Normalization of Blood Carbon Dioxide Levels by Transition From Conventional Ventilatory Support to Noninvasive Inspiratory Aids

Irving I. Haber, DO, John R. Bach, MD

ABSTRACT. Haber II, Bach JR. Normalization of blood carbon dioxide levels by transition from conventional ventilatory support to noninvasive inspiratory aids. Arch Phys Med Rehabil 1994;75:1145-50.

• Chronic hypocapnia seems to be common in long-term ventilator assisted individuals (VAIs) with paralytic/restrictive respiratory conditions. It has predominantly been reported for VAIs using intermittent positive pressure ventilation (IPPV) delivered via tracheostomy tubes. Chronic hypocapnia decreases ventilator-free breathing time (VFBT) and may be associated with increased bone resorption. Attempts to reverse chronic hypocapnia by decreasing minute ventilation and providing supplemental carbon dioxide have failed because of air hunger and patient resistance. We maintained normocapnia in 22 24-hour-a-day VAIs by using noninvasive IPPV. Chronic hypocapnia was corrected in three VAIs and hypercapnia in two VAIs by switching from conventional ventilatory support to the use of noninvasive inspiratory muscle aids. The other 17 VAIs remained normocapneic by being managed by noninvasive ventilatory support from onset of ventilatory failure. Eleven of these VAIs had been intubated or tracheostomized for brief periods but were successfully returned to noninvasive support. We conclude that alveolar ventilation can be maintained within normal range for VAIs who use noninvasive IPPV and can be normalized by transition from conventional tracheostomy IPPV to noninvasive IPPV.

© 1994 by the American Congress of Rehabilitation Medicine and the American Academy of Physical Medicine and Rehabilitation

In autonomously breathing control subjects arterial carbon dioxide tensions ($PaCO_2$) range from 36 to 42 torr. End-tidal carbon dioxide tensions ($EtCO_2$) are 1 to 2 torr lower than $PaCO_2$ for control subjects as well as for ventilator-assisted individuals (VAIs) without significant intrinsic pulmonary disease.[1] Until recently, little attention has been given to the carbon dioxide tensions of chronic VAIs. Then, in 1989, Banzett and coworkers described chronic hypocapnia in a high-level spinal cord injured (SCI) individual receiving 24-hour-a-day intermittent positive pressure ventilation (IPPV) via a tracheostomy tube.[2] In 1992 Manning and colleagues reported severe hypocapnia in five tracheostomy IPPV supported traumatic SCI individuals with vital capacities (VCs) less than 90 mL.[3] We have described chronic hyperventilation in 33 VAIs with a variety of paralytic/restrictive conditions (J.R.B., unpublished data, 1994). Patterson and colleagues reported similarly severe hypocapnia in a poliomyelitis VAI with less than 30mL of VC supported by an iron lung.

Patterson suggested that their hypocapnic patient "was unable to increase the magnitude of the volley of impulses from the stretch receptors of the lung, or the frequency of these volleys (and was therefore) unable to increase by the Hering-Breuer mechanism the inhibition of inspiratory cell discharge in the respiratory center." Earlier, Wright[5] suggested that loss of such inhibition might play a central role in the production of breathlessness. All of the hypocapnic VAIs described were unable to trigger their ventilators and had maintained the same ventilator delivered volumes for extended periods of time. Thus, the absence of control over the inspiratory effort may be in some way related to the increased inspiratory cell activity in the production of breathlessness at hypocapnic levels.

Air satiety is governed by $PaCO_2$.[6] In control subjects any increase in $PaCO_2$ from baseline causes an increase in alveolar ventilation.[7] In an attempt to reverse chronic hypocapnia in VAIs, the tracheostomy IPPV users experienced severe dyspnea when with the addition of CO_2 to inspired gases $PaCO_2$ was increased by a mean of 18 torr to a $PaCO_2$ of 38 torr.[7] In another study of hypocapnic tracheostomy IPPV users elevations of $PaCO_2$ by only 10mmHg created air hunger.[2] When IPPV tidal volumes were decreased to reverse hypocapnia dyspnea began at a $PaCO_2$ of 26 torr.[7] When supplemental oxygen was given breath-holding could be maintained to an increase of 16 torr, however, the increased tolerance to $PaCO_2$ was dependent on continued oxygen supplementation.[8] Gradual reductions in tidal volume also created intolerable air hunger in SCI tracheostomy IPPV users whose $PaCO_2$ and PaO_2 were maintained constant by control of the inspired gas mixture concentrations.[3]

