Positive Pressure Ventilation*
Alternate Modes
Catherine S. H. Sassoon, M.D., F.C.C.P.

(Chest 1991; 100:1421-29)

Alternate modes of low frequency positive pressure ventilation have been developed over the last two decades. Compared with conventional positive pressure ventilation (CPPV), these alternate modes may do the following: (1) correct gas exchange abnormalities when CPPV fails; (2) reduce the risk of barotrauma; (3) cause less hemodynamic compromise; (4) enhance patient comfort; or (5) facilitate weaning. Some of these alternate modes have already been incorporated in commercially available microprocessor-controlled ventilators.

This review will first describe each of the alternate modes, then discuss laboratory and clinical studies, the limitations of and current status of each mode (Table 1).

Inverse Ratio Ventilation (IRV)
IRV was first introduced and utilized by Reynolds in 1971 in treating neonates with hyaline membrane disease.

Description
IRV is a pressure-controlled ventilation in which inspiratory time exceeds expiratory time. A prolonged positive pressure is applied to progressively recruit collapsed alveoli, while the short expiratory time is intended to prevent the alveoli to recollapse. Although IRV can be applied with volume-controlled ventilation, pressure-controlled ventilation is more commonly employed. This has the advantage of adjusting airway pressure to enable the opening of collapsed alveoli (critical alveolar pressure).

With pressure-controlled IRV, the preset variables are peak airway pressure (Paw), the ratio of inspiratory to expiratory time (I:E ratio), respiratory rate (F), and the applied (extrinsic) positive end-expiratory pressure (PEEP). Neither peak inspiratory flow rate nor flow waveform are preset variables. A typical flow waveform is shown in Figure 1. The positive pressure with the decelerating flow waveform allows alveolar units with slow time constants to fill, thereby improving alveolar ventilation. For a given level of oxygenation, the preset peak Paw is lower than that with CPPV, but the mean Paw is inevitably higher. With pressure-controlled IRV, tidal volume (VT) is determined by the patient's respiratory system mechanics. However, two additional factors influence VT. These are the I:E ratio and F. The higher the I:E ratio and F, the shorter the expiratory time (Te). This usually induces a higher intrinsic PEEP (PEEPi). Thus, for a given respiratory system resistance and compliance, the higher the PEEPi, VT is smaller with a given peak Paw. Despite this potential VT limitation and a consequent decrease in total minute ventilation (VE), PaCO₂ can be maintained satisfactorily with the successful use of IRV. This is most likely the result of a lower dead space-to-tidal volume ratio (VD/VT). The improvement in oxygenation is related to increases in both mean Paw and PEEPi or end-expiratory lung volume. At present there are no established guidelines concerning the level of peak Paw to be set when switching.

<table>
<thead>
<tr>
<th>Table 1—Alternate Modes of Positive Pressure Ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Inverse ratio ventilation (IRV)</td>
</tr>
<tr>
<td>2. Airway pressure release ventilation (APRV)</td>
</tr>
<tr>
<td>3. Pressure support ventilation (PSV)</td>
</tr>
<tr>
<td>4. Proportional assist ventilation (PAV)</td>
</tr>
<tr>
<td>5. Mandatory minute volume (MMV)</td>
</tr>
</tbody>
</table>

*From the Departments of Medicine, Veterans Affairs Medical Center, Long Beach, and the University of California, Irvine. Supported by the Department of Veterans Affairs Medical Research Service.

Reprint requests: Dr. Sassoon, Pulmonary and Critical Care Section (111F), VA Medical Center, Long Beach 90822
from CPPV to IRV. One study set initial peak Paw on IRV equal to one half of that observed with CPPV. Other studies adjusted peak Paw to produce a similar Vt as that obtained during CPPV.

With IRV, both the I:E ratio and F have to be adjusted to obtain the desired level of PEEPi and Vt. The desired level of PEEPi is that level at which PaO\(_2\) is adequate with the lowest possible FIO\(_2\). This level of PEEPi should not cause hemodynamic compromise.

