Mouth Intermittent Positive Pressure Ventilation in the Management of Postpolio Respiratory Insufficiency*

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The use of mouth intermittent positive pressure ventilation alone or in combination with other noninvasive respiratory techniques as an alternative to tracheostomy in the home management of respirator-dependent postpolio persons was studied in 75 patients. The onset of polio was at an average age of 15 years. At that time, all were dependent on some form of respiratory assistance, most frequently, the iron lung. Fifty-nine percent of them remained respirator-dependent from the onset. Forty-one percent became respirator-dependent at an average of 18 years after onset of polio. Overall, they lost an average of 1.9 percent of vital capacity per year. All used mouth intermittent positive pressure ventilation as their predominant mode of respiratory assistance for an average of 14.5 years. Four of them who had no measurable vital capacity used only mouth intermittent positive pressure ventilation 24 hours per day. Of the 66 who had no significant tolerance off 24 hours per day respiratory assistance, only six had tracheostomies. Despite severe physical disability and dependence on artificial ventilation, the majority of these persons have married, have been gainfully employed, and lead useful lives in society.

Polioymelitis, which is caused by three antigenically distinct types of enteroviruses, is a disease with a wide spectrum of clinical severity. As many as 99 percent of afflicted persons may be asymptomatic or suffer only minimal symptomatology. However, for the small percentage of persons who develop paralytic polio, the effects can be devastating with maximal muscle weakness occurring within days following the onset of weakness.

Although the last major polio epidemic in the United States was in 1955, over 200 cases have been reported since 1969 with 21 vaccine-associated cases in 1983 alone. Many of those afflicted during the polio epidemics of the 1940s and 1950s are now seeking medical attention for late postpolio complications of which the most life-threatening is insidious progressive respiratory insufficiency.

The respiratory failure associated with polio occurs either by the destruction of anterior horn cells in the spinal cord innervating respiratory muscles or by neuronal destruction of the respiratory center in the medulla or both. Whatever the mechanism, this necessitates mechanical ventilatory assistance by means of the iron lung or tracheostomy tube with tracheostomy intermittent positive pressure ventilation (TIPPV) during the acute management of these patients. This acute stage had a 25 to 75 percent fatality rate.

Although many recover sufficient respiratory muscle control to free themselves from respiratory aid, the muscle weakness and loss of lung volume that occurs with age necessitates the reintroduction of artificial ventilation for some. This mechanical aid has usually been TIPPV despite the potentially serious complications of tracheostomy placement, poor cosmesis and patient reluctance.

Lassen et al reported 232 tracheostomized paralytic polio patients receiving TIPPV in 1956, but only 138 of them survived four years. In a more recent study of respirator-dependent postpolio persons, the largest to date, TIPPV was the predominant method of assistance offered. In that study, 23 used respiratory assistance full-time and 80 used it only overnight. Of these persons, 76 used TIPPV. Twenty-seven used the iron lung or the chest shell overnight, but as their margin of reserve diminished, the author stated that “the specter of a tracheostomy” looms in their management.

To avoid the pitfalls associated with tracheostomy use, others have employed noninvasive means of nocturnal mechanical ventilation. Garay et al reported the overnight use of the Emerson wrap negative pressure respirator by two severely hypercapnic postpolio persons for 12 and 15 years. Nocturnal use alone allowed them to maintain “acceptable” blood gas levels during the day (Pco2, 44 mm Hg, Pao2, 58 to 63 mm Hg) and reversed marked pulmonary hypertension. However, their vital capacities (VC) were 1,000 and 1,060 ml, and thus, they did not require full time respiratory assistance. Likewise, Guilleminault and Motta reported the use of night-time chest shell ventilator in

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five postpolio persons with symptomatic central and/or obstructive sleep apnea. These persons, too, had sufficient pulmonary reserve not to require full time respiratory aid.

We have studied the change in type and amount of respiratory assistance in severely involved postpolio persons with progressive decline in vital capacity. Symptomatic hypoventilation, and in particular, severe nocturnal hypercapnia indicated the need for assistance. Mouth intermittent positive pressure ventilation (MIPPV) was used as the predominant method because of its effectiveness and its advantage in obviating the need for tracheostomy tube placement. Those persons not using overnight aid but employing MIPPV only during daytime hours were not included in this study since they generally had VC greater than 1 L, and thus, were not vitally dependent on mechanical aid.