Tracheostomy IPPV provides essentially a closed system of ventilatory support in which alveolar ventilation is supported passively. Open noninvasive systems of ventilatory support include the delivery of IPPV via nose (fig 1) or mouthpieces (figs 2 and 3) or, occasionally, interfaces that cover both the nose and mouth.[9-14] Although mouthpiece IPPV is delivered via simple mouthpieces fixed adjacent to or held in the mouth for daytime ventilatory support,

From the Continental Rehabilitation Hospital of Terre Haute (Dr. Haber), Terre Haute, IN; and the Department of Physical Medicine and Rehabilitation (Dr. Bach), UMD-New Jersey Medical School, Newark; Kessler Institute for Rehabilitation Ventilator Unit (Dr. Bach), West Orange, NJ.

Submitted for publication January 27, 1994. Accepted in revised form April 29, 1994.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the authors or upon any organizations with which the authors are associated.

Reprint requests to John R. Bach, MD, Department of Physical Medicine and Rehabilitation, University Hospital B-239, 150 Bergen Street, Newark, NJ 07103.

© 1994 by the American Congress of Rehabilitation Medicine and the American Academy of Physical Medicine and Rehabilitation

0003-9993/94/7510-2594$3.00/0

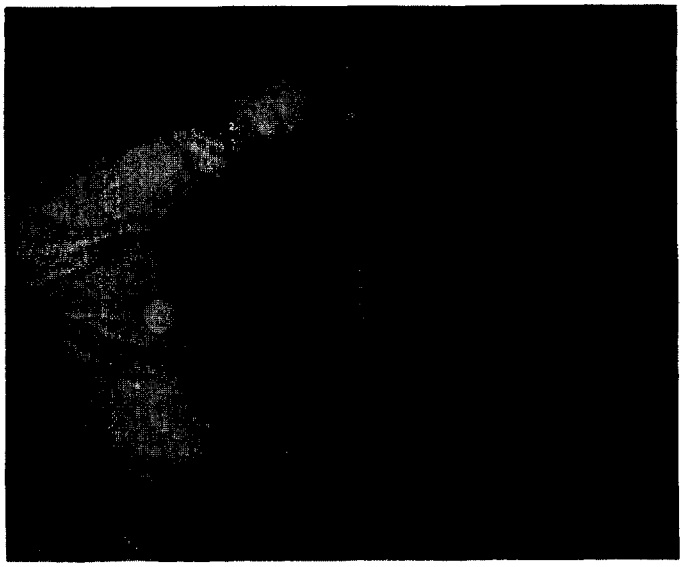

Fig 1—Case 5 from table 1. The tracheostomy tube was removed and the patient converted directly to 24-hour noninvasive IPPV. He used nasal IPPV overnight as pictured here and mouthpiece IPPV during daytime hours.

mouthpiece retention and IPPV effectiveness are greatly improved during sleep when a lipseal[a] is used for mouthpiece retention. Mouthpiece and nasal IPPV can be successful for as much as 24-hour ventilatory support in VAIs with little or no VC or ventilator-free breathing time (VFBT), however, they seem to be dependent on the intactness of ventilatory drive for their success. As VAIs using noninvasive IPPV sleep, ventilator delivered (insufflation) volumes leak out of the nose or mouth rather than go entirely into the lung. Higher ventilator delivered volumes than those typically used during conventional tracheostomy IPPV compensate in part for variable air delivery leakage during noninvasive IPPV.[9] Chemotaxic mediated reflex muscular activity, however, which most often occurs with sleep-stage changes, intermittently decreases or eliminates leakage to normalize oxyhemoglobin saturation and avert excessive hypoxia and hypoventilation during sleep (J.R.B., unpublished data, 1994).

Fig 2—Case 3 from table 1. A gooseneck clamp attached to the wheelchair frame maintained the mouthpiece near this patient's mouth for easy access.

Fig 3—This Duchenne muscular dystrophy patient with no ventilator-free breathing time is using mouthpiece IPPV with lipseal[a] retention for nocturnal ventilatory support.

The purpose of this study was to determine if chronic hypocapnia could be reversed by switching chronically hyperventilated tracheostomy IPPV users with little or no VFBT to noninvasive methods of ventilatory support. No previous studies have attempted this. In addition, we report 17 VAIs who remained essentially normocapnic despite using noninvasive methods of ventilatory support 24 hours a day.