Depending on oxygenation, PEEPe may still be required during IRV. In general, PEEPe is set lower than with CPPV. However, the total PEEP, ie the sum of PEEPe and PEEPi, might be extremely high with consequent hemodynamic compromise and small Vt (see above). Because both the degree of PEEPi and mean Paw influence cardiac output, the importance of monitoring total PEEP and mean Paw during IRV cannot be overemphasized. In addition, without the knowledge of total PEEP value, measurement of respiratory system compliance will be underestimated.

**Laboratory and Clinical Studies**

The main objective of IRV is to improve oxygenation in patients with refractory hypoxemia associated with the adult respiratory distress syndrome (ARDS). This objective should be achieved without excessive peak Paw and with minimal adverse effect on cardiac output. However, in patients with severe ARDS, Gattinoni and coworkers demonstrated that oxygenation was similar when they used either IRV or CPPV and PEEPe with a comparable mean Paw. These authors suggested that mean Paw is an important determinant of oxygenation irrespective of the ventilatory modes or PEEPe level.

To date, there has not been any controlled study that establishes a reduced morbidity or mortality with IRV, in comparison with CPPV. Most uncontrolled studies of IRV in adults have reported improved oxygenation. One study did not report mortality, and another study reported a mortality of 45 percent, irrespective of improvement in oxygenation. In the other two studies, a high percentage of patients were unsuccessfully treated with IRV. Tharrat et al reported that oxygenation did not improve in 18 (55 percent) of the 33 episodes that involved the use of IRV. The mortality in this group of patients was 100 percent. For the patients in whom oxygenation did improve, the mortality was still substantial (53 percent). In the study by Andersen, 38 (36 percent) of 105 patients failed IRV and only four of those patients survived (89 percent mortality). However, 48 (72 percent) of the 67 patients who were successfully treated with IRV survived hospitalization. This is the only study that demonstrates some promise for the use of IRV. It remains to be determined whether IRV is superior to CPPV or APV (see below) when mean Paw is maintained the same under those different conditions.

In terms of morbidity, at an I:E ratio of 2:1, Abraham and Yoshihara found no significant effect of IRV on cardiovascular function. Mean Paw was not reported in that study. Cole and coworkers reported a decrease in cardiac output and oxygen delivery when the I:E ratio was set at 4:1, but no significant decrease when the I:E ratio was less than 4:1. At an I:E ratio of 4:1, mean Paw was significantly higher than at a lower I:E ratio. Mean Paw has an important influence on cardiovascular function as well as oxygenation.

In one study, IRV is associated with a 25 percent incidence of pneumothoraces. The authors suggested that the pneumothoraces were associated with the length of time the patient was receiving IRV, rather than with the I:E ratio or mean Paw. However, PEEPi was not measured. This factor might have contributed to the barotrauma. The above incidence of barotrauma is similar to that reported with CPPV and high levels of PEEPi.

**Limitation**

An I:E ratio that is set greater than 1:1 during IRV will impose an unnatural breathing pattern and cause discomfort. For this reason, the patient will require sedation and/or paralysis. The fact that lung compliance might change significantly during the initial application of IRV necessitates the close monitoring of the patient and frequent adjustment of ventilator variables.
Current Status

Since the editorial comment of Duncan et al in 1987, there has not been any significant progress to alter the status of IRV from an experimental to a commonly applied ventilatory mode in patients with ARDS. Controlled data showing the benefit of IRV over CPPV and PEEP remain insufficient and, despite improved oxygenation, mortality owing to nonrespiratory causes in ARDS is substantial. In addition, the different ventilator modes have not had any significant impact on mortality.

Airway Pressure Release Ventilation (APRV)

APRV was first introduced by Downs and Stock in 1987. The principal goal of APRV is similar to IRV, i.e., to improve oxygenation by opening and stabilizing collapsed alveoli. APRV also augments alveolar ventilation without excessive peak Paw.

