

Original Articles

Acute Respiratory Failure in Infants with Bronchiolitis

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Bronchiolitis, an acute pulmonary infection of infants, is reported to result in acute respiratory failure and death in approximately 5 per cent of hospitalized cases. Sequential arterial pH, P_{aCO_2} and P_{aO_2} levels, determined in 30 infants with bronchiolitis, revealed an uncompensated respiratory acidosis, hypoxemia in air, and a variable degree of veno-arterial shunt. Measurement of physiologic deadspace-tidal volume ratios (V_{Dp}/V_T) and minute volumes in five infants showed that these infants had increased V_{Dp}/V_T ratios but could hold P_{aCO_2} below 65 mm. Hg by maintaining two- to threefold increases in minute volume.

As the infant became fatigued and minute volume fell to predicted basal levels, P_{aCO_2} rose to above 65 mm. Hg, the level at which clinical signs of acute respiratory failure appeared. Nasotracheal intubation, neuromuscular blockade, and mechanical ventilation for an average period of three days prevented fatal asphyxia and restored normal ventilation in infants with extreme hypercapnea (mean P_{aCO_2} 103 mm. Hg) and respiratory failure.

BRONCHIOLITIS, an acute pulmonary infection usually of viral etiology,^{1,2} affects infants less than two years of age. The inflammatory

process results in edema and cellular infiltration of the bronchioles and alveolar ducts. Retractions, hyperinflation of the chest, cyanosis, a respiratory rate over 40 per minute, and wheezing unresponsive to bronchodilating drugs characterize the disease. Bronchiolitis ordinarily runs its course in three to ten days. The typical roentgenogram of the chest shows considerable pulmonary hyperinflation with minimal or no infiltrates. The mortality rates of infants receiving hospital care vary from two to seven per cent, the majority of fatalities occurring in infants less than six months old.^{3,4} Although recovery from the infection itself usually appears complete, 30 per cent or more of these infants subsequently may develop allergic asthma.^{5,6}

Mechanical ventilation has been used successfully to treat moribund infants with bronchiolitis.⁷⁻¹⁰ However, there are no published reports associating the clinical signs of respiratory failure and its treatment with physiologic data. We undertook the present study to determine the pattern of arterial blood gas and acid-base changes in bronchiolitis, to correlate these with certain signs of acute respiratory failure, and to determine the effectiveness and safety of mechanical ventilation in the treatment of respiratory failure.

TABLE 1. Acid-Base Status in Bronchiolitis:
Adequate Ventilation Group

	Mean Values at Peak Severity of Illness in 19 Infants					
	Age (mos.)	Wt. (kg.)	pH	(H ⁺) (nEq./ L)	P_{aCO_2} (mm. Hg)	B.E.* (mEq./ L)
Mean	6.5	7.3	7.31	50	44.2	-4.1
S.E.	1.2	0.7	0.01	1	1.6	0.7

* No intravenous sodium bicarbonate prior to sample.

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Methods

Thirty infants admitted to the hospital with a clinical diagnosis of bronchiolitis comprised the subjects of this study. Mean age was 5.8 months, with a range from 1 to 18 months. Following infiltration of the skin and periarterial tissues with 2 per cent lidocaine (Xylocaine), blood was obtained from the femoral or brachial artery. Samples were taken shortly after admission to the hospital, at the time of peak clinical severity of the disease, and again after clinical improvement had been sustained for at least six hours. No complications resulted from repeated direct arterial sampling. All blood samples were analyzed in duplicate for pH and carbon dioxide tension (P_{aCO_2}) by the method of Astrup, and the base excess was calculated from the Sigaard-Andersen nomogram with corrections for the oxygen saturation of hemoglobin.¹¹

The arterial oxygen tension (PaO_2) was determined in duplicate with a micro-tip platinum electrode (Radiometer-Beckman), which responded linearly over the oxygen tension range studied. The magnitude of mixed venous-to-systemic arterial shunting as a fraction of the cardiac output was estimated from the PaO_2 in six infants who had been breathing 100 per cent oxygen in a small hood for at least 20 minutes.¹² An arterial-mixed venous oxygen content difference of 4.0 ml. per 100 ml. blood was assumed in the calculations. Blood gas and pH values were corrected to the patient's temperature.^{13, 14} A paramagnetic analyzer was used to determine the inspired oxygen concentration.