This is a study of the efficacy of MIPPV as an alternative to TIPPV in postpolio persons vitally dependent on respiratory assistance.

METHOD

Seventy-five postpolio persons on MIPPV were studied. Seventy were managed in the home, five in chronic care institutions. Eight had complete quadriplegia, while 67 had varying degrees of quadriaparesis. None had severe bulbar involvement. All were wheelchair dependent. The majority were mobile only in motorized wheelchairs. Twenty-seven had significant scoliosis. Sixty-six were ventilator dependent 24 hours a day and could tolerate only seconds of time free of assisted ventilation unless capable of effective glossohypopharyngeal breathing.

All persons respirator dependent since onset of polio (1950 to 1956) were maintained on body respirators such as the iron lung and rocking bed until the late 1950s. They were trained in the use of MIPPV for daily periods of positive pressure ventilation as early as 1958. It allowed the postpolio survivor to “sigh” by taking two to three breaths before exhaling. It also provided him with additional ventilation during upper respiratory tract infections (URIs) and periods of respiratory distress. With the increasing availability of portable positive pressure ventilators over the next five years, the use of daytime MIPPV increased. Postpolio survivors no longer needed to be recumbent to receive aid. They left their body respirators each morning in favor of MIPPV with portable ventilators mounted onto motorized wheelchairs. A gooseneck holder maintained the mouthpiece adjacent to the mouth allowing an assisted breath as the person required (Fig 1).

It was soon appreciated that during sleep, excessive loss of insufflated air with resulting poor ventilation could be prevented by holding the mouthpiece in the mouth with a lip seal (Fig 2) and by reflex contraction of the lip and jaw muscles. If needed, the cycling pressure or the tidal volume was increased on the respirator above daytime settings. It was rarely necessary to cover the nostrils to prevent leakage. As early as 1964, patients began to depend on MIPPV alone for their total ventilation 24 hours a day.

Postpolio persons with late onset respiratory insufficiency were also introduced to daytime MIPPV. As their restrictive pulmonary syndromes worsened and respiratory insufficiency progressed, the use of MIPPV gradually increased to full time. Thirty-one of these patients were ventilated adequately overnight for a decade or more with the chest shell and rocking bed. Some began to express symptoms of nocturnal hypoventilation and were found to no longer maintain normal blood gas tensions while on body respirators. These patients switched to overnight MIPPV. Others, already using MIPPV during the daytime, preferred to continue employing it at night as well.

During periods of respiratory distress associated with URIs, patients are often hospitalized. They may be managed in the iron lung with or without supplementary MIPPV while compromised. The effectiveness of ventilation is assessed by overnight capnograph (FCO₂ metabolic analyzer) monitoring correlated with blood gas determinations. Difficulty clearing secretions is managed by assisted coughing techniques. These include deep assisted breaths by mechanical aid or glossohypopharyngeal breathing (GPB) prior to a manually assisted cough, tracheal suctioning via the mouth or nose, and the use of a Cofflator. The Cofflator delivers a deep positive pressure breath via a face mask followed by a drop in pressure to −100 cm H₂O in 0.02 s to provide clearance of bronchial secretions. The GPB is an effective technique in aiding pulmonary toilet and normalizing the speech patterns of many patients. It can also provide from minutes to many hours of time free of ventilator assistance for many patients including some with vital capacities approaching 0 ml.

When overnight MIPPV is ineffective, particularly in the presence of organic lung disease, severe complicating medical conditions, or incompetent buccopharyngeal muscles, intubation or tracheostomy may be indicated.

**FIGURE 1.** Patient with a vital capacity of 240 ml operating her motorized wheelchair by tongue control and receiving MIPPV via mouthpiece held in place by a gooseneck clamp.

**FIGURE 2.** Patient with a vital capacity less than 10 ml set up for overnight MIPPV with the mouthpiece supported firmly in the mouth by the Bennett lip seal.
Table 1—Forty Three Postpolio Persons with no Tolerance off Respiratory Assistance on MIPPV Only

<table>
<thead>
<tr>
<th></th>
<th>Average</th>
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<tbody>
<tr>
<td>Age</td>
<td>47.8 yrs</td>
<td>(21-66)</td>
</tr>
<tr>
<td>Age at onset of polio</td>
<td>14.9 yrs</td>
<td>(0-44)</td>
</tr>
<tr>
<td>VC, most current</td>
<td>607 ml</td>
<td>(0-1700)</td>
</tr>
<tr>
<td>(supine)</td>
<td>17.4% pred</td>
<td></td>
</tr>
<tr>
<td>Age at death</td>
<td>41.6 yrs</td>
<td>(2-29)</td>
</tr>
<tr>
<td>following MIPPV for</td>
<td>17.9 yrs</td>
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*Includes one tracheostomized person.