Description

APRV is a continuous positive airway pressure (CPAP) ventilation that intermittently decreases or "releases" the airway pressure from the preset CPAP level to either a lower level or ambient pressure as the airway pressure release valve opens. With the reduction of airway pressure to a lower level, gas is allowed to leave the lungs and CO₂ is eliminated. APRV differs from CPPV in that there is a decrease rather than an increase in airway pressure during an assisted breath. Figure 2 shows the characteristics of the Paw waveforms during APRV. A stand-alone APRV system consists of the following: (1) a continuous high-flow CPAP system that employs a threshold resistor expiratory valve; (2) a pressure release valve that is capable of attaining the fully opened or closed position in less than 10 ms, allowing airway pressure to fall and rise rapidly, respectively; and (3) a timer. The preset variables in APRV are the CPAP level provided by the continuous high-flow system, the level to which CPAP is reduced during release, the frequency, and the duration of the airway pressure releases. The duration of the pressure release is less than 1.5 s. The amount of machine assistance depends on the gradient between the CPAP level and the level to which airway pressure is reduced, the frequency of pressure releases, and the patient's respiratory system mechanics.

Laboratory and Clinical Studies

In anesthetized dogs with normal lungs, APRV is comparable to CPPV in maintaining gas exchange. In moderate to severe oleic acid–induced lung injury, when mean Paw was maintained the same during APRV as that during CPPV with PEEP, APRV resulted in a significantly higher PaO₂, lower PaCO₂, and lower peak Paw. The lower PaCO₂ was postulated to be the result of lower physiologic dead space ventilation since V̇e was similar during both ventilatory modes. In another study, when PaCO₂ was maintained constant during both ventilatory modes, PaO₂ was similar, but peak and mean Paw were significantly lower and, consequently, cardiovascular function was well-maintained during APRV. In a neonatal lamb model of acute lung injury, Martin and colleagues evaluated the effects of APRV in comparison to both CPAP and CPPV on gas exchange and hemodynamics. Mean Paw was maintained the same on all the ventilatory modes. In comparison to CPAP, APRV resulted in a similar PaO₂ and cardiovascular function, and a lower PaCO₂. In comparison to CPPV in which PaCO₂ was maintained equal to that during APRV, APRV resulted in a similar PaO₂. However, during APRV, peak Paw was significantly lower, and both cardiac output and mean arterial pressure were significantly higher than those with CPPV.

Studies involving humans are still quite limited. In patients with postcoronary revascularization surgery following ventilation with CPPV, APRV was applied until they were weaned. PaCO₂ was maintained the same as that during CPPV. APRV resulted in a similar PaO₂ and hemodynamic function, but evinced a significantly lower peak Paw (11 ± 2 cm H₂O, SD) than during CPPV (38 ± 6 cm H₂O). In a multicenter study, the effects of APRV and CPPV on gas exchange were compared in 19 patients with moderate to severe acute lung injury. Mean Paw was maintained the same during both modes of ventilation. During APRV, PaCO₂ (42 ± 6 vs 50 ± 6 mm Hg) and peak airway pressure (24 ± 12 vs 51 ± 15 cm H₂O) were significantly lower than those during CPPV. On the other hand, PaO₂ and cardiovascular function were similar. It was not clear whether the decrease in PaCO₂ was due to augmented...
Positive pressure ventilation (Catherine S. H. Sassoon)

\[ \dot{V}_E \text{ or to a decrease in physiologic dead space ventilation, or to a combination of the two. Vrillon and coworkers}\] evaluated the effect of APRV in patients who had mild lung injury with and without impaired chest wall mechanics. CPAP and APRV were applied in random order while mean Paw was maintained the same during both ventilatory modes. In the patients with impaired chest wall mechanics and hypercapnia, APRV increased \( \dot{V}_E \) and decreased PaCO\(_2\) significantly, while in the patients without impaired chest wall mechanics and with normocapnia, \( \dot{V}_E \) and PaCO\(_2\) were similar. The effects of these ventilatory modes on PaO\(_2\) were not reported. The authors did not compare APRV with CPPV concerning the effect on gas exchange, as has been done in previous studies. It is possible that in mild lung injury, CPPV would be as effective as APRV in improving gas exchange.

Two case reports concerning the use of APRV have also shown the efficacy of APRV in decreasing PaCO\(_2\) while maintaining lower peak Paw in comparison to either CPPV or synchronous intermittent mandatory ventilation (SIMV).\(^{20,24}\) Oxygenation was well-maintained with APRV, but did not seem to differ from that with CPPV.