In five additional infants, the expired minute volume (V_E), physiologic deadspace (V_{Dp}), and carbon dioxide output (V_{CO_2}) were measured. The patient breathed room air through a low-resistance, non-breath-holding valve and a tightly applied Bennett infant mask. Total deadspace of the system was 6.3 ml. Expired gas was collected in a latex meteorological balloon and simultaneously, blood was obtained from the femoral artery for determination of $PaCO_2$. An aliquot of expired gas was analyzed in duplicate for carbon dioxide concentration with an infrared analyzer. Total gas volume was measured in a water-sealed spirometer, and the minute volume (V_E) and

TABLE 2. Acid-base Status in Bronchiolitis; Impending Respiratory Failure Group

Case No.	Age (mo.)	Wt. (kg.)	Initial					Peak Severity					Improved				
			pH	(H ⁺) (nEq/l.)	P_{aO_2} (mm. Hg)	P_{aE}^* (mEq/l.)	pH	(H ⁺) (nEq/l.)	P_{aO_2} (mm. Hg)	P_{aE}^{**} (mEq/l.)	T_{base}^{***} (hrs.)	pH	(H ⁺) (nEq/l.)	P_{aO_2} (mm. Hg)	P_{aE}^{***} (mEq/l.)	T_{base}^{***} (hrs.)	
10	7	11.2	7.35	44.7	24	-11.5	7.21	01.7	65	-3.2	135	7.31	45.7	44	-2.0	201	
11	4	6.2	7.18	60.1	54	-0.5	7.20	55.0	67	0.0	90	7.41	38.9	51	0.2	102	
12	1	5.0	7.21	01.7	68	-2.5	7.14	72.4	70	-5.0	12	7.30	50.1	63	3.0	17	
22	2	4.9	7.05	87.1	01	-10.0	7.17	67.9	82	-2.0	5	7.31	40.0	46	-2.0	7	
23	2	3.5	7.23	51.3	48	-	7.22	60.3	81	1.5	20	7.20	51.3	54	-2.0	25	
Mean	3	0.2	7.22	62.2	51	-	7.21	63.4	73	-1.7	54	7.33	47.0	52	0.6	70	
S.E.	-	-	0.05	7.3	7	1.8	0.02	3.0	3	1.1	-	0.02	2.2	3	1.7	-	

* No intravenous sodium bicarbonate prior to sample.

** Cases 10, 20 and 22 received intravenous sodium bicarbonate after the initial sample.

*** Time in hours between initial sample and peak severity or improved sample.

TABLE 3. Acid-base Status in Bronchiolitis:

Case No.	Age (mos.)	Wt. (kg.)	Peak Severity				Improved (Mechanical Ventilation)				
			pH	(H ⁺) (nEq./L.)	PaCO ₂ (mm. Hg)	B.E.* (mEq./L.)	pH	(H ⁺) (nEq./L.)	PaCO ₂ (mm. Hg)	B.E. (mEq./L.)	Time** (hrs.)
24	18	10.7	7.09	81.2	99	-4.4	7.22	60.3	78	1.8	1
25	12	11.3	7.00	99.9	125	-3.0	7.31	49.0	54	-0.5	1
26	2	5.2	7.12	75.9	104	0.5	7.51	30.9	32	3.0	2
27	2	4.8	7.15	70.8	125	6.0	7.42	38.0	57	10.0	4
28	3	8.3	7.17	71.7	77	-2.5	7.37	42.7	49	2.5	6
29	2	5.6	7.12	75.9	89	-2.0	7.50	31.6	28	-1.5	7
Mean	6.5	7.6	7.11	79.2	103	-0.9	7.38	42.1	49	2.5	3
S.E.	—	—	0.02	4.4	8	1.5	0.02	3.4	5	1.1	—

* Cases No. 26 and 28 received intravenous sodium bicarbonate prior to this sample.
** Time in hours after instituting mechanical ventilation.

tidal volume (V_T) determined. Using a modified Bohr equation,¹² physiologic deadspace (V_{Dp}), physiologic deadspace-tidal volume ratio (V_{Dp}/V_T) and carbon dioxide output (V_{CO_2}) were calculated. All values were corrected to the infant's body temperature and saturation (BTFS), and for apparatus deadspace.

