Pulmonary dysfunction and its management in post-polio patients

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Abstract

Respiratory dysfunction is extremely common and entails considerable risk of morbidity and mortality for individuals with past poliomyelitis. Although it is usually primarily due to respiratory muscle weakness, post-poliomyelitis individuals also have a high incidence of scoliosis, obesity, sleep disordered breathing, and bulbar muscle dysfunction. Although these factors can result in chronic alveolar hypoventilation (CAH) and frequent pulmonary complications and hospitalizations, CAH is usually not recognized until acute respiratory failure complicates an otherwise benign upper respiratory tract infection. The use of non-invasive inspiratory and expiratory muscle aids, however, can decrease the risk of acute respiratory failure, hospitalizations for respiratory complications, and need to resort to tracheal intubation. Timely introduction of non-invasive intermittent positive pressure ventilation (IPPV), manually assisted coughing, and mechanical insufflation-exsufflation (MI-E) and non-invasive blood gas monitoring which can most often be performed in the home setting, are the principle interventions for avoiding complications and maintaining optimal quality of life. © 1997 Elsevier Science Ireland Ltd.

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1. Pathophysiology of respiratory sequelae

It has been estimated that 75000 people afflicted with paralytic poliomyelitis in the United States from 1928

to 1962 developed ventilatory failure and/or swallowing impairment *[1]*. About 12.5% of the ventilator users could not be weaned from ventilator use and many are still alive and have been ventilator supported 24 h a day, often by non-invasive means, for over 40 years *[2]*. Although these aging post-poliomyelitis ventilator users have been gradually decreasing in number, many other post-poliomyelitis individuals have had new breathing problems *[3]*, and the total number of these patients who now benefit from ventilator use appears to be increasing. Although many patients who were initially weaned, once again require ventilator use *[1]*, other post-poliomyelitis individuals are now using ventilatory assistance for the first time. In one study, chronic ventilatory insufficiency and need for daily ventilatory assistance occurred an average of 18 years post-acute poliomyelitis; however, return to ventilator use is not infrequently required 40 years or more post-poliomyelitis *[1,4,5]*.

Both inspiratory and expiratory musculature are usually weakened by poliomyelitis. Inspiratory muscle weakness often leads to chronic alveolar hypoventilation (CAH). Patients with late-onset ventilatory insufficiency have been reported to lose vital capacity (VC) at rates of 60-90% greater than normal *[4,5]*. Decreases in VC, tidal volumes, and inability to take occasional deep breaths lead to chronic microatelectasis, decreased pulmonary compliance, and increased stiffness of the chest wall and work of breathing *[7,8]*.

Hypercapnia is likely when the VC falls below 55% of predicted normal and it is insidiously progressive *[9]*. Hypoxia, hypercapnia, and rate of loss of VC are exacerbated when intrinsic lung disease, kyphoscoliosis, or obesity complicate inspiratory muscle weakness. When not corrected by appropriate use of inspiratory muscle aids, respiratory control centers accommodate hypercapnia *[10]*, and a compensatory metabolic alkalosis develops. The resulting elevated central nervous system bicarbonate levels contribute to depression of the ventilatory response to hypoxia and hypercapnia. This permits worsening of CAH, and may decrease the effectiveness of the nocturnal use of inspiratory muscle aids once instituted.

Although often ignored, expiratory muscle weakness is often the key factor that results in acute respiratory failure. Expiratory and bulbar muscle weakness can result in severely decreased peak cough flows (PCF) that prevent effective elimination of airway secretions. Whether attained by autonomous coughing or by the use of manually or mechanically assisted coughing, PCF of at least 3-5 l/s are necessary for effective airway secretion and mucus clearance *[12]*. Adequate PCF are most important during intercurrent respiratory tract infections. Likewise, bulbar muscle dysfunction that impairs retention of optimal breaths with a closed glottis can greatly decrease PCF. Bulbar dysfunction can also lead to aspiration of food or saliva or upper airway obstruction for laryngeal incompetence. Laryngeal dysfunction can not only result from the primary neuromuscular process but also commonly results from complications of translaryngeal intubation and tracheostomy. Likewise, fixed lower airway obstruction from concomitant chronic obstructive pulmonary disease (COPD), the presence of tracheal stenosis, or any other irreversible impediment to the generation of optimal PCF greatly increases the risk of pulmonary morbidity. PCF are also decreased by any inspiratory muscle weakness that decreases the inspiratory capacity below 2.5 liters *[13]*.

Smoking, the presence of an endotracheal cannula, or bronchorrhea for any other reason increases the tendency to develop chronic mucous plugging in these individuals. This, in turn, leads to ventilation/perfusion imbalance, atelectasis, pneumonias, pulmonary scarring, and further loss of lung compliance. A large mucous plug can cause sudden death. Post-poliomyelitis individuals with chronic mucus plugging can require repeated intubation, bronchoscopy, and tracheostomy. Mucous plugging that results in acute respiratory failure most often occurs during otherwise benign intercurrent upper respiratory tract infections. Acute respiratory tract infections and hypercapnia itself can exacerbate respiratory muscle weakness *[14]*. In addition, respiratory muscles weaken with age, fatigue, and ultimately with the death or dysfunction of over-worked anterior horn cells which had survived the acute poliomyelitis. This is manifested by a decrease in pulmonary volumes, maximum inspiratory and expiratory pressures, and peak airflows.