**Limitation**

Two factors in the APRV system can potentially cause discomfort in the conscious, spontaneously breathing patient. These are the short pressure release time, which may result in an inverse ratio of inspiratory and expiratory time, and the timing of the pressure release with respect to the respiratory cycle (i.e., the pressure release may occur during inspiratory or midexpiratory cycle). However, such discomfort has been reported to be uncommon.\(^{27}\) If it does occur, light sedation might be needed during the first hours of APRV until the patient becomes accustomed to the cycles.\(^{28}\) The APRV system has been incorporated in a commercially available ventilator (Cesar, CFPO, France) in which the opening of the pressure release valve is synchronized with the onset of the patient's expiratory effort.\(^{28}\) It is not yet clear whether synchronization of the pressure release with the onset of expiratory effort will have any significant effect on comfort or gas exchange.

**Current Status**

APRV is still in the investigational stage. From the limited data available, APRV appears effective in improving alveolar ventilation and decreasing peak airway pressure in comparison to CPPV. Oxygenation has not been significantly affected. It is likely that patients with acute lung injury and hypercapnia will benefit from APRV. Theoretically, the efficacy of APRV in improving alveolar ventilation makes it suitable for application in patients with airflow obstruction in whom alveolar hypoventilation is commonly seen. However, the short pressure release time entailed in the use of APRV poses a problem for the application of APRV to this group of patients, unless the pressure release time can be sufficiently prolonged. Yet, the prolonged expiratory time might have a deleterious effect on oxygenation because of the consequent potential for alveolar collapse and instability. A randomized study to evaluate the effects of APRV as a primary means of ventilatory support and CPPV (i.e., assist-control and SIMV modes) on gas exchange, cardiovascular function, morbidity, and mortality in patients with severe lung injury is urgently needed. The place of APRV in patients with airflow obstruction has yet to be determined.

**Pressure Support Ventilation (PSV)**

PSV was incorporated in a commercially available ventilator as early as 1981 (eg, Siemens Servo 900C, Engstrom Erica).\(^4\) However, it did not receive significant attention until 1986 when MacIntyre\(^{26}\) suggested its utility for both reducing the load on respiratory muscles and improving synchrony between ventilator and patient.

**Description**

PSV is a pressure-limited assist mode of ventilation in which the ventilator delivers a preset pressure and allows the patient to interact with the set pressure to determine the ultimate flow, tidal volume, inspiratory time, and frequency.\(^{35,36}\) The patient has to initially trigger a preset pressure sensitivity. Once triggered, a flow of gas sufficient to meet the patient's demand enters the circuit, allowing the system to rapidly approach the preset pressure level. The pressure support level is maintained until the patient's inspiratory flow decreases to a specified level (eg, 25 percent of the peak flow value in the Siemens Servo 900C or 5 L/min in the Puritan-Bennett 7200a) at which time exhalation occurs. Because the patient has to initiate the pressure-limited breath, an intact respiratory drive is required. The degree of patient effort, the pressure support level, and the patient respiratory system impedance determine \( V_t \). Hence, in addition to intact respiratory drive, a relatively stable respiratory system impedance is another important requirement for the use of PSV as the sole ventilatory support.

The degree of ventilatory assist is proportional to the level of preset pressure.\(^{35-37}\) Maximum inspiratory muscle unloading occurs at virtually the maximum inspiratory pressure support level (PSV max). PSV max is defined as the pressure that provides a \( V_t \) of 10 to 12 ml/kg.\(^{36,38}\) Thus, a PS level above PSV max is unnecessary. MacIntyre and Leatherman\(^{39}\) demonstrated that the transition from partial to maximum unloading can be detected from changes in breathing.
pattern. Maximum unloading of the inspiratory muscles is characterized by an increase in \( V_t \) and a decrease in \( F \) from a relatively constant \( V_t \) and \( F \) during the partial unloaded condition.

The preset variables for PSV are the pressure trigger to initiate the breath (pressure sensitivity) and the pressure support level. Recently, MacIntyre and Li suggested that the initial ventilator flow delivery is another important preset variable. The initial flow delivery affects the patient's breathing pattern and synchrony. When initial flow delivery is matched to the patient's ventilatory demand, \( V_t \) is largest, frequency is lowest, and inspiratory time (TI) is longest in comparison with that obtained when initial flow is either in excess of or less than ventilatory demand. Branson and coworkers showed that during PSV max, patients with a high ventilatory demand preferred a high initial flow delivery. Suboptimal initial flow delivery in those patients resulted in tachypnea and dysynchrony. Both MacIntyre and Li and Branson et al. have recommended that capability for ventilator flow adjustment should be incorporated into the existing PSV system. In contrast to initial flow delivery, the fixed flow criterion for the ventilator to cycle off does not affect either breathing pattern or patient synchrony.