Results

The patients were grouped according to PaCO₂ and clinical appearance at the peak severity of the illness. The following constitute our clinical criteria of acute respiratory failure in bronchiolitis: decreased-to-absent inspiratory breath sounds; severe thoracic retractions on inspiration; maximal hyperinflation of the thorax; cyanosis of lips and mucous membranes in 40 per cent oxygen; decreased-to-absent response to painful stimuli. Three clinical criteria sustained for more than an hour, with a PaCO₂ of 65 mm. Hg or higher, indicated acute respiratory failure.

Six infants with PaCO₂ values higher than 65 mm. Hg who manifested at least three of the clinical criteria constituted the "acute respiratory failure" group. Five infants, despite peak PaCO₂ of 65 mm. Hg or more, failed to meet three clinical criteria for acute respiratory failure and were classified as "impending respiratory failure." The 19 remaining infants did not develop the clinical signs of respiratory failure, maintained PaCO₂ values below 65 mm. Hg, and constituted the "adequate ventilation" group.

ACID-BASE STUDIES

Mean pH, PaCO₂, and base excess values obtained in the 19 infants of the "adequate ventilation" group at peak clinical severity of their illnesses are presented in table 1. These infants tended to have an acute, uncompensated respiratory acidosis. Fourteen infants developed PaCO₂ values above the normal range of 32.2 ± 6.6 (2 S.D.) mm. Hg¹⁵ with pH values below the normal range of 7.400 ± 0.056 (2 S.D.) for this age.¹⁵ The base excess varied from 0.5 to -11.5 mEq./L. but only two infants had values below the normal range of -3.9 ± 3.4 mEq./L. (2 S.D.).¹⁵ Although a metabolic acidosis in these infants may have been uncommon, there was no evidence of metabolic compensation for the degree of respiratory acidosis observed. It should be noted that an acute rise in PaCO₂ to the range of 45 to 65 mm. Hg can be expected to produce an *in vitro* depression artifact in base excess of only 1 to 2 mEq./L.¹⁶ Thus, the base excess values reported are a reasonable approximation of the *in vivo* base excess.

The mean and individual acid-base values in the five infants with "impending respiratory failure" are presented in table 2. Initially each infant had either a combined respiratory and metabolic acidosis or an uncompensated respiratory acidosis. At peak severity of illness all patients had a severe, uncompensated respiratory acidosis with PaCO₂ levels between 65 and 82 mm. Hg. Clinical improvement occurred during the following two to 66 hours,

Acute Respiratory Failure Group

Total Time on Ventilator (hrs)	Improved (Unassisted Ventilation)					Convalescent				
	pH	(H ⁺) (nEq./L.)	Paco ₂ (mm. Hg)	B.E. (mEq./L.)	Time*** (hrs.)	pH	(H ⁺) (nEq./L.)	Paco ₂ (mm. Hg)	B.E. (mEq./L.)	Time**** (days)
85	7.46	34.7	45	2.0	4	7.47	33.9	36	2.5	10
48	7.44	36.3	29	-2.5	5	7.37	42.7	46	0.0	6
61	7.39	40.7	35	-3.0	3	7.46	34.7	34	1.0	11
216	7.40	39.8	60	8.6	6	7.44	36.3	37	1.0	17
54	7.40	39.8	43	2.0	1	7.35	44.7	35	-6.0	8
44	7.44	36.3	33	-1.5	1	7.37	42.7	40	-2.0	3
85	7.42	37.9	41	0.9	3	7.41	39.2	38	0.6	9
—	0.01	1.0	4	1.8	—	0.01	1.4	1	0.7	—

*** Time in hours after cessation of mechanical ventilation.
**** Time in days after peak severity of illness.

by which time the PaCO₂ had fallen below 65 mm. Hg.

