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Sleep and Nocturnal Mouthpiece IPPV Efficiency in Postpoliomyelitis Ventilator Users*

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Study objective: **Intermittent positive pressure ventilation (IPPV) can be delivered via various oral, nasal, or oronasal interfaces as an alternative to tracheostomy for up to 24 h of ventilatory support. Nocturnal nasal IPPV is often associated with frequent transient but at times severe oxyhemoglobin desaturations ($dSaO_2s$) and sleep fragmentation. The purpose of this study was to determine if nocturnal mouthpiece IPPV is also associated with $dSaO_2s$ and sleep disruption.**

Design: **Twenty-seven postpolio ventilator-assisted individuals (VAIs) using mouthpiece IPPV with little or no ventilator-free breathing time (VFBT) underwent nocturnal oxyhemoglobin saturation (SaO_2) monitoring. In addition, 15 underwent nocturnal capnography and 13 underwent polysomnography.**

Results: **Mean nocturnal SaO_2 was normal in 22 of 27 and maximum end-tidal P_{CO_2} was normal in 12 of 15 VAIs. Use of lipseal retention for nocturnal mouthpiece IPPV significantly improved blood gas values during sleep. The polysomnography results demonstrated relatively normal sleep efficiency.**

Conclusions: **Nocturnal mouthpiece IPPV is most effective with lipseal retention. It can provide normal alveolar ventilation and SaO_2 during sleep for VAIs with little or no measurable vital capacity or VFBT. Because transient $dSaO_2s$ can be eliminated with lipseal retention, it may disrupt sleep less than nasal IPPV.**

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CAH=chronic alveolar hypoventilation; $dSaO_2s$=oxyhemoglobin desaturations; IPPV=intermittent positive pressure ventilation; SaO_2 =oxyhemoglobin saturation; VAI=ventilator-assisted individual; VC=vital capacity; VFBT=ventilator- free breathing time

Key words: kyphoscoliosis; mechanical ventilation; polysomnography; respiratory paralysis; sleep

Intermittent positive pressure ventilation (IPPV) delivered via a nasal interface has been described as a method for assisting or supporting inspiratory muscle function and alveolar ventilation for individuals with acute or chronic ventilatory failure of various causes, including prelung transplantation cystic fibrosis,[1] chronic obstructive pulmonary disorders,[2] severe kyphoscoliosis,[3] and various neuromuscular diseases.[4,5] Although mouthpiece IPPV has not had wide application thus far, it has been used for more severely affected individuals, for far longer periods of time, and is ideal for 24-h support.[6] Both techniques can also alleviate symptoms of chronic alveolar hypoventilation (CAH), ameliorate or normalize daytime blood gas values, and improve mean nocturnal oxyhemoglobin saturation (SaO_2).[3,5-8]

Although improved, however, mean nocturnal SaO_2 has not been found to be normal in nasal IPPV users.[3,5,9] A recent study of nocturnal nasal IPPV users with CAH associated with kyphoscoliosis revealed that ventilator insufflation leakage out of the mouth occurred to varying degrees throughout sleep. The leakage was associated with oxyhemoglobin desaturations ($dSaO_2s$) every 6 to 20 min, arousals, severely fragmented sleep, and mean SaO_2 less than 95%.[9] The purpose of this study was to determine if nocturnal mouthpiece IPPV, with or without lipseal retention, is also associated with less than normal SaO_2 and sleep fragmentation and to better understand how insufflation air leakage out of the nose might be corrected or compensated to normalize alveolar ventilation.

Methods

A population of 102 postpoliomyelitis ventilator-assisted individuals (VAIs) using mouthpiece IPPV have been treated by the authors. Forty-three have been VAIs since onset of acute poliomyelitis and 59 have late-onset chronic ventilatory insufficiency. All of the former group were initially treated with body ventilators and then switched to mouthpiece IPPV when symptoms developed because the former were no longer effective. Of the latter group, 13 were initially treated with tracheostomy IPPV and 46 with some combination of body ventilator use and noninvasive IPPV. Thirty-two of the 102 VAIs who had at some time undergone tracheostomy for IPPV were switched to noninvasive IPPV. Eleven VAIs who had been using mouthpiece IPPV were ultimately switched to tracheostomy IPPV. In one of the 11 cases, this was due to inadequate ventilatory assistance. This VAI required insufflation pressures exceeding 45 cm H_2O because of intrinsic pulmonary disease and poor pulmonary compliance. The transitions to tracheostomy were either performed electively due to the prompting of other physicians or performed during acute respiratory illnesses to facilitate airway secretion elimination by tracheal suctioning. Only 1 of these 11 VAIs had access to

