

46. Meyer TJ, Pressman MK, Benditt J, et al: Air leaking through the mouth during nocturnal nasal ventilation. Effect on sleep quality. *Sleep* 20:561-569, 1997.
47. Bach JR, Alba AS: Sleep and nocturnal mouthpiece IPPV efficiency in post-polio myelitis ventilator users. *Chest* 106:1705-1710, 1994.
48. Wysocki M, Tric L, Wolff MA, et al: Noninvasive pressure support ventilation in patients with acute respiratory failure. *Chest* 103:907-913, 1993.
49. Nakayama T, Saito Y, Yatabe K, et al: The usefulness of tracheostomy in Duchenne muscular dystrophy ventilated by a chest respirator [in Japanese]. *Rinsho Shinkeigaku* 39:606-609, 1999.
50. Takasaki Y, Kamio K, Okamoto M, et al: Altered arousability in non-REM sleep during sleep progression in patients with moderate to severe obstructive sleep apnea. *Am Rev Resp Dis* 147:A513, 1993.
51. Bach JR, Alba AS, Mosher K, Delarbar A: Intermittent positive pressure ventilation via nasal access in the management of respiratory insufficiency. *Chest* 92:168-170, 1987.
52. Jounieaux V, Parreira VF, Delguste P, et al: Nasal mask pressure waveform and inspiratory muscle recruitment during nasal assisted ventilation. *Am J Respir Crit Care Med* 155:2096-2101, 1997.
53. Parreira VF, Delguste P, Jounieaux V, et al: Effectiveness of controlled and spontaneous modes in nasal two-level positive pressure ventilation in awake and sleep normal subjects. *Chest* 112:1267-1277, 1997.
54. Parreira VF, Delguste P, Jounieaux V, et al: Glottic aperture and effective minute ventilation during nasal two-level positive pressure ventilation in spontaneous mode. *Am J Respir Crit Care Med* 154:1857-1863, 1996.
55. Delguste P, Aubert-Tulkens G, Rodenstein DO: Upper airway obstruction during nasal intermittent positive pressure ventilation in sleep. *Lancet* 338:1295-1297, 1991.
56. Sanna A, Veriter C, Stanescu D: Expiratory supraglottic obstruction during muscle relaxation. *Chest* 108:143-149, 1995.
57. Malhotra A, Pillar G, Fogel R, et al: Upper-airway collapsibility: Measurements and sleep effects. *Chest* 120:156-161, 2001.
58. Bach JR, Kang SW: Disorders of ventilation: weakness, stiffness, and mobilization. *Chest* 117:301-303, 2000.
59. McCool FD, Mayewski RF, Shayne DS, et al: Intermittent positive pressure breathing in patients with respiratory muscle weakness: Alterations in total respiratory system compliance. *Chest* 90:546-552, 1986.
60. Kang SW, Bach JR: Maximum insufflation capacity. *Chest* 118:61-65, 2000.
61. Hultgren AC, Fugl-Meyer AR, Jonasson E, Bake B: Ventilatory dysfunction and respiratory rehabilitation in post-traumatic quadriplegia. *Eur J Respir Dis* 61:347-356, 1980.
62. Mier A, Laroche C, Agnew JE, et al: Tracheobronchial clearance in patients with bilateral diaphragmatic weakness. *Am Rev Respir Dis* 142:545-548, 1990.

6 — Home Mechanical Ventilation for Neuromuscular Ventilatory Failure: Conventional Approaches and Their Outcomes

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Tradition nicht anders sei als Schlämperei. (Tradition is nothing other than negligence.)