Laboratory and Clinical Studies

1. Effect of PSV on the Work of Breathing: Several studies have shown the advantage of PSV over demand-flow (DF) or continuous-flow CPAP in terms of decreasing either the oxygen consumption of respiratory muscles or the diaphragmatic pressure-time product (PTP) of postsurgical patients or patients recovering from acute respiratory failure. In those studies, the pressure support level used was 10 cm H\(_2\)O or higher. Hence, it is not surprising that PSV is superior to CPAP, since the degree of respiratory muscle unloading is proportional to the pressure support level. When 5 cm H\(_2\)O of PSV was compared with DF CPAP, inspiratory muscle work of breathing (W) was significantly reduced with PSV. This suggests that a low level of PSV is adequate to compensate for the W required to open the demand valve. However, when 5 cm H\(_2\)O of PSV was compared with flow-by (FB) CPAP set at 5 cm H\(_2\)O or ambient pressure (0 cm H\(_2\)O), the PTP of the inspiratory muscles was not significantly different. A FB CPAP is a modified continuous-flow CPAP system in which the opening of the pneumatic valve is based on flow sensitivity. Thus, a low level of FB CPAP is comparable to low levels of PSV in unloading the inspiratory muscles.

2. Role of PSV on Weaning: Two studies assessed the efficacy of PSV in facilitating weaning in comparison to other weaning modalities. In postoperative patients with coronary artery bypass grafts, Prakash and Meij randomized the patients into groups weaned either with PSV set to a pressure that produced a \( V_t \) of 10 to 12 ml/kg or with CPPV. The PSV group had the pressure support level reduced over a period of 1 to 3 h to 3 cm H\(_2\)O, following which the patients were extubated. In the CPPV group, the patient required 6 h of mechanical ventilation before extubation. It was not clear whether any attempts were performed to extubate these patients at any time while receiving CPPV. The other study was of patients with flail chest or pulmonary contusion who had failed several weaning attempts with SIMV. The \( V_t \) of the assist breath was set at 10 to 12 ml/kg with the SIMV rate adjusted to maintain normocapnia and a pH greater than 7.35. PSV (mean 9 cm H\(_2\)O) was added to augment \( V_t \) of the spontaneous breaths to greater than 4 ml/kg while maintaining a constant SIMV rate. The average time to extubation following the added PSV was 2.4 days (range, one to seven days). Although in this study PSV appeared to facilitate weaning, no control group was provided, and it is possible that the difficulty of weaning was related to the DF SIMV system.

3. Role of PSV as a Primary Means of Ventilatory Support: PSV appears to provide a degree of ventilatory support that is similar to CPPV. In patients with diffuse lung injury, the efficacy of PSV in maintaining gas exchange equivalent to that of CPPV can be predicted from the extravascular lung water measured during CPPV. Patients with increased extravascular lung water cannot tolerate PSV despite maximum pressure support levels that produce VTs of 12 to 15 ml/kg. This is likely due to an instability of respiratory system mechanics that causes a variable \( V_t \). In patients with pneumonia and acute respiratory failure, Tokioka et al. found PSV to be as successful in maintaining gas exchange as was the assist-control (AC) mode. During PSV, peak Paw was initially set at a level equal to AC. This was subsequently decreased to a level at which the breathing pattern remained regular with F less than 20 breaths per minute. Lower peak Paw and higher \( V_t \) during PSV than during AC suggests that PSV improved respiratory system mechanics. It is possible that this improvement was related to the decelerating flow waveform as compared with the constant flow waveform used in the AC mode. Conversely, in the study of Specht and colleagues, PSV failed to provide adequate ventilatory support, compared with the AC mode. PSV was not able to provide adequate \( V_t \), but this was likely constrained by the upper limit pressure support level of the ventilator (30 cm H\(_2\)O). We believe that in selected patients PSV will be adequate as a primary means of ventilatory support. However, in patients with highly unstable respiratory drive or changeable respiratory system impedance, PSV is not advisable unless backed.
up by volume-cycled breaths at a frequency sufficient to meet the patient's ventilatory demand.