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mechanical insufflation-exsufflation.[10]

Seventy of the VAIs had been using mouthpiece IPPV overnight for a mean of 14.9 years and up to 35 years. Forty-one of these, including all of the VAIs switched from tracheostomy to mouthpiece IPPV, had less than 10 min of VFBT. All 102 VAIs used mouthpiece IPPV for daytime ventilatory support, although for 10 VAIs the intermittent abdominal pressure ventilator was also a principal method of daytime support. Thirty-five of the 102 VAIs were dead at the time of this study. The reader is referred to reference 6 for discussion of morbidity and mortality issues. Of the remaining 67 VAIs, 27 lived within 2 h commuting distance of our center and agreed to be studied. These 27 VAIs had a mean age of 56 ± 8.1 years and had been using noninvasive ventilatory support methods for 25.3 ± 13.1 years.

Nocturnal oximetry studies were obtained from them. All studies included at least 7 h of sleep monitoring. Fifteen of these 27 were ventilator assisted since having had acute poliomyelitis. At the time of the study, 20 of the 27 required ventilatory support 24 h/d and 23 of the 27 had less than 10 min of VFBT when supine. Fourteen used pressure-triggered ventilators with settings from 20 cm H_2O to 42 cm H_2O and 13 used volume-triggered ventilators with tidal volumes from 1,000 mL to 1,900 mL. Rates were set from 12 to 18/min.

Of these 27 VAIs, 13 (subjects 1 through 13 in Table 1) agreed to undergo inpatient polysomnography. These 13 had a mean age of 54.8 ± 6.8 years and had been using ventilatory support for 23.3 ± 12.4 years. Five were ventilator supported since onset of polio. The other eight VAIs were initially dependent on aid, then weaned, and free of support for 27.7 ± 9.1 years before requiring aid once again. All 13 VAIs used mouthpiece IPPV for daytime aid, although 2 of the 13 alternated mouthpiece IPPV with intermittent abdominal pressure ventilator use. All but two VAIs required 24-h ventilatory support and all but one had no VFBT when in the supine position.

Each of the 13 VAIs was admitted to a sleep laboratory for nocturnal polysomnography that was performed under direct observation (SensorMedics polysomnograph system 1, SensorMedics, Yorba Linda, Calif). The following were monitored: electroencephalography, electro-oculography, genioglossus electromyography, abdominal impedance plethysmography, nasal air flow (Thermistor to Monitor Respiratory Airflow, D. M. Davis Inc, New York), SaO_2 (Biox III Ear Lobe Oximeter, Ohmeda, Madison, Wis), and end-tidal CO_2 with pick up at the expiratory valve (LB3 Continuous CO_2 Monitor, SensorMedics Corp, Yorba Linda, Calif). The observation of expiratory air leakage (by nasal thermistor and observation of air leak at the mouth) is only

Table 1—*Nocturnal Noninvasive Blood Gas Analyses of Mouthpiece IPPV Users

No.	Mean SaO_2	Low SaO_2	Mean WH†	% Time of SaO_2 <90%	<85%	<80%	<70%	$dSaO_2$ Index‡	$EtCO_2$ §
1‖	98	94	98	0	0	0	0	0	37
2	97	86	96	0	0	0	0	0	31
3	99	96	98	0	0	0	0	0	33
4¶	96	58	94	6	3	1	0	13	31
5	97	92	97	0	0	0	0	0	40
6	95	73	93	6	2	0.5	0	5.5	35
7**	82	60	80	80	54	38	6	42	36
8††	89	57	85	43	23	11	2	25	51
9‡‡	97	93	96	0	0	0	0	0	36
10§§	95	70	93	8	3	1	0	1.1	27
11	97	86	95	1	0	0	0	0	33
12**	94	85	93	8	0	0	0	7	44
13	98	92	97	0	0	0	0	0	51
14	99	88	97	0	0	0	0	0	NP‖‖
15	98	84	95	0	0	0	0	0	NP
16	97	94	97	0	0	0	0	0	NP
17	96	90	95	0	0	0	0	0	NP
18	95	85	94	5	0	0	0	0	NP
19	98	87	96	0	0	0	0	0	NP
20	96	81	94	2	0	0	0	0	NP
21	95	73	94	5	5	1	0	6	39
22	94	66	93	10	6	2	0	11	NP
23	93	83	92	7	0	0	0	0	NP
24	98	97	96	0	0	0	0	0	NP
25	95	76	94	3	0	0	0	0	NP
26	95	73	94	5	7	2	0	0	54
27	95	86	94	1	0	0	0	1	NP