The clinical picture of acute respiratory failure developed in six infants whose individual and mean acid-base values are presented in table 3. Figure 1 depicts the mean values at various stages of the illness. Three infants (Cases 26, 27, and 28) had initial PaCO₂ values of 86, 78, and 58 mm. Hg, respectively, prior to the peak-severity sample. Signs of acute respiratory failure subsequently developed over a period of hours. The other three infants (Cases 24, 25, 29) were in acute respiratory failure on admission to the hospital and the samples at peak severity were their initial samples.

Acute respiratory failure is associated with a severe, uncompensated respiratory acidosis. Only one infant (Case 27), who had been severely hypercapneic for 44 hours, had evidence of partial metabolic compensation (base excess 6.0 mEq./L.). The failure of these infants to achieve rapid metabolic compensation in the presence of PaCO₂ levels higher than 75 mm. Hg resulted in severe acidosis with an arterial pH below 7.20.

After nasotracheal intubation, neuromuscular blockade with *d*-tubocurarine, and positive-pressure mechanical ventilation, PaCO₂ values decreased within seven hours to levels below 60 mm. Hg in all but one infant. The duration of mechanical ventilation varied from two to nine days, the average being three days. None of the infants became acidotic during

convalescence, although two had slightly elevated PaCO₂ values.

OXYGEN TENSION STUDIES

The mean value of 40 PaO₂ determinations in 26 infants breathing air during the acute phase of illness is presented in table 4. The PaO₂ varied from 30 to 93 mm. Hg, and all but one infant had PaO₂ values below 90 mm. Hg. A significant inverse linear correlation ($r = -0.45$, $P < 0.01$) existed between the PaCO₂ and PaO₂ during breathing of air, and can be defined by the equation: PaO₂ = 84.4 - 0.50 (PaCO₂). This suggests that alveolar hypoventilation contributes to the arterial hypoxemia. The regression equation indicates that as PaCO₂ exceeds 65 mm. Hg, the minimum level associated with impending respiratory failure, PaO₂ falls below 50 mm. Hg during breathing of air.

The PaO₂ values of six infants breathing 100 per cent oxygen (table 4) reveal that a mixed venous-to-systemic arterial shunt also may contribute to arterial hypoxemia. The magnitude of this shunt varied from 8 to 17 per cent of the cardiac output, and tended to be somewhat greater in patients with acute respiratory failure. Our observations indicated that infants in this age range who are free of cardiopulmonary disease had shunts of less than 10 per cent of their cardiac outputs.

PHYSIOLOGIC DEADSPACE STUDIES

The individual data obtained from the measurement of physiologic deadspace-tidal vol-

ume ratio (V_{D_P}/V_T) and minute volume (V_E) in five infants are presented in table 5. Four of the infants, classified in the "adequate ventilation" group, had V_{D_P}/V_T ratios between 0.55 to 0.69 and $P_{a_{CO_2}}$ values of 38 to 61 mm. Hg. In these infants $P_{a_{CO_2}}$ values remained below 65 mm. Hg despite an elevated V_{D_P}/V_T ratio. This was accomplished by maintenance of a large minute volume with an increase in both tidal volume and respiratory frequency. The magnitude of this increase in minute volume varied from 113 to 317 per cent of the basal values predicted for the infants by the Radford nomogram.¹⁷ The increase in minute volume in infants with $P_{a_{CO_2}}$ values below 65 mm. Hg was associated with increase in carbon dioxide output from 129 to 208 per cent greater than that predicted by the data of Benedict and Talbot.¹⁸ The fifth infant, who was in impending respiratory failure, had a $P_{a_{CO_2}}$ of 70 mm. Hg, a V_{D_P}/V_T of 0.76, a minute volume 113 per cent of the predicted value, and a carbon dioxide output only 74 per cent of that predicted. This case illustrates that, in the presence of a high V_{D_P}/V_T ratio, a minute volume close to predicted basal values results in failure to excrete carbon dioxide and a rise in $P_{a_{CO_2}}$ to levels associated with clinical respiratory failure.