Gustav Mahler

CONVENTIONAL STRATEGIES

Conventional respiratory management strategies for patients with neuromuscular conditions are invasive and reactive rather than noninvasive and proactive. They essentially ignore the problem until the inevitable occurrence of respiratory failure, at which point the patient seeks help in a local emergency department or dies before getting there. Current strategies for managing progressive respiratory muscle insufficiency include the following:

1. Treating ventilatory insufficiency with narcotics, sedatives, and supplemental oxygen to lighten and hasten the pathway to the grave while obtaining a "living will" or "informed consent" that prohibits the use of "heroic measures," generally intubation or tracheotomy, or simply mandates no treatment at all.
2. Treating ventilatory insufficiency and episodes of acute ventilatory failure as though they were respiratory insufficiency with some combination of oxygen, medications, chest physical therapy, airway suctioning, and, ultimately, intubation and tracheostomy.
3. "Prophylactic" tracheostomy.
4. Using nocturnal positive inspiratory pressure and positive end-expiratory pressure (PIP + PEEP) at low pressure spans that are ultimately inadequate to prevent ventilatory failure.

Easing and Hastening Death: Not-so-benign Neglect

Prescribing oxygen therapy with or without morphine is an especially popular approach with clinicians who lack knowledge of noninvasive respiratory aids and preach "therapeutic nihilism."¹ This approach decreases dyspnea while hastening death.^{2,3} Often, without consulting the patient, the physician judges the patient's quality of life to be unacceptable and the disease terminal, ignores options that prevent respiratory complications, and biases the family against ventilator use, which the physician associates with tracheostomy.⁴ This approach emphasizes "palliation" but results in hopelessness and thus augments mental

anguish. Either patients die from carbon dioxide narcosis, or an intermittent chest could result in pneumonia and acute respiratory failure.^{6,6} When patients are desperately short of breath, despite having a living will, they often agree to be intubated and ultimately undergo tracheotomy.

Treating Ventilatory Insufficiency Like Respiratory Insufficiency



If the only tool one knows is a hammer, everything is treated like a nail.



Conventional approaches to managing ventilatory insufficiency are identical to those for respiratory insufficiency and include medical therapy typical for patients with chronic obstructive pulmonary disease (COPD). The goals of medical therapy can be to increase diaphragm contractility, dilate the airways and facilitate airway secretion clearance, improve ventilatory drive, and improve oxygenation without exacerbating hypercapnia. Unfortunately, no medical therapy has been shown to be effective and safe for any of these goals in patients with neuromuscular disease (NMD). In a review of 25 studies of patients with musculoskeletal and neuromuscular respiratory insufficiency, methylxanthine administration was not found to alleviate diaphragm fatigue.⁷ Indeed, in the presence of hypercapnia and hypoxia, theophylline appeared to increase or delay recovery from diaphragm fatigue, and side effects were common.⁸ I have observed two patients who suffered generalized seizures due to long-term methylxanthine therapy, from which they had derived no benefit. Bronchodilators, too, are often used on a long-term basis with no subjective or objective benefits. Bronchodilators often cause anxiety and by increasing heart rate can exacerbate cardiac dysfunction for patients with NMD, who often have severe cardiomyopathies. Bronchodilators can even increase airway secretion production.

Low-flow oxygen in combination with protriptyline⁹ or clomipramine¹⁰ has been used to treat sleep hypoxia in this patient population. However, these medications appear to decrease hypoxia by suppressing rapid-eye-movement (REM) sleep.⁹ Any resulting increase in fatigue may increase the risk of pulmonary complications. Furthermore, the anticholinergic effects of protriptyline preclude regular use and may be hazardous for patients with cardiomyopathy, who often have decreased cardiac reserve and cardiac conduction abnormalities. Other medications such as doxapram hydrochloride, acetazolamide, medroxyprogesterone, and almitrine have not been shown to benefit patients with NMD.^{11,12}

Although first used to treat lung disease in 1924,¹³ oxygen therapy became widely accepted for treating hypoxia only in the mid-1960s. Subsequently, oxygen therapy has been shown to improve significantly the prognoses of patients who suffer primarily from severe respiratory impairment. This success and general familiarity with oxygen therapy have led to its use for treating hypoxia due to ventilatory impairment. However, considering the pathophysiology of ventilatory impairment (see Chapter 2), such patients fare poorly because hypercapnia is exacerbated. Oxygen supplementation results in a significantly higher incidence of pneumonias and hospitalizations and prolonged length of stay compared with untreated patients or patients treated with respiratory muscle aids.⁵ In addition, because of its relatively high pressure gradient for absorption in comparison with nitrogen, supplemental oxygen can cause and help to maintain atelectasis.¹⁴ Most patients with neuromuscular weakness who become comatose from carbon dioxide narcosis do so while receiving oxygen supplementation. Although most conventionally managed patients avoid carbon dioxide narcosis, they inevitably undergo tracheotomy or die from airway secretion retention during chest infections because of failure to teach and equip them to use mechanically assisted coughing (MAC).¹⁵ Attempts to suction patients' airways via the nose