Post-poliomyelitis individuals may also be particularly susceptible to the development of sleep disordered breathing *[15,16]*. Sleep disordered breathing is the occurrence of multiple central and/or obstructive apneas

and hypopneas during sleep. Its incidence increases with age *[17]*. The obstructive sleep apnea syndrome (OSAS) is diagnosed when such individuals are symptomatic and have a mean of 10 or more apneas plus hypopneas per hour *[18,19]*. A higher incidence might be due to the commonly observed high incidence of bulbar muscle weakness in this population *[20,21]* which can increase susceptibility to hypopharyngeal collapse and obstructive apneas during sleep. Damage to respiratory control centers might also have occurred from the encephalitic process of the primary viral infection *[21-24]*. Sleep disordered breathing alone can result in CAH, hypoxia, right ventricular strain, and when severe, acute cardiopulmonary failure.

2. Conventional management

The great majority of patients with neuromuscular pulmonary dysfunction are undiagnosed and untreated until an otherwise benign intercurrent respiratory tract infection leads to the development of acute respiratory failure. Complicating obesity, abdominal distention, dehydration, under-nutrition, and hypercapnia decrease both inspiratory and expiratory muscle function *[25]*. Mucus plugs airways and the patient can often not generate sufficient PCF to eliminate it. This results in atelectasis, ventilation perfusion mismatching, exacerbates CAH and results in acute respiratory failure.

The patients are often hospitalized and receive intermittent positive pressure breathing (IPPB) treatments but at inadequate pressures for effective inspiratory muscle assistance. The bronchodilators and methyixanthines which are often used usually result in no significant clinic benefits for these patients with purely restrictive pulmonary conditions. Instead of providing respiratory muscle aids, patients are also treated with supplemental oxygen that maintains oxyhemoglobin saturation (SaO_2,) often at the cost of permitting life-threatening hypercapnia. Oxygen therapy can also prevent the signaling of bronchial mucous plugging-associated oxyhemoglobin desaturations that might otherwise be cleared with the use of respiratory muscle aids. It is not surprising that many of these patients are eventually intubated. Supplemental oxygen should only be used for acutely ill hypoxemic patients with significant intrinsic lung disease after alveolar ventilation and airway secretions have been optimally managed with the use of respiratory muscle aids.

The failure to provide non-invasive respiratory muscle aids in a timely manner, therefore, often leads to otherwise unnecessary intubation and bronchoscopies. Once intubated the patient may not have sufficient respiratory muscle function for early ventilator weaning, particularly when mucous plugs are inadequately removed, respiratory control centers are dulled by supplemental oxygen administration, and deconditioning and inadequate nutrition complicate the picture. Then, after perhaps refusing it for years, the patient may succumb to tracheostomy. Weaning attempts from tracheostomy IPPV usually continue with some combination of assist control or synchronized intermittent mandatory ventilation (SIMV) used in combination with pressure support ventilation, positive end-expiratory pressure (PEEP), and supplemental oxygen. Occasionally progressive ventilator free breathing with T-piece supplemental oxygen and continuous positive airway pressure (CPAP) approaches are taken. It is hard to know which factors to manipulate as the patient's carbon dioxide levels increase during the weaning attempts. A more effective and physiological weaning approach using non-invasive respiratory muscle aids will be described in the section on pulmonary management.

3. Patient evaluation

Full batteries of pulmonary function studies and arterial blood gas sampling are rarely useful for outpatients with any primarily restrictive pulmonary conditions unless concomitant intrinsic lung disease is suspected. A careful history, simple spirometer, and a peak flow meter are most useful for routine patient evaluation and an oximeter and capnograph are useful for more affected patients.

The symptoms of CAH are essentially the same as those for sleep disordered breathing and include fatigue, headaches, sleep disturbances, difficult arousals, hypersomnolence, impaired concentration, nightmares, irritability, anxiety, nocturnal urinary frequency, impaired intellectual function, depression, and memory

impairment *[10]*. These symptoms are often misinterpreted. Likewise, during acute respiratory tract infections wheelchair users with impending respiratory failure complain more often of anxiety and difficulty falling asleep than of shortness of breath.

The respiratory evaluation should take into account any history of allergies, asthma, smoking, previous respiratory hospitalizations, intubations, and bronchoscopies. The VC, maximum insufflation capacity (MIC), forced expiratory volume in one second (FEV_1), PCF, non-invasive blood gas monitoring, and when the diagnosis is unclear, polysomnography, are also useful tests. Because of the availability of oximetry and capnography, arterial blood gas sampling is rarely indicated in the outpatient setting.

The VC should be measured with the patient sitting, supine, side lying, and when wearing thoracolumbar orthoses when applicable. A properly fitting orthosis can increase VC whereas a poorly fitting one that restricts chest movement will decrease it. The VC is often most reduced when the post-poliomyelitis individual is supine because of inordinant diaphragm weakness. The presence of CAH may not be suspected unless the VC is obtained in this position. Evaluation of FEV_1 should be done whenever COPD is suspected.