*All used nocturnal mouthpiece IPPV with lipseal retention except where indicated.
†The lowest 1-hour mean SaO_2 during the study.
‡The number of $dSaO_2$s greater than 3% per hour of sleep.
§Maximum end-tidal CO_2 in mm Hg recorded during the study.
‖Nose sealed with cotton pledgets kept in place by tape covering.
¶Used low-flow oxygen therapy.
**Used bite plate without lipseal.
††Used mouthpiece with a single strap lipseal.
‡‡Used bite plate with lipseal.
§§Used mask covering mouth and nose.
‖‖NP=not performed.

significant in that total leakage through the nose would not leave any air at the expiratory valve for end-tidal CO_2 analysis. Although oral leakage was not specifically measured, the polysomnographer observed it in those not using a lipseal and felt for it in the others. Analysis for air leakage was limited to five VAIs because it required tedious manual analysis.

During the inpatient studies, nine VAIs used fixed-rate pressure-triggered ventilators (Maxivent, Puritan-Bennett, Boulder, Colo; RBL, Lifecare, Lafayette, Colo) at a rate of 12 to 17/min at 20 to 42 cm H_2O pressure. Four VAIs used volume-triggered portable ventilators in assist-control mode (PLV-100, Lifecare International, Lafayette, Colo, and M-25B, Puritan-Bennett, Boulder, Colo) at rates of 16 to 20/min and 1,300 to 1,900 mL delivered volumes.

Results

The results of the noninvasive blood gas monitoring are listed in Table 1. VAI 1 used mouthpiece IPPV with a lipseal (lipseal IPPV) for 10 years before awakening on several occasions with tachycardia. He was described as "snoring" through his nose before awakening. He subsequently sealed his nose every night by placing cotton pledgets into his nostrils and covering them with strips of tape (Micropore, 3M, Minneapolis). He continues to use this airtight system and has had no further episodes of tachycardia or snoring. His polysomnography showed 19 "central" apneas, 3.1/h, with the longest duration of 25 s. These were not associated with $dSaO_2$s. The "apneas" were classified as central in origin since there was no detected movement of the chest or abdomen. Since this VAI was on a regimen of fixed rate ventilatory support, the "apneas" must have been from transient tongue or glottic obstruction to air flow. This VAI was also studied using nocturnal nasal IPPV. He maintained a mean SaO_2 of 97%, low of 91%, and a mean of three $dSaO_2$s of greater than 3%/h.

VAI 4 used low-flow oxygen in addition to ventilatory support because of persistent $dSaO_2$ in the presence of normal end-tidal P_{CO_2} both when awake and asleep. For VAI 6, the lipseal was padded at the four corners (with Otoform K R, Dreve, Unna, Germany) to decrease the air leakage observed during previous studies. VAI 8, with a thick mustache, had air escape from the upper border of the lipseal that repeatedly slipped down below the mustache. He was dyspneic and symptomatic for CAH. He used increasing periods of mouthpiece IPPV during daytime hours and was converted to nasal IPPV overnight on which his mean SaO_2 was 97%, low 92%, worst hour mean 96% with 0.3 $dSaO_2$s per hour and a maximum end-tidal P_{CO_2} of 46 cm H_2O.