(4) Other Possible Roles of PSV: PSV has also been used in noninvasive ventilation (face mask ventilation).\textsuperscript{55,56} Some system designs tolerate air leaks and are able to maintain the preset pressure support level.\textsuperscript{55} This feature makes it convenient for ventilation using a face or nasal mask. One example is a portable ventilator comprised of both PSV and CPAP systems (BiPAP system, Respironics, Inc, Monroeville, PA), currently used for the treatment of obstructive sleep apnea.\textsuperscript{57}

In one case report, PSV was able to correct respiratory acidosis in a postthoracic surgical patient with acute massive bronchopleural fistula and in whom CPPV had been unsuccessful.\textsuperscript{58} Although in this and the above circumstances the presence of air leaks appears tolerable and PSV provides benefit to the patient, in some circumstances it may create problems (see limitation of PSV below).

PSV has been reported to successfully decrease dynamic hyperinflation in a patient with acute respiratory failure related to COPD.\textsuperscript{59} Dynamic hyperinflation during CPPV was associated with hemodynamic compromise, and the latter condition was not corrected by the application of IMV. Following the use of PSV, PEEFi decreased markedly from 12 and 17 cm H\textsubscript{2}O to 7 cm H\textsubscript{2}O and hemodynamic status improved. The authors attributed the decrease in PEEFi to the lower frequency and prolonged expiratory time (Te) despite a similar VE as during CPPV.

Limitation

The limitations of PSV are well-summarized by MacIntyre et al.\textsuperscript{96} We will discuss some of these limitations.

PSV may deliver a variable VE in patients with unstable respiratory drive or highly changeable respiratory impedance. PSV should be used with caution in these patients.

Positive inspiratory pressure may persist in the presence of a circuit leak. A small circuit leak can produce continued flow "demand," causing a CPAP equal to the preset pressure support level because the expiratory criterion is not met.\textsuperscript{60} Fortunately, most ventilators have secondary breath termination criteria (3 to 5 s) to reduce this potential hazard.\textsuperscript{60}

The patient may fail to trigger the pressure-limited breath. This may occur during PSV when continuous in-line nebulizers are applied with flow rates that exceed the patient mean flow rate.\textsuperscript{61} The high nebulizer flow rate causes difficulty in triggering the pressure-limited breath, resulting in alveolar hyperventilation. In addition, the bias flow is sensed by the ventilator as the patient's minute ventilation, thereby preventing activation of the ventilator apnea alarm.\textsuperscript{62} This adverse effect can be prevented by using metered dose inhalers.

Current Status

PSV has gained wide clinical application. During weaning, PSV has been used in conjunction with DF CPAP or SIMV to decrease the W required to open the demand valve. However, guidelines for the application of PSV during weaning have yet to be adequately formulated. There has not been any randomized prospective study to evaluate the efficacy of PSV in comparison to either SIMV or T-piece weaning on the duration and outcome of weaning. As during SIMV, it is likely that PSV could also be used in conjunction with APRV to augment Vr and decrease the W of the spontaneous breaths.

PSV has the potential to be a primary means of ventilatory support. However, back-up ventilation would be required to meet the patient's ventilatory demand in the event that PSV failed (eg, with mandatory minute ventilation [MMV]). The ventilator design can still be improved by including adjustable initial flow rates\textsuperscript{41,42} and flow-triggering (FB) instead of pressure-triggering.