If the VC is less than 1500 ml, the MIC should be determined. The MIC is a measure of the maximum volume of air that can be held with a closed glottis. To attain the MIC, the patient can receive one maximally tolerated insufflation, air stack volume ventilator delivered tidal volumes, use maximum depth glossopharyngeal breathing (GPB), or perform some combination of these methods *[26]*. The MIC is a function of pulmonary compliance and bulbar muscle control. It is also useful for predicting the glossopharyngeal maximum single breath capacity (GPmaxSBC) *[26]*. The greater the PCF, GPB potential, and voice volume during mouthpiece IPPV, the greater the potential for using non-invasive alternatives to tracheostomy.

Any patient with respiratory symptoms, hypercapnia, or a supine VC less than 40% of predicted normal should undergo SaO_2 monitoring and possibly capnography or transcutaneous PcO_2 monitoring during sleep *[10]*. Non-invasive blood gas monitoring is most conveniently performed on an outpatient basis. The oximeter should be capable of averaging data hourly *[10]*. Hourly SaO_2 means less than 95% in a symptomatic patient with supine VC less than 50% of predicted normal and without intrinsic lung disease is sufficient to make a presumptive diagnosis of CAH and initiate treatment with non-invasive respiratory muscle aids. Maximum nocturnal PcO_2 greater than 50 mmHg is also an indication for treatment, especially when the disease course is clearly progressive or the patient has had recent pulmonary complications.

Although patients with CAH can have many transient and often severe oxyhemoglobin desaturations, a 'sawtooth' pattern with more than 10 transient 4% or greater desaturations per hour in a symptomatic patient with normal supine VC and mean SaO_2 may signal uncomplicated sleep disordered breathing. For symptomatic patients oximetry studies alone are highly sensitive in screening for this condition *[27,28]*. Outpatient polysomnography can assist in the evaluation of patients who are symptomatic despite having inconclusive nocturnal oximetry and carbon dioxide studies and relatively normal VCs *[29]*. Some patients' symptoms are the result of a combination of inspiratory muscle weakness and sleep disordered breathing. Patients should be reevaluated yearly or whenever there is a change in symptoms.

4. The non-invasive respiratory muscle aids

4.1. The inspiratory aids

Inspiratory muscle aids or ventilatory assistance can be provided by negative pressure body ventilators. These devices apply sub-atmospheric pressure changes around the chest and abdomen. These methods are cumbersome, less effective, and less practical, particularly for daytime aid, than non-invasive IPPV. They have been described elsewhere *[30]*. We have found them useful only for providing ventilatory support during tracheostomy site closure when switching patients with little or no ability to breathe from tracheostomy to

non-invasive IPPV *[31,32]*.

Body ventilators that act directly on the body include the rocking bed and the intermittent abdominal pressure ventilator (IAPV). Although the rocking bed is one of the least effective devices, the IAPV continues to be particularly useful. It consists of an inflatable bladder in an abdominal girdle. The bladder is cyclically inflated by a portable positive pressure ventilator. The alternating pressure on the abdominal contents moves the diaphragm and ventilates the lungs. The IAPV generally augments the patient's autonomous tidal volumes by about 600 ml *[33]*. It is most effective in the sitting position at 75-85°. It is the method of choice for daytime ventilatory support for patients with less than 1 h of ventilator-free breathing ability because it provides better appearance than any other method of ventilatory assistance and it is ideal for concurrent GPB and wheelchair use *[34]*.

The inspiratory muscle aids which apply IPPV non-invasively to the airway are the most effective, most practical, and best tolerated alternatives to tracheostomy IPPV for neuromuscular patients with inspiratory muscle dysfunction who require assistance up to 24 h/day *[35]*. They are also greatly preferred by patients and caregivers over the use of tracheostomy IPPV *[36]*.

Non-invasive IPPV can be provided via oral *[35]*, nasal *[10]*, or oral-nasal *[37]* interfaces. For daytime ventilatory support, IPPV is most conveniently provided via simple mouthpieces that are held near the patient's mouth for easy accessibility. The mouthpiece might be fixed adjacent to the sip-and-puff or chin controls of a motorized wheelchair. Oximetry feedback can be used and the patient told to take assisted breaths whenever necessary to maintain normal SaO_2. Thus oximetry can guide the patient with CAH in using an appropriate schedule of IPPV to normalize ventilation during daytime hours *[35]*. The patient is usually instructed to keep the SaO_2 greater than 94% throughout daytime hours *[6,26]*. Patients use mouthpiece IPPV for increasing periods of time as respiratory muscles weaken and they often eventually use it 24 h/day (Fig. 1). It is the most effective and generally the preferred method of daytime support *[35]*.

Fig. 1. This patient had acute poliomyelitis in 1955 and was a 24 h ventilator user until his death from leukemia in 1995. He switched from relatively ineffective body ventilator use to mouthpiece IPPV for daytime ventilatory support (seen here) in 1958 and for nocturnal ventilatory support in 1982.