VAI 7 had no VFBT when supine but was able to breathe autonomously with hypercapnia when erect. Sleeping while sitting and using supplemental oxygen failed to improve SaO_2 and hypercapnia worsened. The addition of low-flow oxygen to lipseal IPPV improved nocturnal SaO_2 from a mean of 82% to 92% but there were a mean of 40 $dSaO_2$s per hour and a maximum end-tidal P_{CO_2} of 48 mm Hg. He used mouthpiece IPPV with low-flow oxygen for 4 years but in 1992 switched to a closed, custom lipseal system using a strapless interface with bite-plate retention and an outer shell covering the nose and mouth. Supplemental oxygen therapy was discontinued and nocturnal SaO_2 values improved to a mean of 94% with five $dSaO_2$s per hour. He also reported less fatigue and felt better rested in the morning.

VAI 27 was converted from 24-h rocking bed ventilator use to mouthpiece IPPV in 1980. In 1988,

Table 2—*Sleep Stage Time During Mouthpiece IPPV*

No	Awake*	STA1†	STA2‡	STA34§	REM‖
1	75	5	14	6	0
2	7	12	40	22	19
3	18	30	8	17	26
4	31	17	0	40	5
5	11	26	26	12	25
6	28	3	19	20	28
7	20	35	1	27	11
8	18	11	28	16	19
9	14	49	14	0	17
10	5	4	35	0	10.3
11	13	32	5	42	0
12	22	24	4	20	17
13	24	18	21	14	19
Mean	20.9 ± 18.7	22.6 ± 16.5	15.9 ± 13.0	20.7 ± 14.3	13.1 ± 10.1

*Percentage of sleep period time (SPT) awake, normal (age 40 to 70 years) 0-22%.
†Stage 1 sleep as a percentage of SPT, normal=3-14%.
‡stage 2 sleep as a percentage of SPT, normal=40-72%.
§stage 3, 4 sleep as a percentage of SPT, normal=0-24%.
‖REM sleep as a percentage of SPT, normal=17-31%.[15]

Table 3—*Sleep Efficiency During Mouthpiece IPPV*

No.	Latency*	SPT†	Awake, min‡	TST§	Efficiency‖
1	19	315	237	77	21
2	32	275	20	415	95
3	35	425	76	455	98
4	29	335	103	265	72
5	39	417	47	409	90
6	20	249	71	198	73
7	24	389	78	377	83
8	29	269	48	350	89
9	37	438	63	412	87
10	20	419	21	439	95
11	10	357	44	316	86
12	18	225	50	221	82
13	18	401	95	328	74
Mean	27.3 ± 9.5	349.0 ± 64.2	69.9 ± 57.9	330.8 ± 108.3	81.0 ± 19.9

*Sleep latency time which is from "lights out" to the occurrence of the first sleep stage in minutes; normal is 0 to 22 min.
†Sleep period time: time in minutes spent in bed from the onset of sleep until the morning arousal.
‡Minutes awake during the sleep period time.
§Total sleep time: time in bed minus awake time.
‖Percentage sleep efficiency (index): the total sleep time divided by the time in bed, normal (age 40 to 70 years=78-100%).

Table 4—*Percentages of Time of Nasal Air Leakage*

Patient No.	Settings*	Leakage Time Percent†					Sleep Efficiency, %	Leak as Percent of	
		Awake	1	2	3&4	REM		SPT	TST
During insufflation									
3	28 cm H_2O	100	34	100	29	14	98	45	33
4	40 cm H_2O	100	37	0‡	38	0	72	56	35
9	22 cm H_2O	10	57	47	0‡	85	87	54	61
12	22 cm H_2O	3	36	16	5	3	82	13	17
13	1,800 mL	50	99	23	90	74	87	64	69

Patient No.	Leakage Time Percent†						
	Awake	1	2	3&4	REM	SPT	TST
During expiration							
3	98	22	10	8	11	34	15
4	14	2	0‡	38	0‡	21	25
9	2	50	0‡	40	100	51	60
12	0	3	0	2	3	2	3
13	46	87	21	87	74	60	64

*Subjects 3, 4, 9, and 12 used pressure-triggered ventilators and subject 13 used a volume-triggered ventilator.
†Leakage time as a percentage of the time spent in sleep stages: awake, stages 1, 2, 3&4, REM.
‡No sleep in this stage.

using nocturnal mouthpiece IPPV without a lipseal, his SaO_2 mean was 89%, the low was 72%, the worst hour mean was 86%, and it was below 90% for 59% of the night. He was converted to lipseal IPPV and the subsequent sleep oximetry results over a two-night period are averaged in Table 1.