RESULTS

Seventy-five respirator dependent postpolio persons were separated into three categories as a function of the amount and method of respiratory assistance. Table 1 represents those persons with no tolerance off mechanical aid who used only MIPPV 24 hours per day. Twenty-three of these persons had been dependent on aid since the onset of polio. Twenty returned to at least overnight respiratory aid an average of 18.1 years (one to 37) postpolio and subsequently progressed to full-time respirator dependence. Twenty-five of these persons had used the rocking bed, iron lung, or chest shell for an average of 14.7 years (two to 34) prior to switching to full-time MIPPV. Only four were recently tracheostomized and have used TIPPV an average of 2.5 years (1-4 years). One of these is deceased.

Table 2 lists 23 persons dependent on full-time respirator assistance using MIPPV combined with some other technique compatible with home management. Twenty-one of these patients had been totally dependent on mechanical ventilation since onset of polio. Two became ventilator-dependent, three and 25 years postpolio, respectively. Twenty of the above persons had used a combination of the iron lung, chest shell, and/or rocking bed for an average of 14.2 years (two to 30) prior to employing MIPPV. The MIPPV was used overnight and the pneumobelt was used during the day in seven persons. Daytime MIPPV was used in conjunction with overnight use of the other body respirators in the remaining 16 persons. Within the past two years, only two of these patients were tracheostomized and placed on TIPPV.

In these two groups of postpolio survivors (average VC 565 ml), only six have had tracheotomies after an average of nearly 30 years of dependence solely on noninvasive techniques of totally assisted ventilation.

Table 3 concerns postpolio survivors who became dependent on MIPPV overnight, and daytime as needed, an average of 26.6 years (17 to 35) after onset of polio. They were an average of six years younger than the patients on full time aid and had vital capacities an average of 450 ml greater. The CO2 monitoring of these individuals off aid often revealed severe nocturnal hypercapnia to levels greater than 75 mm Hg with only moderate Pco2 elevations (50 to 60 mm Hg) or even normal levels when they were awake. Oxygen saturation dropped from normal levels during waking hours to 70 to 80 percent and occasionally to 50 percent during sleep without aid.

Thirty-eight subjects were studied retrospectively over 150 patient-years to determine the rate of loss of VC. There was an average loss of 18.6 ml per year or 1.9 percent (SD, 1.2). The average annual decrement in VC in the normal population is 1 to 1.2 percent. Our results are similar to those previously cited. Possible accelerated anterior horn cell drop out in postpolio survivors, scoliosis, and increased frequency of serious respiratory infections and atelectasis are probable factors related to the increased loss of VC.

The 75 persons in this study have used MIPPV as a major part of their aid regimen for a total of 1,028 patient-years or 14.8 years per person. Seventeen persons died while using MIPPV (one death per 60.5 patient-years of MIPPV). Sixteen of 32 men have married and 19 have been gainfully employed. Twenty-six of the 42 women have married and 15 have been gainfully employed.

Case 1 (Fig 2)

This person developed complete quadriplegia and respiratory insufficiency from paralytic polio in August 1955 at age 15 years. He has no vertebral deformity and has normal bulbar muscle control, but his VC has been 0 ml since onset. He used an iron lung for several months. He was then placed on a chest shell ventilator overnight. He also used MIPPV, as well as the Pneumobelt during the daytime from 1956 to 1981. In 1981, his Pco2 rose to 45 to 50 mm Hg with a maximum tidal volume of 320 ml received by chest shell ventilator use. Because of suboptimal ventilation and the inability to "sigh" on

Table 2—Twenty Three Postpolio Persons with no Tolerance off Respiratory Assistance on MIPPV and Body Respirators