For nocturnal use many patients choose to use mouthpiece IPPV with lipseal retention (Puritan-Bennett, Boulder, CO (Fig. 2)); however, most patients prefer nasal IPPV *[10,38-40]*. Each patient should try at least 3 or 4 commercially available CPAP masks as interfaces for IPPV to determine the ones which best optimize fit,

seal, and comfort. If none are adequate, custom interfaces are constructed *[10,41]*. These interfaces can also be useful for the delivery of CPAP in managing simple sleep disordered breathing *[42]*. Several interfaces that can be custom molded to the patient's nose are commercially available (SEFAM kit, Lifecare International Inc. [now Respironics Inc], Westminster, CO). These interfaces are comfortable and effective at higher pressures but are also expensive, delicate, and require frequent re-fabrication. Transparent, durable, custom molded low profile nasal interfaces can also be prepared from a plaster moulage as in Fig. 3 *[37]*. They are lighter, durable, comfortable and cosmetic in ap- pearance but require several patient visits.

Fig. 2. This patient with no measurable vital capacity had acute poliomyelitis in 1956 and since switching from relatively ineffective use of negative pressure body ventilators has been using mouthpiece IPPV for daytime support since 1956 and for nocturnal ventilatory support with lipseal and strap retention (seen here) since 1980.

Fig. 3. Custom acrylic nasal interface for nocturnal nasal IPPV.

If nocturnal oximetry and possibly capnography do not demonstrate adequate improvement of ventilation

using nasal IPPV, the problem is usually that the patient's lungs are underventilated during daytime hours. If this is the case, the patient is asked to use sufficient daytime aid to normalize daytime SaO_2. The patient can also be switched to nocturnal use of mouthpiece IPPV with lipseal retention. A custom molded acrylic bite plate and outer shell (lipseal) can also be fabricated to decrease the risk of orthodontic deformity *[6,35]*. When using mouthpiece IPPV a complete seal can be obtained by plugging the nose with cotton pledgets and tape, thus creating a closed system *[35]*.

For ventilator users who live alone and who are unable to manage a strap retention system, a strapless interface can be constructed *[37,41]*. Oral and oro-nasal interfaces are often used with bite plate rather than with strap retention. Thus, air can be delivered via comfortable custom molded interfaces for nasal, oral, or oro-nasal IPPV.

A 31-year-old Texan woman who had poliomyelitis at 4 years of age developed a respiratory tract infection which led to acute respiratory failure and a respiratory arrest while on route to a local hospital. She was intubated and subsequently tracheostomized. Attempts at ventilator weaning failed and she was reluctantly transferred to a chronic nursing care facility despite the fact that she required only nocturnal ventilatory support and when sitting had a VC of 1030 ml but only 630 ml when supine. She was not permitted to return home to her husband who had a 'nine to five' job because third party payers would not pay for tracheal suctioning by other than licensed health care professionals and licensed professionals were deemed too expensive. She, therefore, remained at the chronic care facility using tracheostomy IPPV with an inflated cuff for 6 months. This caused severe tracheomalacia.

She was transferred to a rehabilitation facility for decanulation. On the first night her cuff was deflated and the delivered ventilator volumes increased to compensate for insufflation leakage through the upper airway. This relieved the pressure on the trachea and permitted speech. Then, her tracheostomy tube was removed; a firm occlusive dressing was placed over the site; and the patient was placed on nocturnal nasal IPPV. She used this for 2 weeks until she complained of nasal bridge discomfort from the nasal interface. Once again an attempt at weaning failed when after 10 days off aid, severe fatigue and blood gas deterioration resulted in her being placed in an iron lung overnight for 2 weeks. She was converted to using a Pulmowrap Ventilator overnight and discharged home. Although this adequately assisted her inspiratory muscle function, it was inconvenient to wear and she switched to using a strapless oral nasal interface (SONI) for IPPV. She continues to use nocturnal SONI IPPV and has not required re-hospitalization.

4.2. The expiratory aids

The expiratory muscles can be assisted by providing maximal insufflations then manually applying thrusts to the abdomen timed to glottic opening or by using mechanical insufflation-exsufflation (MI-E) *[12]*. There are at least 12 different methods of manually assisted coughing *[43]*. Manually assisted coughing should not be used following meals because of the risk of regurgitation and aspiration of food. Since normal cough volumes are from 2.3 to 2.5 l and PCF diminish greatly when expiratory volumes are less than 1500 ml, a good rule of thumb is to provide a maximal insufflation for any patient whose VC is under 1500 ml before applying the abdominal thrust to assist the cough *[12]*.