PATIENTS AND METHODS

The patient records were reviewed of VAIs managed in two long-term rehabilitation ventilator units. All of the VAIs used portable volume or pressure triggered ventilators around the clock. The inclusion criteria was severity of respiratory insufficiency such that no more than a maximum of 40 minutes of VFBT was possible at any time during the study period. Subjects were excluded when the presence of parenchymal lung disease necessitated the use of supplemental oxygen while using IPPV. All were surveyed for symptoms of sleep-disordered breathing or chronic alveolar hypoventilation and underwent arterial or $EtCO_2$ evaluations. Five VAIs who were transferred from tracheostomy IPPV to noninvasive IPPV had arterial blood gas and/or $EtCO_2$ measurements performed both before and after transition. Their ages, pulmonary function, CO_2 tensions, and ventilator use histories are shown in table 1.

In addition, 17 chronically VAIs who underwent multiple evaluations of $EtCO_2$ yearly for 4 to 20 consecutive years while having less than 40 minutes of VFBT were identified and studied. Although all used noninvasive ventilatory support 24-hours-a-day throughout the study period, 9 had been temporarily intubated to receive IPPV during surgical procedures, and 2 were briefly tracheostomized. These 11 VAIs all successfully returned to 24-hour noninvasive ventilatory support. The population consisted of 11 men and 6 women

Table 1: Carbon Dioxide Tensions for Ventilator Users Switched From Tracheostomy to Nontracheostomy Methods

				At the Time of Transition to Noninvasive IPPV					
							CO_2 (torr)[¶]		
Case	Diagnosis	Age*	Sex	VC[†]	VFBT[‡]	Trach IPPV	Range	Mean	Pts
1	SCI	71.4	M	120	<1	7/90-11/90	18-23	21	4A
2	SCI	17.2	F	180	<1	2/93-6/93	16-22	19	7B
3	MD	38.6	F	125	<1	5/87-7/92	24-31	27	8A
4	MD	48.7	F	820	[‖]	11/88-8/90	65-73	68	6A
5	Res	53.6	M	570	<1	2/90-7/90	47-68	55	7A

	After Transition to Noninvasive IPPV			
		CO_2 (torr)		
VFBT**	Noninvasive IPPV	Range	Mean	Pts
<1	11/90-3/92	32-36	34	5B
20[††]	6/93-present	31-34	33	3B
480[††]	7/92-6/93	32-41	36	4B
120	8/90-present	35-42	39	3B
20	7/90-present	34-41	37	5B

Abbreviations: MD, muscular dystrophy; Res, restrictive pulmonary syndrome associated with congenital lymphedema and lung resection; trach, tracheostomy.

* Age at onset of ventilator dependence.

† Vital capacity in milliliters at the time of transition from tracheostomy to noninvasive IPPV.

‡ VFBT in minutes at the time of transition from tracheostomy IPPV to noninvasive IPPV. VFBT is defined as the longest period of time that autonomous breathing can be tolerated without dyspnea, blood gas derangement, and need to return to ventilatory support.

‖ Chronically hypercapnic during unassisted breathing but able to decrease $PaCO_2$ to normal limits for 5 to 10 minutes by volitionally increasing tidal volumes.

¶ CO_2 range, mean, and the number of CO_2 determinations (pts) in different months by arterial blood gas analyses (A) or by end tidal PCO_2 analyses (B).

** VFBT in minutes 3 months after transition to noninvasive IPPV.

†† With glossopharyngeal breathing.

with a mean age of 47 ± 15 years at the time of the first recorded $EtCO_2$ in the study. Their diagnoses, VCs, and ventilator-use histories are listed in table 2.

The $EtCO_2$ values were averaged in 6-month blocks as one interval mean. At the time of each $EtCO_2$ evaluation the VAIs had no signs of acute respiratory illness during which increased weakness and airway secretions might have decreased ventilatory capacity and increased $EtCO_2$. The $EtCO_2$ was measured with a LB3 capnograph[b] or a Microspan 8090 capnograph.[c] These devices were calibrated before each measurement. All measurements were noted when the VAIs with no VFBT were using their ventilators or while breathing comfortably within minutes of beginning autonomous breathing when possible.