<table>
<thead>
<tr>
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<th>Average</th>
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<tbody>
<tr>
<td>Age</td>
<td>48.0 yrs</td>
<td>(18-71)</td>
</tr>
<tr>
<td>Age at onset of polio</td>
<td>17.6 yrs</td>
<td>(3-37)</td>
</tr>
<tr>
<td>VC, most current</td>
<td>485 ml</td>
<td>(0-1600)</td>
</tr>
<tr>
<td>(supine)</td>
<td>12.3% pred</td>
<td></td>
</tr>
<tr>
<td>MIPPV + chest shell</td>
<td>16.7 yrs</td>
<td>(2-30)</td>
</tr>
<tr>
<td>(nine pts),</td>
<td>+ pneumobelt (seven points),</td>
<td></td>
</tr>
<tr>
<td>+ rocking bed (six pts),</td>
<td>+ iron lung (two pts),</td>
<td></td>
</tr>
<tr>
<td>24 hours/day</td>
<td>44.3 yrs</td>
<td>(15-49)</td>
</tr>
<tr>
<td>Age at death (seven persons)</td>
<td>14.6 yrs</td>
<td></td>
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<tr>
<td>following MIPPV for</td>
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Table 3—Nine Postpolio Persons on MIPPV Overnight Plus Daytime prn

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<th>Average</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age</td>
<td>42.3 yrs</td>
<td>(31-54)</td>
</tr>
<tr>
<td>Age at onset of polio</td>
<td>8.3 yrs</td>
<td>(0-16)</td>
</tr>
<tr>
<td>VC, most current</td>
<td>1034 ml</td>
<td>(690-1720)</td>
</tr>
<tr>
<td>(supine)</td>
<td>29.9% pred</td>
<td></td>
</tr>
<tr>
<td>MIPPV, overnight plus daytime prn</td>
<td>7.7 yrs</td>
<td>(2-14)</td>
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this device, he switched to overnight MIPPV which he continues to use. His tidal volume on MIPPV with the Bennett lipguard is 600 ml and his Pco2 from 32 to 38 mm Hg with a respirator cycling pressure at 24 cm H2O. His O2 saturation is 95 percent or better during continuous monitoring. He receives regular sighs of 1.85 L with the sigh pressure adjusted to 40 cm H2O.

Although not required, if MIPPV 24 hours a day, he usually uses it during the daytime only while transferring into his pneumobelt. He continues to receive tidal volumes of 380 to 400 ml with pneumobelt use. This is sufficient to maintain normal blood gas levels during the daytime.

The GPB allows him four hours or more of time free from his respiratory assistance. Recently monitored on GPB alone, he demonstrated O2 saturation greater than 95 percent and Pco2 less than 42 mm Hg for the full four hours of the test while carrying on normal conversation. His maximum GPB assisted breath is 1.6 L.

**Case 2**

This person developed paralytic polio at age nine years in 1934. It resulted in quadriparesis with severe involvement of the chest wall and the right upper extremity. He required iron lung assistance for several months but was effectively weaned from aid. Severe kyphoscoliosis, for which he eventually underwent two spinal fusion procedures, began at age 11. In 1970, with a four-year history of shortness of breath, nightmares, lightheadedness, and cor pulmonale, he was tracheostomized and received TIPPV for several months. His VC was 1,600 ml. The tracheostomy site was allowed to close, but he was tracheostomized and received TIPPV on two other occasions, in 1971 and then in 1972 when he suffered respiratory arrest during sleep. The site was allowed to close for the last time when a phrenic pacemaker was implanted in December 1972. He was hospitalized several times in 1974 and 1975 and treated for symptomatic cor pulmonale. Oxygen administration markedly depressed his respiration. The pacemaker did not provide an adequate tidal volume. In April 1975, the chest shell ventilator was used in synchrony with the pacemaker; but this also was not effective in relieving his severe symptomatic nocturnal hypoventilation. Finally, in November 1976, with a VC of 1,281 ml, he was placed on overnight MIPPV which normalized his blood gas values. He reported: "The first morning after being on the Bantam was truly like a miracle. I woke up bright-eyed and bushy-tailed, like my old self. It has been five years since I had woken up fully or even modestly alert." He continues 24 hour a day MIPPV and has not been hospitalized since 1976.

**Discussion**

Respiratory insufficiency in the majority of postpolio patients is due primarily to respiratory muscle weakness. Most of our patients requiring full time aid have VC s below 1 L. In others requiring full time aid, VCs as high as 1,700 ml have been noted. These patients may have intrinsic lung disease, central hypoventilation, or a pattern of upper airway collapse and sleep apnea episodes as well as thoracic deformity. Symptoms of hypoventilation are insidious in onset but lead to cor pulmonale and progressive cardiopulmonary failure. Often, these persons with signs of right heart failure progress despite treatment with nasal O2 and perhaps medroxyprogesterone.