Mechanically assisted coughing is used when manual techniques are inadequate to eliminate airway secretions to maintain normal SaO_2, when the stomach is full, and when the care provider cannot generate sufficient force for or effectively coordinate abdominal thrusts. In particular, manually assisted coughing is often inadequate for patients with severe scoliosis or obesity. Mechanically assisted expulsion of airway secretions is less labor intensive, more effective, and can be achieved with MI-E devices (Fig. 4, Mechanical In-Exsufflator, J.H. Emerson Co., Cambridge, MA) *[12,44-46]*. MI-E delivers an optimal insufflation via an oral-nasal mask, mouthpiece, or endotracheal tube, which is followed by a decrease in pressure, usually of about 80 cmH_2O over a 0.2 s period, to create a forced exsufflation of 6-10 l/s. An abdominal thrust may be timed to the machine's exsufflation cycle to further increase PCF. These forced exsufflation flows carry

airway secretions up into the mouth, mask, or tubing. This can immediately increase VC, maxi- mum pulmonary airflows, and SaO_2 *[12]*. Manually assisted coughing and MI-E can be necessary every 10-15 min around-the-clock during respiratory tract infections and following surgical anesthesia *[12,46]*.

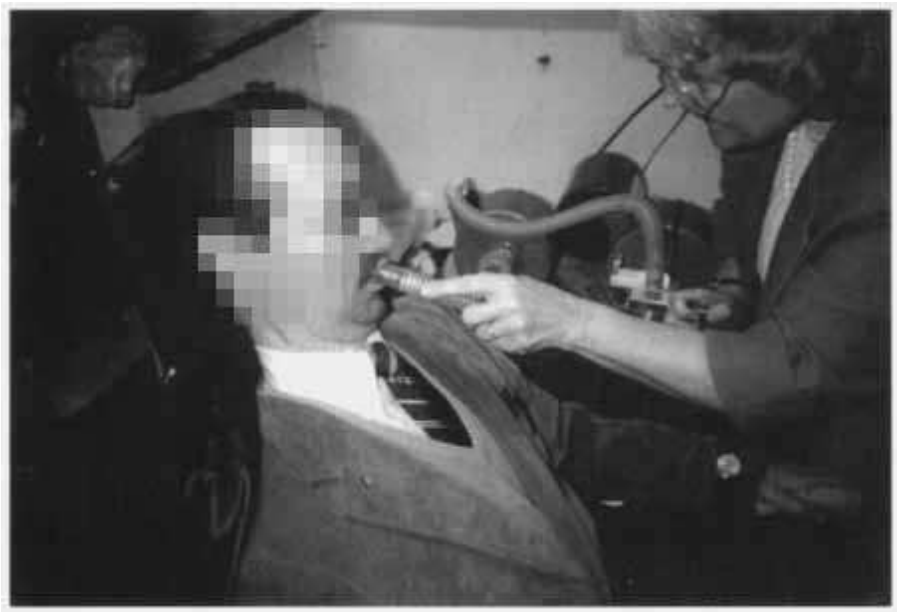

Fig. 4. Post-poliomyelitis ventilator user undergoing mechanical insufflation-exsufflation (pressures +40 to -40 cmH_2O) via a simple mouthpiece.

Chest physical therapy techniques may be useful for patients with COPD but even for these patients chest physical therapy has not been shown to reduce the risk of pulmonary complications or hospital stays and it is not as effective as generating optimal PCF *[47-49]*. Other mechanical approaches for eliminating secretions may be useful for the patient with concomitant COPD whose airways collapse excessively at high exsufflation pressures. Rapidly oscillating pressure changes can be applied to the chest wall or directly to the airway. These methods may be particularly useful in combination with postural drainage techniques. Their use may also be helpful prior to using manually assisted coughing for some patients *[50,51]*.

4.3. Glossopharyngeal breathing

Avoiding or eliminating a tracheostomy tube permits the ventilator user to more readily learn and benefit from GPB. GPB should be taught to patients with less than 1000 ml of VC and adequate oropharyngeal muscle strength for functional swallowing and speech *[26]*. During GPB the tongue and pharyngeal muscles add to inspiratory efforts by projecting boluses of air past the vocal cords. Effective GPB permits patients with little or no measurable VC to sustain autonomous ventilation for hours. It also normalizes speech production and provides deeper breaths for shouting and increasing PCF. Progress with GPB should be monitored by regularly measuring the volume of air per gulp and the number of gulps per breath.

5. The management of post-poliomyelitis respiratory sequelae

Patient counseling includes cautioning to avoid dehydration, heavy meals, extremes of temperature, humidity, excessive fatigue, exposure to respiratory tract pathogens, obesity, sedatives, and narcotic use. The need for appropriate flu and bacterial vaccinations and early attention to maintaining alveolar ventilation and eliminating airway secretions during respiratory tract infections are important to decrease the risk of pneumonia. Likewise, local hospitals and family physicians need to be advised on the patient's needs in advance of any elective or unanticipated surgical procedures. If present, reversible bronchospasm should he treated. Therapeutic exercise programs, extremity bracing, energy conservation, assistive equipment needs,

and day to day functioning should also be addressed *[52,53]*. The key therapeutic goals are to maintain normal alveolar ventilation around the clock, to provide 'range of motion' to the lungs and chest wall, and to provide sufficient PCF to effectively clear airway secretions. Therapeutic options for accomplishing these goals should be presented and the patient ultimately trained and equipped.