RESULTS

None of the VAIs in this study reported respiratory symptoms. The mean $EtCO_2$ levels for the 17 noninvasive VAIs are listed in table 3. The pre-transition and posttransition (from chronic tracheostomy IPPV to noninvasive IPPV) carbon dioxide tensions of the 5 VAIs so managed are indicated in table 1. Only case 4 had significant VFBT.

Case 1 was a 71-year-old man with complete C2 tetraplegia. He had frequent aspiration and pooling of food and

Table 2: Extent of Need and Methods of Ventilatory Support

		Noninvasive Ventilatory Assistance Methods*			
Diagnoses[†]	Users	Age[‡]	VC[‖]	Years[¶]	Years**
ALS	2	69 ± 4	300 ± 113	4.0 ± 0.2	0.2 ± 0.1
Encephalopathy	2	71 ± 1	440 ± 240	8.0 ± 2.6	1.5 ± 2.3
Myopathy	7	37 ± 9	137 ± 116	17.3 ± 7.8	8.1 ± 4.0
Polio	5	44 ± 8	204 ± 319	32.8 ± 7.9	11.6 ± 3.8
Myelopathy	1	51	360	19	0.5
Total	17	48 ± 15	264 ± 252	19.8 ± 7.7	4.4 ± 3.4

* Daytime methods: 12, mouthpiece IPPV; 5, intermittent abdominal pressure ventilator (IAPV); nocturnal use methods: 12, lipseal IPPV; 4, IAPV; 1, iron lung.

† They had the following diagnoses: ALS, amyotrophic lateral sclerosis; encephalopathy, brainstem hemorrhage, arthrogryposis multiplex, Friedrich's ataxia, meningitis, multiple sclerosis, cerebrovascular disease; myopathy, Duchenne muscular dystrophy and other myopathies; polio, postpoliomyelitis; and myelopathy.

‡ Age at the last $EtCO_2$ measurement in the study.

‖ Vital capacity at the first $EtCO_2$ measurement in the study.

¶ Years of ventilator use.

** Years of ventilator use before first $EtCO_2$ measurement.

Table 3: End-Tidal PCO_2 Levels for 17 Noninvasive IPPV Users

	In the Sitting Position			In the Supine Position On Noninvasive IPPV		
Diagnosis	6-Month*	Data Pts†	$EtCO_2$ (torr)	6-Month*	Data Pts†	$EtCO_2$ (torr)
ALS	11	2.3	31.4 ± 3.6	12	2.1	30.3 ± 3.5
Encephalop	19	2.3	39.6 ± 5.8	18	2.3	34.8 ± 5.7
MD	58	3.2	40.8 ± 8.6	42	2.9	37.6 ± 7.6
Polio	54	2.6	30.8 ± 8.7	37	2.3	30.8 ± 8.8
Myelopathy	11	2.1	34.2 ± 2.3	10	2.0	31.1 ± 6.8
Total	153	2.7	36.0 ± 6.8	119	2.5	33.8 ± 8.0

* 6-month intervals with at least one $EtCO_2$ level determination.
† Independent observations of $EtCO_2$ per 6-month interval with observations separated by 1 month or more.

fluids in his right piriform sinus. He was converted to 24-hour noninvasive IPPV and relied heavily on manual and mechanical coughing aids, ie, mechanical insufflation-exsufflation,[15] to eliminate airway secretions and other debris without the need for tracheal intubation and suctioning.

Case 2 was a 17-year-old woman with complete C2 tetraplegia who following conversion to 24-hour noninvasive IPPV mastered glossopharyngeal breathing (GPB) sufficiently to be free from ventilatory support for up to 20-minute periods.

Case 3 was a patient who used tracheostomy IPPV for 5.5 years with no VFBT. She had more than 30 febrile episodes associated with tracheitis treated by intravenous antibiotic therapy in 5.5 years. She has experienced no febrile episodes during at least the first year following conversion to nocturnal nasal IPPV and daytime mouthpiece IPPV and has mastered GPB sufficiently for up to 8 hours of VFBT. Figure 4 is a graphic representation of her pretransition and posttransition blood carbon dioxide tensions.