CLINICAL MANAGEMENT

All infants received humidified oxygen in concentrations between 35 and 60 per cent in order to provide normal $P_{a_{O_2}}$ values. Restoration of the $P_{a_{O_2}}$ to or above normal levels did not result in any consistent increase in $P_{a_{CO_2}}$ as has been described in adults with chronic hypoxemia.¹⁹ Throughout the acute phase of the illness, dextrose in 0.2 per cent saline solution was infused intravenously in a volume calculated to correct dehydration and provide maintenance fluids.²⁰ To provide partial compensation for respiratory acidosis and restore arterial pH to a level above 7.25, the extracellular base deficit of the acidotic infant was corrected to zero with intravenous sodium bicarbonate. Assuming 30 per cent of the body weight as the extracellular volume in infants,²¹ the following formula was used: $\text{NaHCO}_3 \text{ mEq. (total)} = \text{base deficit (mEq./L)} \times 0.3 \text{ body wt. (kg.)}$. Broad-spectrum antibiotics were used in each case because of the inability to exclude bacterial bronchopneumonia as an etiologic or associated factor. All infants with impending or acute respiratory failure also received intravenous hydrocortisone in a dose of 10 mg./kg./day, without apparent benefit. In several instances, chest physiotherapy²² followed by direct laryngoscopy and tracheal aspiration resulted in improved inspiratory

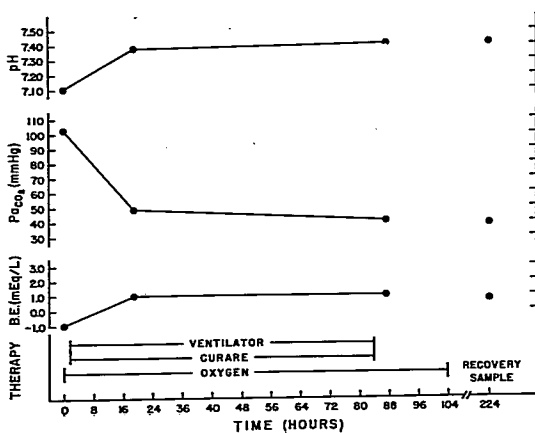


FIG. 1. Mean acid-base values in six infants with acute respiratory failure due to bronchiolitis. The time in hours is calculated from the time of the sample obtained at peak clinical severity of the illness.

TABLE 4. Arterial Hypoxemia in Bronchiolitis

		Age (mos.)	Wt. (kg.)	PaO ₂ (mm. Hg)	PaCO ₂ (mm. Hg)	D _{A-O₂} * (mm. Hg)	Q _s /Q _t **	Group
Breathing air during acute disease	Mean	—	—	62	46	—	—	Adequate ventilation
	N	—	—	40	40	—	—	
	S.E.	—	—	12	11	—	—	
Veno-arterial shunt during acute disease	Case 5	5	5.9	540	44	129	0.09	Adequate ventilation
	Case 10	8	6.0	490	38	185	0.12	Adequate ventilation
	Case 21	1	5.0	525	68	117	0.08	Impending respiratory failure
	Case 24	18	10.7	340	99	274	0.17	Acute respiratory failure
	Case 25	12	11.3	427	125	158	0.11	Acute respiratory failure
	Case 29	2	5.6	410	89	214	0.14	Acute respiratory failure

* D_{A-O₂} = alveolar-arterial O₂ tension gradient.

** Q_s/Q_t = fraction of cardiac output shunted assuming arterial-mixed venous O₂ content difference is 4.0 ml.

breath sounds, decreased retractions, and a decreased PaCO₂.