With the use of MIPPV for only short periods several times a day, Lane et al. have reported improved tolerance of and reduced frequency of URIs, loss of ankle edema, and sometimes healing of peripheral ulcers. He also noted decreases in systemic hypertension, enhanced mental activity, and sustained improvement in blood gas values. Improvement in Po2 with a decrease in the P(A-a)O2 suggested improvement in ventilation/perfusion relationships probably from diminished atelectasis and better basal ventilation with deeper breaths ("sighs"). Bergofsky et al. reported sustained improvements in Po2 and Pco2 and normalization of pulmonary artery pressures, increased lung compliance and decreased work of breathing following frequent short periods of mouth intermittent positive pressure breathing.

Rochester et al. studied the respiratory muscle sparing effect of body respirator use for patients with both obstructive and restrictive respiratory insufficiency. The complete relief of dyspnea and EMG evidence of diaphragm rest observed were not associated with correction of blood gas values. Although Po2 levels were mildly increased from 52 mm Hg to 64 mm Hg, Pco2 levels averaging 54 mm Hg were not significantly changed by body respirator use. Carryover of the mildly improved Po2 levels to periods while not using body respirators was not discussed. In a later work, Braun et al. studied 35 patients, 18 with thoracic diseases, i.e., COPD, and 17 with neuromuscular diseases. All were chronically hypercapnic. They received four to ten hours a day of positive or negative pressure respiratory assistance. Braun reported increases in VC, maximum inspiratory and expiratory pressures, and maximum voluntary ventilation as a result of the treatment. Arterial Pco2 measured when off aid had decreased from 54 to 43 mm Hg and hospital admissions over a two-year period were significantly reduced in the treated group.

We have found complete relief of dyspnea, normalization of O2 saturations and Pco2 levels in the majority of our postpolio persons while on MIPPV despite VC approaching 0 ml in many cases. Abnormal although improved blood gas tensions might be anticipated with MIPPV use in patients with significant intrinsic lung disease.

The long-term significance of the muscle-sparing effect of assisting ventilation is unknown. Despite the transiently improved off-aid blood gas values, vital capacities, and sense of well-being that we observe in our patients using daily periods of respiratory assistance, neuromuscular and pulmonary deterioration continue. The average yearly loss of 1.9 percent of VC which we observed appears to occur irrespective of the style of respiratory assistance employed. Twenty-two of our patients who had been using respiratory aid for periods only during the day progressed to 24-hour respirator dependency. The nine patients in Table 3 also continue to require increasing periods of respiratory aid.

Theoretic concerns regarding the mode of respira-
tory aid to be employed to provide optimal muscle sparing also has little bearing on these cases. Until the 1970s, portable ventilators were available with only control mode ventilation. Although most of our patients now employ assist-control ventilation, in the absence of conscious respiratory drive during sleep, the patient quickly becomes habituated to allowing the cycling respirator to breathe for him and he triggers few assisted breaths. As VC deteriorates, patients trigger fewer assisted breaths during the daytime as well. We cannot discern any clinical difference in the pulmonary function or sense of well-being of those who trigger some breaths during the day as opposed to those on MIPPV using only control ventilation in the past or present. We feel that normalization of blood gas values and adequate “sighing” by regular respirator-assisted or glossopharyngeal breathing assisted breaths are more important considerations in preventing long-term cardiopulmonary sequelae in these patients.

The administration of nasal O\textsubscript{2} alone, even at low flow, to hypoventilating persons may partially relieve some symptoms and decrease pulmonary artery tensions, but it may also decrease the patient’s breathing effort and increase hypercapnia. In 1967, Bergofsky et al\textsuperscript{16} observed the importance of relieving both hypoxia and hypercapnia and preventing acidosis in normalizing pulmonary artery pressures with positive pressure ventilation. He noted that mechanical ventilatory assistance will also decrease the O\textsubscript{2} consumption and energy demands of respiratory muscles. Other work demonstrated the pulmonary artery pressor response to hypercapnia as well as hydrogen ion concentration.\textsuperscript{16}