Proportional Assist Ventilation (PAV)

The prototype of PAV was first described by Younes et al.\textsuperscript{65} in 1987 and was applied in critically ill patients in 1989.\textsuperscript{63}

Description

PAV is a closed-loop positive pressure ventilation in which the ventilator changes pressure at the airway in proportion to the inspired volume (elastic assist), inspired flow (resistive assist), or both the inspired volume and flow (elastic and resistive assist). Gain controls on the flow and volume signals determine the proportionality, and therefore the degree of resistive and/or elastic unloading. In contrast to PAV, in the CPPV, PSV, or APRV modes, either the delivered volume (in CPPV) or the delivered pressure (in PSV and APRV) is fixed. The delivered volume or pressure in PAV varies according to the feedback signal received by the ventilator proportionate to the patient's elastic and/or resistive load. The patient leads the ventilator and the ventilator follows by providing pressure in proportion to inhaled flow and volume. PAV unloads the patient's inspiratory muscle work without altering the patient's breathing pattern. The advantages of PAV include the following: (1) the ventilator responds automatically to varying metabolic needs; (2) peak Paw is reduced, decreasing the likelihood of barotrauma, since the ventilator delivers pressure only to the desired level as determined by the patient; the patient tends to defend against overdistention; (3) synchrony between ventilator output and patient effort should
enhance patient comfort, avoiding the necessity for sedation or paralysis; (4) potential for muscle atrophy due to prolonged respiratory muscle disuse is reduced.

Laboratory and Clinical Studies

In anesthetized rabbits, Lebowitz and Poon compared the effect of PAV (designated by the authors as negative impedance ventilation [NIV]) and PSV on the relative risk of barotrauma in a model of increased resistive and elastic load. The risk of barotrauma was assessed by a noninvasive estimate of peak alveolar pressure (Palv). During resistive assist, PAV outperformed PSV by causing a substantially lower Palv for a given W and vice versa. During elastic assist, PAV approximated PSV, while peak Palv was variable in proportion to subject demand.

In a preliminary study of conscious critically ill patients with lung injury, elastic assist with PAV provided lower peak Paw (11 to 22 cm H₂O vs 25 to 40 cm H₂O with SIMV) and enhanced patient comfort, compared with SIMV. Oxygenation was similar with both modes of ventilatory support. From these preliminary studies, PAV appears capable of providing adequate gas exchange and unloading the patient's inspiratory muscle work. Furthermore, it has the advantage of lower peak airway or alveolar pressure in comparison to either CPPV or PSV.

Limitation

Because the ventilator-delivered volume or pressure requires a feedback signal from changes in the patient's respiratory system mechanics, it will not be possible to use PAV in apneic patients. In addition, because breathing pattern is determined by the patient, the abnormal breathing pattern (eg, bradypnea) will not be corrected during PAV.

Current Status

Although PAV is still in a preliminary stage of development, it has great potential for use as a primary means of ventilatory support or weaning in the near future. It can be applied to patients with respiratory failure due to both diffuse lung injury and/or airflow obstruction, provided that the patient maintains his or her spontaneous effort. A PAV system with means of back-up apneic ventilation would be ideal. Clinical studies are needed to evaluate the performance of PAV in comparison to CPPV with regard to morbidity, mortality, and safety. At the time of this writing, a multicenter study is being planned and will be started soon (M. Younes, personal communication March, 1991).

Mandatory Minute Volume (MMV)

MMV was first introduced by Hewlett et al in 1977 with the rationale of facilitating weaning from mechanical ventilation.

Description

It is the first type of closed-loop ventilation that allows the patient to breathe spontaneously. The ventilator provides a guaranteed minute ventilation (preset by the physician) if the patient's ventilation drops below a preset level. In the event that this occurs, a feedback mechanism is actuated and the machine delivers pressurized breaths of fixed volume until the preset minute volume is regained. The level of machine support can vary between total ventilatory support and complete patient dominance. As the patient's spontaneous ventilation increases, machine support decreases and vice versa.

Unlike the SIMV mode wherein the ventilator delivers a fixed number of breaths at all times, in the MMV mode the ventilator does not provide any assistance until patient minute ventilation falls below the preset level. Furthermore, the ventilator helps only to the point of restoring the preset minute ventilation.

Laboratory and Clinical Studies

Although MMV was meant to facilitate weaning, to my knowledge, there has not been a single study that systematically evaluated the effect of MMV in comparison to either SIMV or T-piece weaning on the duration, morbidity, or outcome of weaning.