or mouth are rarely effective and extremely uncomfortable and may further impair already severely impaired breathing. There is even less chance to enter the left mainstem bronchus in this manner than when suctioning via a tube. Lung suctioning via the upper airway should rarely if ever be required or used as an alternative to mechanically assisted coughing.

Prophylactic Tracheotomy

Because a higher rate of complications is associated with emergency than elective tracheotomy and because respiratory failure is inevitable for conventionally managed patients, physicians unfamiliar with respiratory muscle aids or the indications for tracheostomy¹⁶ (see Chapter 7) may recommend it prophylactically.¹⁷ Most patients, however, appropriately refuse this option. In general, prophylactic tracheotomy may be appropriate only for patients with severe bulbar amyotrophic lateral sclerosis (ALS) and the rare adult patient with maximally assisted peak cough flows less than 160 L/min and SpO₂ persistently less than 95% (see Chapter 7).

Low-Span PIP + PEEP (Bilevel Positive Airway Pressure)

Modern therapy with continuous positive airway pressure (CPAP) was described in 1976.¹⁸ As the widespread prevalence of obstructive sleep apnea syndrome (OSAS) became appreciated in the 1980s, CPAP was used to maintain upper airway patency. CPAP pneumatically splints open the airway and increases functional residual capacity (FRC). CPAP, however, is ineffective for patients whose hypercapnia is based on absolute or relative inspiratory muscle weakness because it does not directly assist inspiratory muscle function. Although this point may appear to be obvious, it was confirmed by a recent study of pressure support ventilation vs. CPAP.¹⁹

In pressure support ventilation, the ventilator delivers a preset inspiratory pressure to assist spontaneous breathing efforts. Most ventilators permit patient triggering with a set back-up rate. With pressure control ventilation, preset time-cycled inspiratory and expiratory pressures are delivered at a controlled rate with adjustable inspiratory-to-expiratory ratios. Ventilators can cycle to expiration when they sense a fall in inspiratory flow below a threshold value or at a pre-set time. Flow triggering appears to be more sensitive than pressure triggering.²⁰

The BiPAP machine (Respironics, Inc., Murrysville, PA) is a pressure-cycled ventilator. It was developed because of the frequent ineffectiveness of CPAP and the difficulties of tolerating high CPAP levels. The quick acceptance and widespread use of the BiPAP device were due to its light weight, inspiratory pressures greater than 20 cmH₂O, PEEP (equivalent to expiratory positive airway pressure [EPAP]) capabilities, similarity to CPAP (CPAP plus inspiratory positive airway pressure [IPAP]), and its relatively low cost compared with volume-cycled ventilators.

The BiPAP-ST, Synchrony, and BiPAP Harmony (Respironics, Murrysville, PA); the PB335 Respiratory Support System and the Quantum (Mallinckrodt, Pleasanton, CA); the ResMed VPAP-STII (ResMed, San Diego, CA); and other similar units are essentially pressure-limited blowers with pressures up to 22-40 cmH₂O and plateauing of delivered volumes at the higher pressures. The IPAP and EPAP are adjusted manually, and the ventilator delivers air flows to achieve the set pressures.