The percentage of time the 13 VAIs who underwent polysomnography spent in the stages of sleep and their sleep efficiencies are indicated in Tables 2 and 3. The percentage of time that 5 of the 13 mouthpiece IPPV users leaked air out of their nostrils during the ventilator inspiratory and expiratory cycles is noted in Table 4. The three VAIs who used mouthpiece IPPV without a lipseal or with a lipseal held by only one elastic strap (Table 1), at the nadirs of their $dSaO_2$s and without any apparent cognizance of having been aroused, were consistently observed to grip their mouthpieces with the teeth and oral musculature to eliminate air leakage and thereby increase alveolar ventilation and normalize SaO_2.

Discussion

The SaO_2 monitoring is accurate at any point in time to within 2% of actual SaO_2 for values of 70% or greater.[11] End-tidal P_{CO_2} is an accurate gauge of alveolar ventilation for VAIs without severe pulmonary diffusion deficits or cardiovascular failure.[12] It, thus, should have been accurate in our population provided that an adequate sampling of end-alveolar expiratory flow could be analyzed. The fact that expiratory passage of air through the nose occurred less than 65% of the time and during an average of only 33 ± 27% of the total sleep time for the five VAIs for whom it was measured implies that the maximum end-tidal P_{CO_2} measurements could only have been higher than what was actually observed.

Despite the fact that only 3 of the 27 mouthpiece IPPV users studied had sufficient vital capacity (VC) to support VFBT for over 10 min when supine and only 1 used supplemental oxygen, all slept supine, 22 (82%) of the nocturnal oximetry studies yielded normal mean SaO_2, and 12 of 15 had maximum end-tidal P_{CO_2} levels under 45 mm Hg. The lowest mean nocturnal SaO_2 for anyone using nocturnal mouthpiece IPPV with the mouthpiece properly retained by a two-strap lipseal was 92%. The VAIs using mouthpieces or bite-plates without firm lipseal retention had a "sawtooth" pattern of $dSaO_2$ and mean SaO_2 under 95%. This pattern is similar to that which has been reported during nocturnal nasal IPPV use.[3,5] These VAIs and all whom we have subsequently observed asleep using mouthpiece IPPV without an adequate lipseal have demonstrated loosening of grip of the mouthpiece, without it falling from the mouth, accompanied by air leakage and $dSaO_2$. At the nadir of the $dSaO_2$, this has invariably been followed by the VAI firmly regripping the mouthpiece with his teeth and oral musculature to eliminate leakage until SaO_2 had increased to the preleakage baseline.

Both nasal and mouthpiece IPPV, without adequate oral-nasal seal to prevent insufflation leakage, are open systems. When a $dSaO_2$ caused by excessive insufflation air leakage occurs to a certain degree, the VAI usually undergoes a brief arousal, of which he is unaware, or lightening of sleep stage, and muscular activity occurs to seal off the nasal or oral leakage.

This improves alveolar ventilation and returns SaO_2 to baseline.[9] These transient 4% or greater $dSaO_2$s tend to become more severe in VAIs who are hypercapnic during daytime as well as overnight. Of the 13 mouthpiece IPPV users who underwent polysomnography, only five had mean SaO_2 less than 95% for 1 h or more, SaO_2 less than 90% for 6% or more of the recording time, and many transient $dSaO_2$s. Three of these five used mouthpiece IPPV without a lipseal (cases 7 and 12) or with a lipseal with inadequate retention (case 8). All three were also symptomatic with hypoventilation during daytime hours but did not use daytime ventilatory assistance. For VAIs 8 and 12, this was in large part because they were functional ambulators. VAI 7 subsequently supplemented nocturnal nasal IPPV with daytime mouthpiece IPPV and now uses IPPV 24 h/d. The normalization of central nervous system chemotaxic sensitivity in this VAI appears to have resulted in earlier central nervous system-mediated recruitment of oropharyngeal muscular activity to diminish insufflation leakage during sleep and restore normal alveolar ventilation and SaO_2 around-the-clock. VAI 8 now also uses daytime mouthpiece IPPV periodically and VAI 12 has switched to using nocturnal strapless oral-nasal interface IPPV.[13,14]