6. Pulmonary compliance and oximetry feedback

Once the VC has decreased to approximately 50% of predicted normal in any position our patients are instructed to take deep insufflations via a mouthpiece several times a day. A manual resuscitator is usually used to provide the deep insufflation. The goal is to approach the predicted maximum inspiratory capacity and thus provide 'range of motion' to the lungs and chest wall, maximize the MIC, reduce microatelectasis, and at least temporarily increase dynamic pulmonary compliance and decrease the work of breathing. It is very important that the clinician ascertain that the patient has learned this technique so that he or she can use mouthpiece IPPV to effectively ventilate the lungs during intercurrent respiratory tract infections. We also introduce the patient to MI-E at this time if PCF are noted to be below 4-4.5 l/s. MI-E is made to be quickly accessible to treat bronchorrhea when airway mucus becomes a problem *[12]*.

During intercurrent respiratory infections the VC drops precipitously and hypercapnia and hypoxemia can develop or worsen, The patient will then need a portable ventilator to use mouthpiece IPPV (with lipseal retention) up to 24 h a day. Daytime mouthpiece IPPV and nocturnal nasal IPPV, manually assisted coughing, and an in-exsufflator (J.H. Emerson Company, Cambridge, MA) and an oximeter to signal hypoventilation and mucus plugging can he very important. Provided that the patient maintains normal alveolar ventilation with or without the use of IPPV, acute oxyhemoglobin desaturation can only be explained by mucous plugging, atelectasis, or intrinsic lung disease. Sudden decreases in SaO_2 are usually due to mucous plugging, and are reversed by manually or mechanically assisted coughing, returning the SaO_2 to baseline. With effective elimination of airway secretions baseline SaO_2 depressed to 92-94% by the presence of microatelectasis normalizes over a period of days with these treatments and supportive medical therapy. If the baseline SaO_2 continues to fall, pneumonia becomes likely and the patient usually requires hospitalization for more intensive and invasive therapy. This is uncommon, however, for the patient using respiratory muscle aids and oximetry as described.

7. Tracheal decanulation and weaning

Most patients with neuromuscular respiratory muscle dysfunction are first recognized to have CAH only after an intercurrent respiratory tract infection has resulted in endotracheal intubation and, not infrequently, tracheostomy. The trachcostomized patient, however, can be decanulated and converted to the use of strictly non-invasive respiratory muscle aids provided that he or she satisfy the criteria noted in Table 1.

Table 1. Criteria for the safe transition from IPPV delivered via an endotracheal tube to non-invasive IPPV

The candidate must:

- Be medically stable
- Be alert, coopertive, and not receiving sedatives or narcotics
- Have unassisted or assisted PCF exceeding 3 l/s
- Have no seizure disorder
- Have no orthopedic impediments to facial interface use
- Have baseline SaO_2 greater than 91% on full ventilatory support and room air once airway secretions have been optimally eliminated

We first switch any indwelling tracheostomy tube to a fenestrated cuffed tube. The cuff should be deflated for

increasing periods of time since virtually all tracheostomy IPPV users can be adequately ventilated with the cuff fully deflated, day or night, provided that a tracheostomy tube with the proper diameter is used *[54]*. The patient uses an inflated cuff only during MI-E. Whether used via a facial interface or an endotracheal or tracheostomy tube, MI-E that eliminates airway secretions results in immediate increases in VC and SaO_2. Thus, these patients are aggressively exsufflated through the indwelling tubes and maintained free of supplemental oxygen.

We then introduce the patient to mouthpiece and nasal IPPV with the tracheostomy tube capped. Once learned *[31]* the tube can be removed, a tracheostomy button placed, and the PCF measured with the airway now clear. Mouthpiece IPPV is used during daytime hours with oximetry feedback and either nasal or lipseal IPPV used overnight. Whether or not the patient has little or no ability to breathe or can breathe autonomously with a VC exceeding 2000 ml, it is necessary to attain unassisted or assisted PCF of 3 l/s or more to permit safe decanulation and conversion to non-invasive respiratory aids as needed. Once the patient is comfortable using non-invasive IPPV and PCF of 3 l/s have been documented, we remove the button and the tracheostomy site closes. For patients with little or no measurable VC it can be useful to use negative pressure body ventilators for 24 h support until the site is closed. If the site does not close completely within 7 days, it should be sutured closed. Airway secretions will 'dry up' in 2-3 weeks after tracheostomy site closure unless the patient has post-nasal drip, allergic bronchorrhea, or chronic bronchitis The patient and family are instructed to monitor SaO_2 during future episodes of respiratory infection, distress, or fatigue, and to use non-invasive IPPV and manually and mechanically assisted coughing as needed to maintain normal SaO_2 in the future. The patient is also instructed to notify his or her physician at the first signs of infection or airway congestion for antibiotics and other supportive therapy. Thus, using MI-E as necessary and oximetry as feedback, patients wean from tracheostomy IPPV by taking fewer and fewer mouthpiece assisted breaths while maintaining normal alveolar ventilation on room air until weaning is completed or until the extent of ongoing need for non-invasive IPPV is determined.