The failure of all of the ventilator-delivered air to enter the lungs (i.e., insufflation leakage) is due to both mask leakage and leakage from the nose for mouthpiece ventilation or from the mouth for nasal ventilation. Increased airway resistance (e.g., due to mucus plugging) results in a decrease in delivered volumes. The inspiratory pressure minus the PEEP (IPAP-EPAP difference) or pressure span is essentially the amount of inspiratory muscle assistance that the patient receives. Excessive mask leakage can impair the expiratory trigger cycling mechanism and result in prolonged duration of the inspiratory phase, auto-PEEP, and patient-ventilator dyssynchrony. In the presence of air leaks, time-cycled expiratory triggers facilitate patient-machine synchrony more effectively than flow-cycled expiratory triggers.²¹

due to inspiratory muscle weakness.²⁶ CPAP is thought to be the primary treatment for sleep-disordered breathing, and patients are treated with CPAP or low-span PIP + PEEP. Inappropriately low spans are used because of confusion in the interpretation of polysomnography results, failure to appreciate the need for respiratory muscle rest, unjustified fear of barotrauma (see Chapter 7), and physicians' much greater familiarity with CPAP, OSAS, and lung disease than with neuromuscular weakness in patients with otherwise normal lung tissues.

NOCTURNAL-ONLY NASAL VENTILATION

Patients with symptoms of ventilatory insufficiency and hypoxia can benefit from nocturnal nasal ventilation whether it be provided as PIP + PEEP or as IPPV.⁵ Nasal ventilation typically relieves fatigue and other symptoms of which patients may have been unaware. When such patients feel better the day after using nocturnal nasal ventilation, they do not need to be prompted to continue use.

Ventilatory insufficiency progresses insidiously. A consensus conference in 1991 suggested the following indications and considerations for instituting nocturnal nasal ventilation in stable patients with Duchenne muscular dystrophy (DMD): rapid speed of disease progression, presence of hypercapnia or end-tidal carbon dioxide exceeding 50 mmHg during sleep, mean nocturnal SpO₂ less than 95%, and symptoms of ventilatory insufficiency. In addition, it was agreed that in the absence of symptoms, nocturnal nasal ventilation is indicated for patients with DMD when PaCO₂ exceeds 45 mmHg and/or PaO₂ is less than 60 mmHg in blood gas samples taken early in the morning, late in the day, or during periods of oxygen desaturation.²⁷ Another consensus conference in 1999 decided that for patients with NMD and chest wall deformities the indications for nocturnal nasal ventilation were symptomatic hypoventilation, PaCO₂ greater than 45 mmHg, nocturnal SpO₂ less than 89% for at least five consecutive minutes, or maximal inspiratory pressures less than 60 cmH₂O or forced vital capacity less than 50% of predicted.²⁸ For patients with COPD, indications were listed as symptoms plus one of the following: PaCO₂ greater than 55 mmHg, PaCO₂ of 50–54 mmHg, nocturnal SpO₂ less than 89% for at least five continuous minutes while the patient is receiving oxygen therapy of 2 L/m or more, or PaCO₂ of 50–54 mmHg and hospitalizations related to recurrent episodes of hypercapnic respiratory failure.²⁸ Some of these indications (e.g., speed of progression) are irrelevant, and others are draconian. The only true indication is relief of symptoms, although before treatment an increase from normal in episodes of nocturnal oxyhemoglobin desaturation is usually present.²⁹

Patients tend to be symptomatic when diurnal hypercapnia causes the SpO₂ to decrease below 95%. However, even when untreated, many patients can tolerate hypercapnia for years—or until they have an intercurrent chest cold—when diurnal SpO₂ is normal. Although nocturnal low-span PIP + PEEP can benefit mildly affected patients, clinicians too often prescribe oxygen or recommend tracheotomy instead of increasing the spans or switching to the use of volume-cycled ventilators for daytime IPPV via a mouthpiece when low-span pressure assistance is no longer adequate.^{27,30} Once the patient is intubated or a tracheostomy tube is placed, he or she receives appropriate tidal volumes at adequate peak inspiratory pressures (usually 20–25 cmH₂O) to maintain normal lung ventilation and facilitate coughing. Ironically, if these pressures had been provided noninvasively, the patient would not have needed invasive management.

Along with suboptimal PIP + PEEP spans, conventional management includes medical interventions, chest physical therapy, and occasionally IPPB, used for inadequate periods and at inadequate pressures, to expand fully the lungs and chest walls or to support or rest inspiratory muscles. Chest physical therapy, including chest percussion and postural



FIGURE 1. A woman with spinal muscular atrophy and chronic respiratory muscle insufficiency who was born in 1959. She used nasal IPPV via a standard CPAP mask for nocturnal ventilatory assistance from 1971 until March of 1988, when she underwent tracheotomy during hospitalization for a respiratory tract infection. She died of pneumonia in April of 1990.