Only case 4 had significant VFBT, albeit with chronic hypercapnia, when transferred to noninvasive aid. She did not respond to weaning attempts over a 6-month period and was being managed on synchronized intermittent mandatory ventilation with supplemental oxygen despite arterial blood gas carbon dioxide tensions ranging from 65 to 73 torr. During a trial of chest-shell use her $PaCO_2$ was 70 torr. After transition to daytime mouthpiece IPPV and nocturnal nasal IPPV, her $EtCO_2$ returned to normal range and she can now tolerate up to 2 hours of VFBT without hypercapnia despite having continued diminution in her VC.

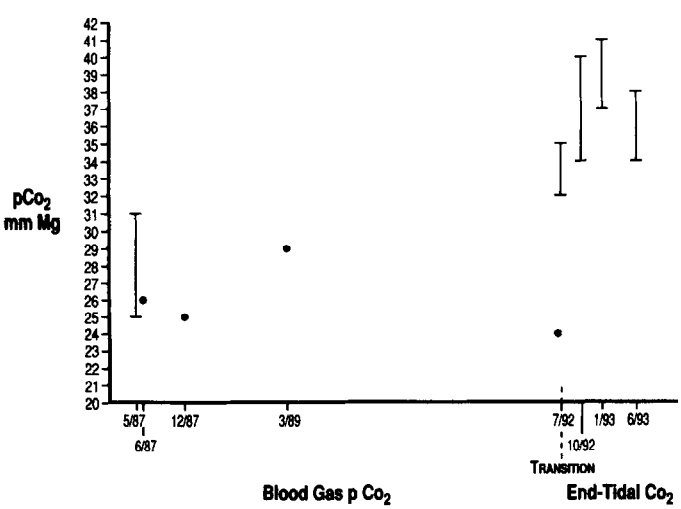

Fig 4—Graph of pretransition and posttransition carbon dioxide tensions for case 3.

Case 5 also had a significant increase in VFBT but this occurred with an increase in VC after transition to noninvasive IPPV. He had congenital lymphedema and had had a partial left lung resection during childhood. He complained of symptoms suggestive of chronic alveolar hypoventilation and progressive dyspnea for over 10 years for which he was hospitalized on three occasions over the past 3 years. He also noted that he had been taking bronchodilators for asthma since 1980 without apparent benefit. He was dyspneic on admission April 18, 1990, with an arterial blood gas of pH 7.43, pCO_2 50, pO_2 61, and bicarbonate 33.7. He developed progressive respiratory failure and was intubated on May 16th, subsequently tracheostomized, and lost all VFBT while using tracheostomy IPPV. Weaning attempts terminated as $PaCO_2$ levels quickly reached 60 torr. On August 30th he began using mouthpiece IPPV during daytime hours with pulse oximetry biofeedback[16] and was able to tolerate mouthpiece IPPV most of the day and nasal IPPV overnight within 2 weeks. On September 11th the tracheostomy tube was removed and the site buttoned for 1 week before being allowed to close. Bronchodilators were discontinued. In January 1991, after discharge from rehabilitation, he returned to work as an accountant using a walker with a ventilator tray despite requiring 24-hour ventilatory support with less than 2 minutes of VFBT.[17] His VFBT has subsequently increased to 20 minutes supine and 4 hours erect with increases in VC to 1,080mL. He has had no further respiratory symptoms or hospitalizations, and discovered that he never had asthma.

DISCUSSION

Neuromuscular tracheostomy IPPV users with less than 40 minutes of VFBT have a tendency to develop early and sustained hypocapnia irrespective of cuff status (J.R.B., unpublished data, 1994).[4] When VAIs are switched from tracheostomy to noninvasive IPPV methods they can better initiate and control air delivery and vary insufflation volumes by air stacking and by controlling insufflation leakage.[9] Lung insufflations also vary considerably during sleep as variable insufflation leak occurs around the interface and out of the nose or mouth.[9-11,13]

Although during sleep central chemotaxic centers are less sensitive to $PaCO_2$ and hypoxia the successful use of noninvasive IPPV is dependent on central nervous system (CNS)

mediated reflex muscular activity to avoid excessive insufflation leakage and hazardous hypoxia and hypoventilation. During nasal IPPV the oropharynx is intermittently reflexively sealed off to maintain adequate alveolar ventilation,[13] whereas during mouthpiece IPPV the nasopharynx is intermittently sealed off by the soft palate. Thus, for noninvasive IPPV users, inspiratory volumes vary around the clock and chemotaxic ventilatory drive plays a key role in maintaining adequate alveolar ventilation. Negative pressure body ventilator use, on the other hand, does not seem to be dependent on CNS-mediated activity for effective use and can be associated with obstructive sleep apneas, which may cause hypoxia and chronic alveolar hypoventilation.[18]