Patients may wean to nocturnal-only use of non-invasive IPPV or require daytime IPPV as well. Patients, however, wean themselves on their own schedules. This incurs less anxiety than weaning from tracheostomy IPPV using synchronized mandatory ventilation (SIMV) or periods of CPAP and free breathing because they know that they always have immediate access to deep breaths. Thus, we first wean the patient from supplemental oxygen administration by using assisted coughing and MI-E to eliminate mucous plugging; he or she is then weaned from tracheostomy IPPV and the site allowed to close; and finally, if possible, he or she is weaned from non-invasive ventilatory assistance. There is often no need to try to solve the common puzzle of which to decrease first - the SIMV, the positive end-expiratory pressure, the oxygen, or the pressure support.

If a patient who is experienced in taking deep insufflations via a mouthpiece or nasal interface is translaryngeally intubated, he or she can be safely extubated and switched to 24 h mouthpiece or nasal IPPV and assisted coughing provided that the criteria noted in Table 2 are satisfied [LPP Library Note: A Table 2 is not present in the original publication. It is possible that the intended reference is Table 1.] For patients who are untrained in non-invasive IPPV techniques, this should not be attempted without using a negative pressure body ventilator for effective ventilatory support during extubation and training in non-invasive IPPV techniques. For untrained individuals with little ability to breathe, extubation and attempts at supporting ventilation by non-invasive IPPV without back-up body ventilator support are difficult and hazardous because dyspnea, oxyhemoglobin desaturation, and panic can occur during training attempts under these conditions.

8. Sleep disordered breathing

There are patients for whom neither sleep disordered breathing, nor COPD, nor their restrictive pulmonary syndrome are severe enough to warrant treatment but who with multiple conditions require the use of respiratory muscle aids to remain healthy *[6]*. Any reversible conditions associated with OSAS should he

identified and treated *[55,56]*. However, for the majority of patients with simple OSAS, nocturnal nasal CPAP is effective. CPAP works as a pneumatic splint to maintain airway patency. CPAP of 5-15 cmH_2O is usually adequate. Independently varying the inspiratory (IPAP) and expiratory (EPAP) pressures with the use of bi-level positive airway pressure (BPAP) can improve effectiveness and comfort.

To optimize treatment efficacy nocturnal recordings of SaO_2 are done at various CPAP or BPAP settings. For patients with a combination of neuromuscular ventilatory insufficiency and sleep disordered breathing, both CPAP and BPAP with low IPAP/EPAP spans are ineffective. Either a high IPAP/EPAP (e.g. 20/4) span or the use of volume-triggered ventilators should be used for non-invasive IPPV. Non-invasive IPPV both supports the patient's ventilation and maintains upper airway patency *[6,10,15,38-41]*. The use of demand positive airway pressure (Medical Systems Inc., Hampton, NH) in which positive pressure varies with autonomous breathing airflows has yet to be adequately compared with BPAP in clinical settings.

9. Preparation for surgical anesthesia

Post-poliomyelitis ventilator users require surgical intervention more frequently than the general population *[57]*. Mastery of the use of respiratory muscle aids prior to surgery can greatly decrease the risk of pulmonary morbidity. A hospital of the patient's choice is made aware of the patient's equipment and special needs. For the majority of hospitals which do not own portable ventilators or In-Exsufflators, this will require advanced notice. Nursing and respiratory therapy in-services are required.

The VC, PCF, PcO_2 and SaO_2 are sensitive indicators of risk of post-surgical pulmonary complications. The lower the VC below 60% and the PCF below 5 l/s the greater the likelihood of complications. These patients are trained in receiving maximum insufflations and non-invasive IPPV and in manually assisted coughing and MI-E in anticipation of post-operative ventilator weaning difficulties. Access to MI-E is particularly important for patients who undergo abdominal surgery *[58,59]*. The use of MI-E requires no abdominal muscle effort and abdominal pressures are increased 70% less than during unassisted coughing *[60]*.

General anesthesia should in general be avoided in favor of local or regional anesthesia whenever possible and non-essential elective procedures avoided. Simple inhalation anesthetic techniques should be used whenever possible. The use of opioids and muscle relaxants should be used sparingly or avoided *[57]*.

Provided that the presurgical PCF exceeded 3 l/s and high dose narcotics, sedatives, and supplemental oxygen are avoided, once the ventilator user is fully alert following anesthesia, he or she can be safely extubated whether or not capable of autonomous ventilation. Immediately upon extubation the patient receives IPPV via a mouthpiece or nasal interface and uses nasal or lipseal IPPV during sleep with oximetry monitoring. Except during the surgical procedure itself patients should be permitted to use their own equipment because, besides the differences in flow characteristics, the expiratory volume alarms of the usual hospital ventilators can make it impractical to use non-invasive IPPV. The secretions stimulated by the intubation and anesthesia can usually be efficiently eliminated by manually assisted coughing or MI-E and pulmonary and laryngeal complications are averted by avoiding prolonged intubation.