Nocturnal-only delivery of PIP + PEEP at low spans (IPAP minus EPAP less than 10 cmH₂O) using commonly available, inexpensive, generic CPAP masks as patient-ventilator interfaces provides a small pressure boost to assist inspiratory effort (Fig. 1). This approach is widely used even in the absence of diurnal hypercapnia or symptoms of ventilatory insufficiency. Although it can be helpful early on, low spans of typically 5–7 cmH₂O allow inadequate rest of respiratory muscles and insufficient assistance to inspiratory muscle function to sustain patients as inspiratory muscle weakness progresses.

Increasing the EPAP without increasing the IPAP decreases the span. EPAP is conventionally used even though it does not benefit patients with predominantly inspiratory muscle impairment. The greater the level of EPAP that is used, the greater the level of IPAP that must be used to provide the same level of inspiratory assistance and the greater the resulting intrathoracic pressure. It has been shown that increases in intrathoracic pressure cause dose-dependent decreases in cardiac stroke volume and cardiac output.²² This scenario can be hazardous for ventilator users with cardiomyopathies.

Even the maximal spans of most commonly used machines generally deliver less than 1200-ml volumes to patients with normal lung impedance. These volumes do not permit the cough volumes or flows needed to clear airway secretions or optimally raise voice volumes. When lung impedance is increased, even adequate ventilation may not be achieved. Thus, the fact that pressure-cycled ventilators are widely used at suboptimal spans plays a large role in the development of ventilatory or respiratory failure during intercurrent chest infections in patients with neuromuscular weakness.²³

Nocturnal low-span PIP + PEEP has become part of the conventional management of patients with NMD. A recent survey of MDA clinic directors indicated that nocturnal PIP + PEEP was used in about 80% of clinics. Unfortunately, physicians confuse the nocturnal use of low-span PIP + PEEP with the use of “noninvasive ventilation.”^{32,34,35}

clear advantage could be appreciated using either system. Hill concluded that "the choice between the two comes down to clinician preference and a consideration of the particular advantages and disadvantages."³⁷

Advantages and disadvantages of using volume-cycled and pressure-cycled ventilators are listed in Table 1. Because the currently available pressure-cycled units are limited to pressures of less than 40 cmH₂O, they can be inadequate for managing conditions characterized by poor pulmonary compliance or high work of breathing, as in patients with severe scoliosis or obesity. On the other hand, PIP + PEEP can be useful for managing COPD and sleep-disordered breathing. Even low-span PIP + PEEP can effectively assist inspiratory muscle function for emphysematous patients with hypercompliant lungs. The positive pressures also can stabilize the airway in the event of concurrent obstructive sleep

TABLE 1. Pressure Assist Vs. Portable Volume Ventilators

Advantages of volume ventilators

1. Can deliver higher volumes and at potentially higher pressures, as needed, for patients with poor lung compliance
2. Can adjust flow rates for comfort
3. Uses 3-8 times less electricity for comparable air delivery, permitting greater patient mobility for the same battery capacity
4. Operates more quietly
5. Less mean thoracic pressures create less untoward hemodynamic effects on cardiac preload; particularly hazardous for patients with cardiomyopathies
6. Permits air-stacking to obtain maximal insufflations for increasing dynamic pulmonary compliance as well as for raising voice volume and increasing cough flows
7. Can be used to operate intermittent abdominal pressure ventilators as well as for noninvasive IPPV
8. Has alarm systems that can facilitate effective use of nocturnal noninvasive IPPV

Disadvantages of volume ventilators

1. Heavy
2. Annoying alarms (low-pressure alarm can be eliminated by setting alarm to minimum and using flexed mouthpiece, bacterial filter, or a regenerative humidifier to create sufficient back pressure)
3. Needlessly complicated; ventilators with fewer modes should be available