The five VAIs for whom leakage time was quantitated had inspiratory cycle (insufflation) leakage during a mean of $43 \pm 21\%$ of total sleep time. Since ventilator settings, especially those of VAIs using volume-triggered ventilators, exceeded that which would have been necessary to achieve adequate alveolar ventilation in a closed system, it was not surprising that insufflation leakage occurred from 3% to 100% of the time the VAIs were awake to avoid hyperventilation. It is also understandable that the VAIs using the higher ventilator pressures and volumes tended to have the longest leakage periods. The pressure-triggered ventilators compensate for some insufflation leakage and higher delivered volumes permit the VAI the convenience of taking fewer "leakless" mouthpiece-assisted breaths per minute.

Insufflation leakage is stopped or markedly decreased and SaO_2 normalized during the oropharyngeal muscular activity triggered by $dSaO_2$s. This can occur during sleep whether the VAI is using mouthpiece IPPV with or without a lipseal. Firm lipseal retention with completely passive seal of the lips and mouth can change the sawtooth $dSaO_2$ pattern into a normal pattern that reflects effective compensation of nasal air leakage. Because no central nervous system triggered muscular activity is necessary to maintain adequate alveolar ventilation in this case, more restful sleep with less sleep fragmentation would be expected this way than during mouthpiece IPPV without a lipseal or during nasal IPPV. No method has been reported to be successful for compensating or preventing excessive oral air leakage during nasal IPPV except for taping the mouth closed, a procedure tolerated by few, or using a plugged lipseal.[15] Therefore, the use of lipseal IPPV is a logical alternative to nasal IPPV when oral leakage leads to significant $dSaO_2$s. We have routinely switched nasal IPPV users with acute respiratory failure due to nasal congestion precluding effective use of nasal IPPV to nocturnal lipseal IPPV.

Total arousals, sleep stage changes, and arousals with change of SaO_2 were not calculated in this study. However, percentages of sleep in the various stages were relatively normal. Further, the relative absence of the frequent transient $dSaO_2$s associated with sleep disturbances in studies of nocturnal nasal IPPV users with severe kyphoscoliosis[9] implies that sleep may be more restful for lipseal IPPV users. This deserves further study. The one VAI with no measurable VC who was studied using both lipseal and nasal IPPV had more $dSaO_2$s using the latter.

Using an essentially closed system with a lipseal and nostril plugging or switching to masks that cover both nose and mouth has been shown to normalize nocturnal SaO_2 by eliminating insufflation leakage and, thereby, any need to resort to centrally mediated mechanisms for effective alveolar ventilation.[13,16] However, covering the nostrils was found to be needed to maintain adequate alveolar ventilation in only 5 of 163 nocturnal mouthpiece IPPV users with little or no VFBT.[6]

Volume-triggered ventilator-delivered volumes should usually be greater than 800 mL, and 2,000 mL or more have been used to compensate for insufflation leakage. Although portable pressure-triggered ventilators can also automatically compensate for some insufflation leakage, cessation of all leakage would cause hyperventilation and possibly aerophagia. The latter has not been an insurmountable problem for any of our noninvasive IPPV users. Although volume-triggered ventilators have predominated since 1978, a portable pressure-triggered ventilator (Maxivent, Puritan-Bennett, Boulder, Colo) has remained on the market. Bi-PAP machines (Bi-PAP ST, Respironics, Murrysville, Pa) are more recently released pressure-triggered ventilators that can also be practical for delivering IPPV, but inspiratory pressures are limited to about 20 cm H_2O. This is inadequate for many patients.

For the 13 nocturnal mouthpiece IPPV users undergoing inpatient polysomnography, the time periods in the deeper sleep stages tended to be normal as a percentage of sleep period time.[17] Ten had normal and three had increased duration stage 3 and 4 sleep. Eight had normal and five had decreased rapid eye movement (REM) sleep duration as a percentage of

sleep period time. Although awake time was increased for eight of the 11 subjects, this is not surprising considering that they were admitted for a one-night, inpatient study with many leads.

