Ventilator Graphics and Respiratory Mechanics in the Patient With Obstructive Lung Disease

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Obstruction of the large and small airways occurs in several diseases, including asthma, chronic obstructive pulmonary disease, cystic fibrosis, bronchiectasis, and bronchiolitis. This article discusses the role of ventilator waveforms in the context of factors that contribute to the development of respiratory failure and acute respiratory distress in patients with obstructive lung disease. Displays of pressure, flow, and volume, flow-volume loops, and pressure-volume loops are available on most modern ventilators. In mechanically ventilated patients with airway obstruction, ventilator graphics aid in recognizing abnormalities in function, in optimizing ventilator settings to promote patient-ventilator interaction, and in diagnosing complications before overt clinical signs develop. Ventilator waveforms are employed to detect the presence of dynamic hyperinflation and to measure lung mechanics. Various forms of patient-ventilator asynchrony (eg, auto-triggering and delayed or ineffective triggering) can also be detected by waveform analysis. Presence of flow limitation during expiration and excessive airway secretions can be determined from flow-volume loops. Abnormalities in pressure-volume loops occur when the trigger sensitivity is inadequate, with alterations in respiratory compliance, or during patient-ventilator asynchrony. Thus, ventilator waveforms play an important role in management of mechanically-ventilated patients with obstructive lung disease. *Key words: chronic obstructive pulmonary disease, COPD, mechanical ventilation, waveforms.* [Respir Care 2005;50(2):246–259.

Introduction

Several diseases, including asthma, chronic obstructive pulmonary disease (COPD), cystic fibrosis, bronchiectasis, bronchiolitis, and others, cause narrowing of the small and large airways.[1] Patients with obstructive lung diseases constitute a substantial proportion of patients receiving mechanical ventilation. In an international survey of mechanical ventilation, approximately 12% of patients were receiving mechanical ventilation primarily because of obstructive lung disease.[2] Episodes of respiratory failure that require mechanical ventilation often follow exacerbations of COPD. Moreover, the presence of COPD may prolong the duration of mechanical ventilation in many patients who receive mechanical ventilation for various other disorders. For example, postoperative patients are frequently unable to wean from the ventilator because of underlying COPD. Patients with obstructive lung disease present unique and complex challenges during mechanical ventilation. The optimal management of mechanically-ventilated patients with obstructive lung disease has been reviewed.[3,4] The purpose of this article is to discuss the pathophysiologic changes in ventilator-supported patients with obstructive lung disease and the role of routinely available ventilator waveforms in detecting alterations in pulmonary physiology and lung mechanics. Routine monitoring of ventilator waveforms could play an important role in the management of mechanically-ventilated patients with obstructive lung disease, and this article will highlight the role of ventilator waveforms in the care of such patients.

Primary Goals of Ventilator Waveform Monitoring in Patients With Obstructive Lung Disease

Ventilator graphics provide an immediate display of patient-ventilator interaction, and they allow the clinician to use pattern-recognition to evaluate normal and abnormal pulmonary function. In the last decade, the ability to display pressure-time, flow-time, and volume-time waveforms as well as pressure-volume and flow-volume loops at the bedside has become routinely available in most modern mechanical ventilators.[5] Clinicians can measure static and dynamic lung compliance, inspiratory and expiratory resistance, and intrinsic positive end-expiratory pressure (PEEPi) by performing inspiratory and expiratory pauses during lung inflation and deflation. Placement of an esophageal balloon allows more sophisticated calculations, including the work of breathing and determination of chest wall compliance.[5–8] Esophageal balloons are, however, not routinely employed in clinical practice. The ventilator waveforms that are routinely available in clinical practice are discussed below.