Once the diagnosis of acute respiratory failure was made, the trachea was intubated orally. Following tracheobronchial toilet and manual hyperventilation of the lungs, nasotracheal intubation was performed with a polyvinyl chloride tube,⁹ and the tube was secured according to a technique previously described.²³ Then neuromuscular blockade was achieved with intravenous *d*-tubocurarine, 0.6 mg./kg. initially and 0.2 mg./kg. at one- to six-hour intervals. A Bird Mark VIII ventilator provided mechanical ventilation in five infants. A frequency of 20 to 24 breaths per minute with a slow inspiratory flow rate, an inspiratory-expiratory time ratio of 1:2, and an initial inspiratory line pressure of as much as 45 cm. water were used. In one infant (Case 29) the lungs were ventilated with a constant-volume ventilator using a 400 ml. cylinder.⁹

Care of the airway included instillation of 1.0 ml. saline solution into the trachea every hour, followed by gentle chest percussion and vibration,²² sterile tracheobronchial aspiration, and manual hyperventilation. The electrocardiogram and rectal temperature were monitored continuously, and the infants received constant intensive nursing care. After the

first day of mechanical ventilation, a milk formula was given through a nasogastric tube. Arterial pH, P_{CO₂}, and P_{O₂} were determined at 12- to 24-hour intervals.

Within an average of three days, wheezing and thoracic hyperinflation had disappeared. The PaCO₂ and PaO₂ could be maintained at normal levels with inspiratory line pressures lower than 25 cm. water and inspired oxygen concentrations of less than 40 per cent. At this time, the neuromuscular blockade was reversed with intravenous neostigmine (0.07 mg./kg.) preceded by atropine (0.02 mg./kg.). Apparently normal muscle strength returned within minutes. The infant then breathed humidified oxygen without assistance for one to six hours, after which the PaCO₂ was 45 mm. Hg or less, except in one infant (Case 27), who had developed bronchopneumonia due to *Klebsiella aerobacter* which required parenteral kanamycin, intensive pulmonary care, and nasotracheal intubation to facilitate removal of secretions for an additional nine days. In the other infants, tracheal secretions became minimal within two days after discontinuing mechanical ventilation, thus permitting removal of the nasotracheal tube. All infants were kept in a high-humidity atmosphere for seven days following extubation.

Three complications occurred in conjunction with the management of acute respiratory failure. A unilateral pneumothorax occurred in one infant on the second day of mechanical ventilation. Another infant developed a

⁹ Portex tube, S. Smith and Sons, Ltd., Jamaica, Long Island, New York.

²² Pediatric modification of the Emerson Post-operative Ventilator, J. H. Emerson Co., Cambridge, Massachusetts.

TABLE 5. Physiologic Deadspace and

Case No.	Age (mos.)	Wt. (kg.)	Ht. (cm.)	B.S.A. (M ²)	Temp. (°C)	Observed V_E (l./min.)	Predicted* V_E (l./min.)	f (br./min.)	V_T (l.)
8	5	7.4	64	0.32	36.5	4.540	1.430	75	0.060
21	1	5.2	54	0.26	36.5	1.130	1.000	56	0.020
22	2	6.1	59	0.30	38.0	2.310	1.120	67	0.035
23	2	3.5	49	0.20	36.7	1.840	0.660	34	0.054
30	3	5.1	53	0.25	36.9	1.670	0.960	59	0.028

* From Radford nomogram.¹⁷

degree of subglottic stenosis three weeks following extubation; this has improved with subsequent tracheal dilations. The most serious apparent complication was the *Klebsiella* bronchopneumonia mentioned above.

All of the infants studied recovered without major pulmonary or nervous system sequelae except two that are known to have developed bronchial asthma.

Discussion

Recent pulmonary function studies have shown that bronchiolitis is associated with an increased respiratory frequency and minute volume, considerable decrease in dynamic compliance, and as much as a sixfold increase in the work of breathing.^{24, 25} Our data confirm the findings by others of arterial hypoxemia^{7, 26} and moderate respiratory acidosis^{7, 27, 28, 29} at the peak severity of illness in the majority of these infants.