Both hypercapnia and hypocapnia can cause disturbing symptoms,[13] and when severe, lead to morbidity and mortality. Hypocapnia, which occurs at high altitudes, seems to be of minimal significance in habituated healthy individuals. The consequences of hypocapnia to wheelchair-confined VAIs may be very different. Hypocapnia necessitates the development of compensatory metabolic changes to avoid chronic respiratory alkalosis. A study by Schaefer on acclimatization to $PaCO_2$ affirmed previous investigations into calcium and phosphorus metabolism indicating that bone plays a major role in the storage of CO_2.[19] Freeman and Fenn noted an inverse correlation between CO_2 tensions and calcium excretion in hyperventilating rats.[20] Urinary bicarbonate excretion derived primarily from bone CO_2 stores is linked with concurrent elevation in calcium excretion in the urine. This provides a theoretical relationship between hypocapnia and accelerated osteoporosis. In a wheelchair-confined population this might increase the risk of pathological fractures. It deserves further research.

Chronic hypocapnia can also lead to an iatrogenic increase in ventilator dependence. Just as progressively severe metabolically compensated hypercapnia increases the dyspnea threshold associated with hypoxia and many severely hypercapnic individuals survive without ventilator use at the expense of severe symptomatology and increased risk of morbidity and mortality,[13] as hypocapnia worsens for VAIs, the maximum tolerated VFBT decreases and dyspnea occurs with small increases in $PaCO_2$. Decreases in blood and chemotaxic center bicarbonate levels may contribute to the diminution in VFBT. This is apparently one reason why we have observed that once individuals are switched from the use of noninvasive methods of ventilatory support to tracheostomy IPPV, often they soon require support 24-hours-a-day, whereas VFBT is increased by transition from tracheostomy IPPV to noninvasive IPPV. The loss in VFBT by transition to tracheostomy IPPV is counter to the goals of pulmonary rehabilitation, which include increasing independence and quality of life while decreasing morbidity and cost.

Noninvasive IPPV users only take assisted breaths when they feel the need. This optimizes the use of their respiratory muscles and relieves anxiety. Long-term tracheostomy IPPV, on the other hand, is usually prescribed at levels that eliminate or minimize autonomous breathing activity, especially for VAIs with little VC or VFBT. Thus, respiratory muscle deconditioning may be another explanation for the decrease in VFBT observed in tracheostomy IPPV users. Four of the five VAIs reported in table 3 developed significantly greater VFBT after transition to noninvasive aids. In three cases this was without significant changes in pulmonary volumes. In two cases it was achieved by mastering GPB, a technique that these VAIs were unable to use while they had indwelling tracheostomy tubes.[10]

The appearance of hypocapnia can also hamper the ability to learn the compensatory inspiratory assist technique of GPB by decreasing the VFBT that facilitates practicing the technique. The presence of a tracheostomy tube can also allow too much air leakage around the tube during GPB to permit effective use.[12]

CONCLUSION

This study shows that VAIs switched from tracheostomy IPPV to the use of noninvasive IPPV methods can experience normalization in alveolar ventilation and an increase in VFBT. The volitional control of insufflation volumes and the nocturnal dependence on normal chemotaxic ventilatory drive seem to protect noninvasive IPPV users from chronic hyperventilation. Awareness of the frequency of chronic hypocapnia in tracheostomy IPPV users, and its possible reversal by transition to noninvasive IPPV, adds to the list of reasons that include decreased morbidity and cost,[21] patient preference,[22] and improved quality of life,[23-25] for avoiding or eliminating tracheostomy in favor of the use of noninvasive respiratory muscle aids in appropriate VAIs.[14]