Advantages of BIPAP

1. No annoying alarms
2. Light weight
3. Lower cost
4. Can compensate to some extent for small insufflation leaks

Disadvantages of PIP + PEEP

1. Inability to air stack
2. Fixed, high initial flow rates can cause mouth drying and gagging, especially with insufflation leakage, and arousals from sleep
3. High power utilization limits patient mobility
4. Inadequate pressure generation capabilities for some patients
5. Discomfort and increased thoracic pressures due to unnecessary expiratory positive airway pressure
6. The gauge of pressure-cycled devices is less useful for feedback about insufflation leakages
7. No alarms to facilitate effective nocturnal IPPV for some patients
8. Noisier
9. EPAP unnecessary for most patients
10. Ventilatory assistance at higher mean intrathoracic pressures than with the use of volume-cycled ventilators
11. Significant carbon dioxide rebreathing occurs; can be corrected by using a nonbreathing valve at the cost of greater expiratory resistance¹⁷
12. EPAP can make eating difficult or hazardous for 24-hour ventilator users

and plasma catecholamine concentrations.³¹ These therapeutic measures do not address the fundamental need to support inspiratory muscles and increase cough flows. Patients receiving chest physical therapy who are able to increase minute ventilation do so by about 35%, and those not able to increase minute ventilation require ventilatory assistance to the same extent.³¹ Failure to assist the cough must inevitably result in respiratory failure for patients with NMD. Even now airway mucus accumulation tends to be considered a "complication" of the use of noninvasive ventilation rather than the failure of the clinician to institute insufflation therapy and MAC.³²

A recent review of nocturnal nasal ventilation inadvertently provided a good example of common misunderstandings related to the use of noninvasive IPPV. The review notes that "patients with severe neuromuscular weakness, prominent pulmonary secretions, or significant bulbar dysfunction" may require tracheostomy. This review ignored the fact that patients with no measurable vital capacity successfully use noninvasive IPPV; it failed to consider assisted coughing to clear secretions; and it failed to quantitate bulbar muscle dysfunction (see Chapter 7). The review arbitrarily recommended normalizing end-tidal carbon dioxide by "no more than 15 mmHg." It advised that ventilatory assistance be initiated in a monitored environment, even though we have instituted noninvasive IPPV for hundreds of patients, many of whom eventually became 24-hour users, in the outpatient clinic and home settings. The review notes that polysomnography and supplemental oxygen are required when PIP + PEEP does not increase SpO₂ to greater than 90%.³³ Both suggestions are harmful and dangerous for patients with primarily ventilatory impairment.

VOLUME-CYCLED VENTILATORS

The expiratory volume alarm of intensive care volume ventilators makes them impractical for the delivery of noninvasive IPPV, and they are usually not sufficiently portable for home use. Nasal ventilation has been most frequently described using portable volume-cycled ventilators.³⁴⁻³⁶ Portable volume-cycled ventilators, however, are most often conventionally used for providing long-term tracheostomy rather than noninvasive IPPV.

Volume-cycled ventilators—such as the PLV-100 (Respironics, Inc., Murrysville, PA), the Achieva, LP-10, and LP-20 (Mallinckrodt, Pleasanton, CA), and LTV-900, a volume- or pressure-cycled machine (Pulmonetic Systems Inc., Colton, CA)—can generally deliver volumes up to 2500 ml. Typically, control, assist-control, and synchronized intermittent mandatory ventilation (SIMV) modes can be used. The most commonly used mode for home noninvasive ventilation is assist-control, which delivers set volumes of air are delivered, triggered by the patient's inspiratory effort. A back-up rate provides for air delivery in the absence of effort. For volume-cycled ventilators, the volume to be delivered to the ventilator circuit—once set—remains constant regardless of the insufflation leakage, pulmonary compliance, and airway resistance. However, because insufflation leakage can change from breath to breath, the volumes delivered to the lungs change from breath to breath. The volumes delivered to the lungs correlate with ventilator gauge pressures. Thus, the pressures indicated on the volume ventilator pressure gauge vary, depending on ventilator-delivered volumes, insufflation leakage, and lung impedance. Volume-cycled ventilators also have low- and high-pressure alarms, sensitivity controls that permit the patient to trigger ventilator-delivered breaths, and flow-rate adjustments. They can be pressure-limited. Like pressure-cycled ventilators, by increasing the delivered volumes and lowering the high-pressure alarm.