To our knowledge, there are no published data on the size of physiologic deadspace or the relation of deadspace to tidal volume in infants free of cardiopulmonary disease in this age group. However, Nelson, *et al.*,³⁰ have shown that the V_{D_P}/V_T ratio of normal, spontaneously breathing newborn infants is 0.25 ± 0.07 (S.D.), that is, equivalent to that found in normal adult man. It would seem reasonable to assume, therefore, that our patients had V_{D_P}/V_T ratio of approximately 0.3 when healthy.

The data in table 5 indicate that the volume of the physiologic deadspace varies independent of tidal and minute volumes. Thus, Case 23 had the largest deadspace per unit body size (61 ml./cm. height) despite a minute and tidal volume nearly three times basal predicted levels. The V_{D_P}/V_T ratio of 0.55 in this case illustrates that even at large tidal volumes a

disproportionate increase in physiologic deadspace can exist. This does not preclude normal alveolar ventilation, signified here by a P_{aCO_2} of 38 mm. Hg. However, normal ventilation can be achieved only by a considerable increase in minute volume.

As the infant fatigues to the point where his minute volume falls to basal predicted levels, in the presence of a V_{D_P}/V_T ratio above 0.70, severe carbon dioxide retention and the clinical signs of acute respiratory failure occur. Our finding that these signs appear as the P_{aCO_2} exceeds 65 mm. Hg agrees closely with similar observations in adults³¹ and in children with status asthmaticus.^{32, 33}

In the infant whose work of breathing has been greatly increased, leading to fatigue and potentially fatal impairment of alveolar ventilation, the most reasonable therapeutic approach is to relieve the work and augment alveolar ventilation. This can be achieved most readily by muscular relaxation and mechanical positive-pressure ventilation. In our experience, alternatives such as tracheotomy³⁴ or bronchoscopy³⁵ do not provide a sustained improvement of alveolar ventilation in bronchiolitis.

What percentage of infants with bronchiolitis meeting the clinical criteria of respiratory failure will progress to cardiac arrest without mechanical ventilation? Are the risks to the infant who meets these criteria less with mechanical ventilation than with allowing him to continue unassisted? These questions cannot be answered definitely at this time.

Reynolds⁷ described the role of mechanical ventilation in the successful management of one infant with respiratory failure from bronchiolitis. All seven moribund infants with bronchiolitis reported by July¹⁰ recovered without major sequelae following mechanical

Minute Ventilation in Bronchiolitis

P_{aCO_2} (mm. Hg)	P_{ECo_2} (mm. Hg)	V_{Df}/V_T	V_{Df} (l.)	Observed V_{Co_2} (l.)	Predicted** V_{Co_2} (l.)	$V_{E,obs}$ as % of $V_{E,pred}$	$V_{Co_2,obs}$ as % of $V_{Co_2,pred}$
45	17	0.62	0.037	0.104	0.050	317	208
70	17	0.76	0.015	0.026	0.035	113	74
61	19	0.69	0.024	0.062	0.035	206	177
38	17	0.55	0.030	0.044	0.023	281	191
48	19	0.60	0.017	0.044	0.034	174	129

** From data of Benedict and Talbot.¹³

ventilation. When these are considered in addition to the six infants we have treated, it would seem that mechanical ventilation will prove to be justifiable and effective in the management of bronchiolitis with respiratory failure.

Conclusions

Bronchiolitis is reported to result in acute respiratory failure and death in 5 per cent of hospitalized cases. Sequential determination of arterial pH, P_{CO_2} , and P_{O_2} levels in infants with bronchiolitis revealed a moderate-to-extreme uncompensated respiratory acidosis, hypoxemia in air, and veno-arterial shunts of variable degrees. By maintaining a two- to threefold increase in minute volume, these infants could maintain P_{aCO_2} values below 65 mm. Hg despite considerably enlarged physiologic deadspaces. As the infant becomes fatigued, minute volume falls to predicted basal levels, and P_{aCO_2} rises to above 65 mm. Hg, the level at which clinical signs of acute respiratory failure appear. Nasotracheal intubation, neuromuscular blockade, and mechanical ventilation for an average period of three days can prevent fatal asphyxia and restore normal ventilation in the infants with extreme hypercapnea and respiratory failure.

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