References

1. Jones NJ, Robertson DG, Kane JW. Difference between end-tidal and arterial PCO_2 in exercise. J Appl Physiol 1979;47:954-60.
2. Banzett RN, Lansing RW, Reid MB, Adams L, Brown R. Air hunger arising from increased PCO_2 in mechanically ventilated quadriplegics. Respir Physiol 1989;76:53-68.
3. Manning HL, Shea SA, Schwartzstein RM, Lansing RW, Brown R, Banzett RB. Reduced tidal volume increases air hunger at fixed pCO_2 in ventilated quadriplegics. Respir Physiol 1992;90:9-30.
4. Patterson JL, Mullinax PF, Bain T, Kreuger JJ, Richardson DW. Carbon dioxide-induced dyspnea in a patient with respiratory muscle paralysis. Am J Med 1962;32:811-6.
5. Wright GW, Branscomb BV. The origin of the sensations of dyspnea. Trans Am Clin Climatatol Assoc 1954;66:116-25.
6. Fowler WS. Breaking point of breath holding. J Appl Physiol 1954;6:539-45.
7. Opie LH, Smith AC, Spalding JM. Conscious appreciation of the effects produced by independent changes of ventilation volume and of end-tidal pCO_2 in paralyzed patients. J Physiol 1959;149:494-9.
8. Rahn H, Bahnson HT, Muxworthy JF, Hagen JM. Adaptation to high altitude: changes in breath-holding time. J Appl Physiol 1953;6:154-7.
9. Bach JR. Alternative methods of ventilatory support for the patient with ventilatory failure due to spinal cord injury. J Am Paraplegia Soc 1991;14:158-74.
10. Bach JR. New approaches in the rehabilitation of the traumatic high level quadriplegic. Am J Phys Rehabil 1991;70:13-9.
11. Bach JR, O'Brien J, Krotenberg R, Alba A. Management of end-stage respiratory failure in Duchenne muscular dystrophy. Muscle Nerve 1987;10:177-82.
12. Bach JR, Alba AS, Bodofsky E, Curran FJ, Schultheiss M. Glossopharyngeal breathing and non-invasive aids in the management of post-polio respiratory insufficiency. Birth Defects 1987;23:99-113.
13. Bach JR, Alba AS. Management of chronic alveolar hypoventilation by nasal ventilation. Chest 1990;97:52-7.
14. Bach JR, Beltrame F. Alternative methods of ventilatory support. In: Rothkopf MM, Askanazi J, editors. Intensive homecare. Baltimore: Williams & Wilkins, 1992:173-97.

15. Bach JR. Mechanical insufflation-exsufflation: comparison of peak expiratory flows with manually assisted and unassisted coughing techniques. Chest 1993;104:1553-62.
16. Bach JR. Pulmonary rehabilitation considerations for Duchenne muscular dystrophy: the prolongation of life by respiratory muscle aids. Crit Rev Phys Rehabil Med 1992;3:239-69.
17. Kirshblum SC, Bach JR. Walker modification for ventilator assisted individuals. Am J Phys Med Rehabil 1992;71:304-6.
18. Bach JR, Penek J. Obstructive sleep apnea complicating negative pressure ventilatory support in patients with chronic paralytic restrictive ventilatory dysfunction. Chest 1991;99:1386-93.
19. Schaefer KE, Hastings BJ, Carey CR, Nichols G. Respiratory acclimatization to carbon dioxide. J Appl Physiol 1963;18:1071-8.
20. Freeman FH, Fenn WO. Changes in carbon dioxide stores in rats due to atmospheres low in oxygen or high in carbon dioxide. Am J Physiol 1953;174:422-30.
21. Bach JR, Intintola P, Alba AS, Holland IE. The ventilator-assisted individual cost analysis vs. rehabilitation and in-home management. Chest 1992;101:26-30.
22. Bach JR. A comparison of long-term ventilatory support alternatives from the perspective of the patient and care giver. Chest 1993;104:1702-6.
23. Bach JR, Barnett V. Ethical considerations in the management of individuals with severe neuromuscular disorders. 1994;73:134-40.
24. Bach JR, Campagnolo DI, Hoeman S. Life satisfaction of individuals with Duchenne muscular dystrophy using long-term mechanical ventilatory support. Am J Phys Med Rehabil 1991;70:129-35.
25. Bach JR, Campagnola D. Psychosocial adjustment of post-poliomyelitis ventilator assisted individuals. Arch Phys Med Rehabil 1992;73:934-9.

Suppliers

a. Lipseal, Puritan-Bennett, Incorporated, 4865 Sterling Drive, Boulder, CO 80301.
b. LB3 capnograph, Sensor Medics Corporation, 22705 Savi Ranch Parkway, Yorba Linda, CA 92687.
c. Microspan 8090 capnograph, Biochem International, West 238 North 1650 Rockwood Drive, Waukesha, WI 53188-1199.