10. Not all 'asthmatics' have asthma

A patient, diagnosed with asthma, reported having had fatigue, difficulty concentrating, and extreme daytime drowsiness for 10 years. He had been taking bronchodilators for 12 years without objective or subjective relief. His VC was 1.15 l (20% of predicted normal); FEV_1, 0.94 l (23%); and FEV_1/FVC was 103%. He was intubated, temporarily ventilator supported, and weaned on three occasions, but during a forth hospitalization, weaning attempts failed and he underwent tracheostomy on the 52nd hospitalization day. He then lost all autonomous breathing ability and 4 days following tracheostomy he became septic. Atelectasis, lobar collapse, and bronchial mucus plugging became persistent problems. Episodes of respiratory arrest during weaning attempts, and septic shock, necessitated pressor support. Although the effusion and infiltrate

eventually cleared weaning attempts continued to fail over a 3 month period with $PaCO_2$ levels quickly exceeding 60 mmHg. Weaning was attempted in the conventional manner of gradually decreasing synchronized intermittent mandatory ventilation (SIMV) rates and volumes while providing pressure support ventilation, positive end-expiratory pressure (PEEP), and oxygen. These attempts continued despite the fact that his maximum effort VC was 570 ml; he had no ventilator-free breathing ability; and he had extremely hypersecretory airways and profuse mucous plugging. He also continued to require full alimentation via a nasogastric tube.

We managed this patient by changing his tracheostomy tube to a fenestrated tube and capping it to permit him to practice receiving mouthpiece IPPV. After several days, he was able to tolerate mouthpiece IPPV most of the day and nasal IPPV overnight. Oxygen therapy was discontinued. As he breathed room air SaO_2 was 90-95% during daytime hours and he had a mean nocturnal SaO_2 of 93% with typical 'sawtooth' oxyhemoglobin desaturations. The tracheostomy tube was then removed and the site buttoned for 5 days before being allowed to close. Thus, despite absence of any ability to breathe without a ventilator, he was converted directly from continuous tracheostomy IPPV to daytime mouthpiece and nocturnal nasal IPPV. Pa,O_2 was maintained over 60 mmHg by providing adequate ventilatory support and assisting coughing by air stacking. Since his abdominal muscles were functional, manually assisted coughing techniques were not useful. PCF increased from 1.3 l/s to 4.4 l/s when he maximally air stacked via a mouthpiece. Transition to non-invasive IPPV and tracheostomy site closure took 2 weeks. Ventilator-free breathing ability remained under 5 min despite an increase in VC to 640 ml and the alleviation of airway hyper-secretion by tracheostomy site closure.

His rehabilitation included training in GPB, general strengthening, mobility, and endurance exercises. His ability to walk increased but his VC remained less than 700 ml and he still had no ability to breathe independently. With inadequate strength to propel himself in a standard wheelchair carrying a ventilator, and not wishing to use a motorized wheelchair, a rolling walker with a tray was used.

After a rehabilitation stay of 60 days the patient was discharged home independent in ADL, transfers, automobile use, and he could ambulate greater than 400 feet using mouthpiece IPPV with the rolling walker. He maintained normal ventilation and SaO_2 only while using his ventilator but he returned to full-time employment as an accountant despite using non-invasive IPPV continuously. He has now been working and living at home for the last 6 years. He has had no further hospitalizations or 'asthma' episodes and has discontinued all medications. His VC has increased to 1050 ml and he now can tolerate 15-180 min periods of ventilator-free breathing before becoming dyspneic and hypercapnic.

This patient demonstrated that:

1. Non-invasive IPPV can be used as an alternative to tracheostomy IPPV for long-term 24-h ventilatory support.
2. Except when there is premorbid chronic bronchitis, chronic aspiration, or other reasons for chronic airway hypersecretion, the hypersecretory state of the airways associated with tracheal intubation is reversed by decanulation and site closure.
3. Non-invasive IPPV and oximetry biofeedback can be used as an alternative to conventional ventilator weaning strategies provided that ventilatory drive is not suppressed by oxygen therapy, sedatives, or narcotics. The patient simply takes fewer and fewer assisted breaths as needed rather than following a more constraining and imprecisely derived clinician's schedule.
4. Wheezing that can be mistaken for asthma can be due to peribronchial edema that is secondary to chronic alveolar hypoventilation and is reversible by increasing thoracic pressures and providing ventilatory support by IPPV.
5. Despite having GAH for over 10 years, he was inappropriately weaned from ventilator use on three previous occasions. Hypoventilation was permitted because his clinicians were unfamiliar with non-

invasive alternatives to tracheostomy, an option the patient had repeatedly refused.

6. Autonomous breathing ability can decrease rapidly following tracheostomy because of inspiratory muscle deconditioning, induction of airway hypersecretion and bronchial mucus plugging, and the tendency for respiratory chemotaxic centers to reset, bicarbonate levels to decrease, and frequent patient requests to increase ventilation despite having low $PaCO_2$.

11. Conclusion

The failure to make timely appropriate management decisions often leads to episodes of acute respiratory failure and unnecessary hospitalizations, endotracheal intubations, bronchoscopies, and tracheostomies. The use of these invasive approaches adversely affects quality of life and can increase the risk of pulmonary complications and mortality for post-poliomyelitis patients *[47]*. Episodes of acute respiratory failure which most often result from otherwise benign upper respiratory tract infections can be reversed by the timely use of non-invasive respiratory muscle aids.

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