Along with evaluation for symptoms and signs of CAH and measurement of VC with the patient supine,[5] nocturnal oximetry monitoring should be used to establish the need for and document improvement in nocturnal alveolar ventilation when initiating any open system of ventilatory support,[5,6,18] when changing between methods,[19] and periodically thereafter. We find polysomnography helpful only when symptoms are not accompanied by severe restriction in pulmonary volumes (generally VC less than 40% of predicted normal), subnormal mean nocturnal SaO_2, or nocturnal hypercapnia.

In conclusion, the nocturnal use of mouthpiece IPPV should be with firm lipseal retention, otherwise insufflation leakage during sleep can cause subnormal SaO_2 and probably sleep fragmentation similar to that seen in nocturnal nasal IPPV users. During periods of excessive insufflation leakage, central nervous system mediated muscular activity occurs at the SaO_2 nadirs to eliminate leakage and renormalize SaO_2. For those who continue to have periods of $dSaO_2$ despite firm lipseal retention, the use of nasal pledgets or a strapless oral-nasal interface for IPPV can normalize alveolar ventilation and sleep SaO_2 without resorting to endotracheal intubation.[13] Thus, this study supports the safety of noninvasive IPPV systems as alternatives to tracheostomy IPPV for up to 24 h of ventilatory support for selected populations.

References

1 Piper AJ, Parker S, Torzillo PJ, Sullivan CE, Bye PTP. Nocturnal nasal IPPV stabilizes patients with cystic fibrosis and hypercapnic respiratory failure. Chest 1992; 102:846-50

2 Marino W. Intermittent volume cycled mechanical ventilation via nasal mask in patients with respiratory failure due to COPD. Chest 1991; 99:681-84

3 Leger P, Jennequin J, Gerard M, Robert D. Home positive pressure ventilation via nasal mask for patients with neuromuscular weakness or restrictive lung or chest-wall disease. Respir Care 1989; 34:73-7

4 Barois A, Estournet-Mathiaud B. Nasal ventilation in congenital myopathies and spinal muscular atrophies. Eur Respir Rev 1993; 3:275-78

5 Bach JR, Alba AS. Management of chronic alveolar hypoventilation by nasal ventilation. Chest 1990; 97:52-7

6 Bach JR, Alba AS, Saporito LR. Intermittent positive pressure ventilation via the mouth as an alternative to tracheostomy for 257 ventilator users. Chest 1993; 103:174-82

7 Ellis ER, Bye PTB, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with neuromuscular disease. Am Rev Respir Dis 1987; 136:188-91

8 Bach JR, Alba AS. Pulmonary dysfunction and sleep disordered breathing as post-polio sequelae: evaluation and management. Orthopedics 1991; 14:1329-37

9 Bach JR, Robert D, Leger P, Langevin B. Nasal IPPV and sleep fragmentation in individuals with severe kyphoscoliosis. Chest (in press)

10 Bach JR. Mechanical insufflation-exsufflation: comparison of peak expiratory flows with manually assisted and unassisted coughing techniques. Chest 1993; 104:1553-62

11 Welch JP, DeCesare R, Hess D. Pulse oximetry: instrumentation and clinical applications. Respir Care 1990; 35:584-97

12 Kacmarek RM. Noninvasive monitoring in respiratory care: conference summary. Respir Care 1990; 35:740-46

13 Bach JR, McDermott I. Strapless oral-nasal interfaces for positive pressure ventilation. Arch Phys Med Rehabil 1990; 71:908-11

14 McDermott I, Bach JR, Parker C, Sortor S. Custom-fabricated interfaces for intermittent postive pressure ventilation. Int J Prosthodont 1989; 2:224-33

15 Bach JR, Sortor S, Saporito LR. Interfaces for non-invasive intermittent positive pressure ventilatory support in North America. Eur Respir Rev 1993; 3:254-59

16 Bach JR. New approaches in the rehabilitation of the traumatic high level quadriplegic. Am J Phys Med Rehabil 1991; 70:13-20

17 Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Los Angeles: Brain Information Service/Brain Research Institute University of California, 1968: 1-12

18 Bach JR, Alba AS. Tracheostomy ventilation: a study of efficacy with deflated cuffs and cuffless tubes. Chest 1990; 97:679-83

19 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

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