Sleep apnea syndrome in polio patients was recognized as early as 1958.\textsuperscript{17} The MIPPV with correction of blood gas values provides an alternative to tracheostomy for many of these persons. Despite this, overnight nasal O\textsubscript{2} alone is still commonly recommended in severely hypercapnic persons (P\textsubscript{CO\textsubscript{2}} > 60 cm H\textsubscript{2}O).\textsuperscript{18}

We have observed 18 deaths; 17 patients died while receiving MIPPV or only one death for every 60.5 patient-years on MIPPV. Providing better ventilation protects against the development of right ventricular decompensation over a period of years. The deaths were related to pulmonary conditions in seven cases, noncardiopulmonary medical conditions in four cases, substance abuse in three cases, motor vehicle accident in one case, complications of tracheostomy in one case, and unknown causes in two cases. Of the seven deaths related to pulmonary causes, one patient died overnight while using the chest piece. She had been warned that it was no longer adequately ventilating her and was advised to change to using only MIPPV two years earlier. Two patients died when they fell asleep without using the lipguard. They lost the mouthpiece while sleeping. Two patients, one of whom was severely obese, died from complications of acute bronchopneumonia. One died from complications of recurrent massive atelectasis. He smoked heavily, had chronic bronchorrhea, frequent bouts of pneumonia, and died with advanced bullous emphysema. Finally, one patient died suddenly and was thought to have had acute obstruction due to a mucous plug probably during a URI.

Daytime use of negative pressure respiratory assistance does not permit normal daytime activities since persons have to be stationary and remain at least semirecumbent in these assistive devices. The chest shell, poncho, and rocking body respirators may provide optimal ventilation for years before loss of pulmonary compliance and complications of advancing respiratory insufficiency may render these methods ineffective. These body respirators are also ineffective in the presence of severe scoliosis and are not tolerated by many patients. Twenty-five of the persons noted in Table 1 switched to full time MIPPV because of this. Sixteen persons noted in Table 2 switched to daytime MIPPV and may follow with nighttime MIPPV in the future. The other seven employ MIPPV overnight and are still able to use the more cosmetic pneumobelt during the daytime. Only two of our patients on MIPPV have recently required supplemental O\textsubscript{2} because of intrinsic lung disease.

The Table 3 postpolio patients are at an “earlier stage,” requiring MIPPV only overnight at the present time. The use of MIPPV alone obviates the need for obtaining both positive pressure and negative pressure pumps. The MIPPV also provides optimal ventilation with tidal volumes exceeding those which can be delivered by the present models of the iron lung.

There are a number of patients who, although not dependent on respiratory assistance when well, employ MIPPV overnight and for periods during the daytime when ill. Still other patients not included in these tables require aid for particular reasons. One man, a victim of polio in 1944, developed severe postural respiratory insufficiency and was unable to tolerate recumbency since 1962. He slept in the sitting position for 18 years. He was misdiagnosed and treated for narcolepsy because of severe somnolence due, in reality, to progressive respiratory insufficiency. The MIPPV has allowed him to sleep recumbent since 1981.

In a few patients, including the one noted in the previous paragraph, there is a severe leak of insufflated air from the nose during sleep due to an incompetent soft palate. Sealing the nose or a mask covering the nose and mouth has been used. Substituting for the Bennett lipseal with molded dental appliances or other modifications are occasionally required.

We have observed no serious complications due to positive pressure ventilation such as pneumothorax or
symptoms related to increased intrathoracic pressure. Aerophagia, although often a source of discomfort at first, is generally relieved by the passing of flatus during normal daily activities. Occasional severe abdominal distension and discomfort can be relieved with the use of a rectal tube. This has never been a reason to discontinue MIPPV.

The MIPPV should, therefore, be introduced early to postpolio survivors with slowly progressive restrictive pulmonary syndrome and signs or symptoms of hypoventilation. It can help prevent acute cardiopulmonary complications associated with chronic alveolar hypoventilation and allow optimal activities of daily living while providing an effective alternative to tracheostomy. Similar studies indicating its effectiveness in prolonging life for 31 patients with Duchenne muscular dystrophy and other neuromuscular conditions have been reported. It is the technique of choice for patients without severe intrinsic lung disease who require 24-hour respiratory aid or who are likely to progress to 24-hour respirator dependency and who have sufficiently intact oropharyngeal muscles to prevent excessive inspiratory leak.

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Mouth IPPV in Postpolio Respiratory Insufficiency (Bach et al)