VOLUME-CYCLED VS. PRESSURE-CYCLED VENTILATORS

The following table was attempted to compare the efficacy of ventilatory assistance

apneas or bulbar muscle dysfunction with airway collapse. The PEEP can decrease the work of breathing by relieving the effort required to reverse the expiratory flows associated with air trapping, thus countering the auto-PEEP effect. In general, depending on settings, the work of breathing can be equally decreased during pressure-cycled and volume-cycled mechanical ventilation.³⁸ Because patients cannot air-stack when using pressure-cycled ventilators, their long-term use in patients with NMD is suboptimal unless the patient is equipped with the means to expand the lungs and assist the cough.

Currently available pressure-cycled ventilators can weigh as little as 5.5 lb. They are becoming lighter, smaller, and more quiet. The Harmony, for example, weighs only 2.5 kg, operates quietly, and can provide IPAP up to 30 cmH₂O. It uses only 10% more amperage than typical portable volume-cycled ventilators and can operate most of the day on a fully charged external battery. The ResMed VPAP-STII is 1 kg heavier but provides IPAP up to 40 cmH₂O and therefore may be more useful for deep insufflation therapy in patients who do not have Cough Assist devices or volume-cycled ventilators and who are unable to air-stack via a manual resuscitator. These machines are useful for air delivery without high- and low-pressure alarms. This feature can be an advantage or a disadvantage, depending on the particular patient. Such devices do not have internal batteries, use more electricity than volume-cycled ventilators, and, therefore, run for shorter periods on an external battery source when they are equipped to do so.

The LTV-900 is a 12-pound, versatile, lap-top sized ventilator. Air flows are created by turbine, and the ventilator can be volume- or pressure-cycled. The LTV-900, however, is noisier than other volume-cycled ventilators, it is the most expensive portable ventilator on the market, and frequent technical glitches have been reported. This and other small units, however, may be ideal for patients who are able to walk despite ventilator dependence (see Chapter 15) as well as for others who need small, light units to optimize mobility.³⁹

Air-stacking to facilitate coughing, to increase voice volume, and to expand the lungs optimally is not possible with pressure-cycled ventilators. Depending on the unit and lung impedance characteristics, most patients cannot obtain the minimal 1.5 L insufflation volumes generally needed for an effective assisted cough.⁴⁰ In addition, despite increases in minute ventilation and reductions in respiratory effort, PIP + PEEP does not always reduce PaCO₂—perhaps because of the carbon dioxide rebreathing inherent with the standard exhalation device (Whisper-Swivel, Respirationics, Inc., Murrysville, PA). With the fixed-resistance exhalation Whisper-Swivel, the patient rebreathes air exhaled into the ventilator tubing, as dead-space ventilation is increased.⁴¹ Elimination of rebreathing would require a completely unacceptable EPAP level of 8 cmH₂O. The problem can be solved by using nonrebreather valves and exhalation plateau valves. However, nonrebreather valves can malfunction if valve materials stiffen or secretions become impacted within it, and exhalation plateau valves are not yet commercially available. A generally better alternative is to switch to volume-cycled ventilation at appropriate settings. The use of PIP + PEEP by clinicians with little experience in noninvasive aids almost invariably leads to failure to intervene appropriately during intercurrent chest infections.

CONVENTIONAL EXTUBATION

Like any emergency department patient presenting with respiratory distress, intubated patients using ventilatory support typically receive supplemental oxygen along with bronchodilators, mucolytics, chest physical therapy, and, possibly, sedation. Ventilator-weaning attempts are made with some combination of assist-control ventilation, SIMV, pressure support, PEEP, and supplemental oxygen. Occasionally, periods of ventilator-free breathing are tried with the patient receiving CPAP or oxygen and humidification by T-piece. With these approaches, "weaning schedules" are imposed on the patient. This approach usually involves one of two problems: either it causes anxiety because the patient is not ready to breathe