Limitation

In the MMV mode, the quality of the spontaneous minute volume is not evaluated. Hence, minute volume induced by rapid shallow breathing is indistinguishable from minute volume induced by slow deep breathing as long as V̇E of both breathing patterns is similar. The former is inadequate to actuate the ventilatory support mechanism, and if undetected, may result in alveolar hypventilation. To overcome the above limitation, Norlander and Jarberg suggested that inspiratory pressure support or PSV be added to MMV. PSV would ensure adequate tidal volume in patients with rapid shallow breathing, while MMV would provide backup for PSV in patients with unstable respiratory drive. The efficacy of MMV as a backup for PSV to prevent alveolar hypventilation was demonstrated by East et al. In a study of anesthetized dogs conducted at various degrees of depressed central respiratory drive, MMV plus PSV prevented alveolar hypventilation in comparison to the use of PSV alone.

Current Status

Since its development in 1977, MMV has gained very limited clinical acceptance. Part of the reason for this is that the advantage of MMV over the more
commonly used weaning modalities has yet to be established.

MV has been incorporated in various commercially available ventilators and some of these have the capability to apply PSV to the MMV mode (eg, Bear 5, Engstrom Erica, Hamilton Veolar). The combined PSV and MMV mode has the potential for use as a primary means of ventilatory support. Its efficacy in terms of gas exchange, safety, morbidity, and mortality in comparison to CPPV has yet to be determined.

In summary, I have discussed five alternate modes of positive pressure ventilation, three of which are pressure-cycled (IRV, APRV, and PSV) and two of which are closed-loop ventilation (PAV and MMV). To date, IRV, APRV, PSV, and MMV have been incorporated in commercially available ventilators. Their basic function holds promise in terms of safety, patient comfort, and perhaps morbidity. However, the superiority of these alternate ventilatory modes to CPPV concerning patient survival cannot be established on the basis of the number of controlled studies to date. In addition, it should be noted that clinical use of these modes of ventilation requires specialized knowledge and expertise.

REFERENCES
12 Andersen JB. Ventilatory strategy in catastrophic lung disease: inverse ratio ventilation (IRV) and combined high frequency ventilation. Acta Anesthesiol Scand 1989; 90(suppl):145-48
18 Boros SI. Variations in inspiratory-expiratory ratio and airway pressure waveform during mechanical ventilation: the significance of mean airway pressure. J Pediatr 1979; 94:114-17
20 Pepe PE, Marini JJ. Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction. Am Rev Respir Dis 1982; 126:166-70
22 Duncan SB, Rizk NW, RaBn TA. Inverse ratio ventilation. PEEP in disguise [editorial]. Chest 1987; 92:390-92
34 Florete OG, Banner MJ, Banner TE, Rodriguez JC, Kirby RR. Airway pressure release ventilation in a patient with acute
35 MacIntyre NR. Respiratory function during pressure support ventilation. Chest 1986; 89:677-83
38 FassoulaK A, Efrikopoulou M. Cardiovascular, respiratory, and metabolic changes produced by pressure-supported ventilation in intensive care unit patients. Crit Care Med 1989; 17:527-29
40 MacIntyre NR, Ho LJ. Effects of initial flow rate and breath termination criteria on pressure support ventilation. Chest 1991; 99:134-38
41 Branson RD, Campbell RS, Davis K, JohannaGman JA, Johnson DJ, Hurst JM. Altering flowrate during maximum pressure support ventilation (PSVmax): effects on cardiacorespiratory function. Respir Care 1990; 35:1056-64
48 Prakash O, Meij S. Cardiopulmonary response to inspiratory pressure support during spontaneous ventilation vs conventional ventilation. Chest 1985; 88:403-08
52 Al-Saady N, Bennett E.D. Decelerating inspiratory flow waveform improves lung mechanics and gas exchange in patients on intermittent positive pressure ventilation. Intensive Care Med 1985; 11:69-75
65 Ravenscroft Pj. Simple mandatory minute volume. Anesthesia 1978; 33:246-49
66 Norlander O, Jarnberg PO. Control mode ventilation and mandatory minute ventilation. Ann Chirurg Gynaecol 1982; 71(suppl):196:64-7
67 East TD, Elkhuizen PHM, Pace NL. Pressure support in mandatory minute ventilation supplied by the Ohmeda CPU-1 prevents alveolar hypoventilation due to respiratory depression in a canine model. Respir Care 1989; 34:795-800