and a higher R_{A-I} than the less ill infants. Patient 2 also had an exceptionally high R_{A-E} , indicating severe lower airways obstruction. Chest X-rays of both infants showed extreme hyperinflation. Patients 3-7, all recovering from pulmonary disease, had elevated V_E and V_T with normal or moderately elevated PECO₂ (36-46 torr). Five of the 6 patients (1, 2, 3, 5, 7) in whom R_A was measured had significant elevations in either R_{A-I} and/or R_{A-E} . Three patients (2, 4, 5) had borderline low C_L .

Discussion: Data in these infants with chronic respiratory failure associated with BPD, resulting from premature birth and severe acute pulmonary disease, indicate that mismatching of ventilation and perfusion with a large physiologic dead space (wasted ventilation) probably exists. Also, elevated R_A and borderline low C_L contribute to increased work of breathing and probable increases in metabolic rate. These abnormalities of ventilation and mechanics diminish as the infant recovers (Patient 6). We conclude that chronic respiratory failure with BPD in

infancy is characterized by high minute volumes, lower airways obstruction, and moderate to severe hypercapnia.

References.

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TABLE	Pat. No.	Diag- nosis	Age (Mos.)	Wt. (kg)	Duration Study		f	V_T (kg (ml))	V_E (kg (ml))	PECO ₂ (torr)	C_L (kg (ml))	R_{A-I} (cmH ₂ O/l/sec)	R_{A-E} (cmH ₂ O/l/sec)
					MV (mos.)	IMV rate							
	1	BPD	7	4.6	7	50	60	2	117	53	2.6	50	30
	2	BPD	8.5	3.4	9	30	50	4.4	221	50	1.1	67	230
	3	BPD	15.5	7.5	16	30	60	5.5	331	46	1.3	10	77
	4	BPD	13	6.3	13	4	50	7.1	393	45	1.1	10	37
	5	BPD	4.5	2.1	4	1	90	9.5	855	40	2.1	41	50
	6	Aspir.	10	6.7	8	0	30	13	390	36	-	-	-
	7	Apnea- BPD	3	2.8	2.5	0	60	12.5	750	42	3.2	48	71
	Normal Range:												
						30-50	5-7	150-300	30-40	2	1.5-2	10-30	40