Table 1. Role of Ventilator Waveforms in Ventilator-Dependent Patients

1. Identify pathophysiologic processes
2. Recognize a change in patient's condition
3. Optimize ventilator settings and treatment
4. Determine effectiveness of ventilator settings
5. Detect adverse effects of mechanical ventilation
6. Minimize risk of ventilator-induced complications

Waveforms can serve several functions with ventilator-supported patients (Table 1). Not only do abnormalities in waveforms serve as an excellent tool for clinical diagnosis, they also further extend the diagnostic capabilities by providing information regarding the pulmonary mechanics and patient-ventilator interaction. In addition, recognition of various waveform patterns is also helpful in optimizing ventilator settings, in detecting adverse effects due to mechanical ventilation per se, and in minimizing complications due to mechanical ventilation.

Pathophysiologic Changes in Mechanically-Ventilated Patients With Obstructive Lung Disease

Table 2 shows the pathophysiologic changes that contribute to the development of respiratory distress and acute respiratory failure in patients with obstructive lung disease. Analysis of ventilator waveforms is particularly helpful in determining changes in airway resistance, dynamic hyperinflation, patient-ventilator asynchrony, and excessive airway secretions.

Increase in Airway Resistance

Airway narrowing in patients with obstructive lung diseases produces a critical increase in airway resistance, especially during exhalation. Breathing through narrowed

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Table 2. Pathophysiologic Changes in Mechanically-Ventilated Patients With Obstructive Lung Disease

Physiologic Abnormality	Mechanisms
Increased airway resistance	Bronchoconstriction, mucosal edema, increased secretions, loss of elastic recoil, peribronchiolar inflammation
Dynamic hyperinflation	High minute volume, prolonged expiratory time constants, low I:E
Patient-ventilator asynchrony	Inadequate exhalation time, intrinsic PEEP, ventilator-nontriggering
Increase in airway secretions	Airway inflammation, airway edema
Increased ventilatory demand	Hypoxemia, lung inflammation, overdistention
Abnormalities of gas exchange	$\dot{V}/\dot{Q}$ mismatch, shunt, hypoventilation
Cardiovascular dysfunction	Pulmonary hypertension (loss of capillary bed, hypoxic vasoconstriction), increased RV afterload, gas exchange abnormalities, reduced venous return, increased LV afterload, LV failure

I:E = ratio of inspiratory time to expiratory time
PEEP = positive end-expiratory pressure
$\dot{V}/\dot{Q}$ = ventilation/perfusion
RV = right ventricle
LV = left ventricle

airways imposes an additional load on the respiratory muscles.

Dynamic Hyperinflation

In the presence of high resistance to expiratory flows and short expiratory times, the respiratory system is unable to return to its resting volume at the end of exhalation. As a result, a new resting state is established, such that there is a positive recoil pressure (PEEPi) at the end of expiration (Fig. 1).[9–11] This state of air trapping or dynamic hyperinflation is common in patients with obstructive lung disease.[11] Initially, hyperinflation tends to keep the airways open, reduces airway resistance, increases elastic recoil, and tends to improve expiratory flow. However, hyperinflation has several deleterious effects. The positive pressure within regions of hyperinflated lung raises the mean intrathoracic pressure and causes the inspiratory muscles to operate at a higher than resting lung volume. Thus, dynamic hyperinflation places the respiratory muscles at a considerable mechanical disadvantage[12–14] and further impairs respiratory muscle function. PEEPi also imposes a substantial inspiratory threshold load, because a negative intrapleural pressure equal to the level of PEEPi has to be generated before inspiratory flow begins within alveoli.[15] This threshold load imposed by PEEPi interferes with ventilator triggering (see below). Moreover, regions of hyperinflated lung may compress adjacent areas of normal lung and adversely affect ventilation/perfusion relationships. PEEPi also decreases cardiac output and predisposes patients to barotrauma.[10,16,17] Therefore, decreasing dynamic hyperinflation should be a key consideration while treating mechanically-ventilated patients with obstructive lung disease. Unfortunately, too much emphasis is placed on the *inspiratory* parameters while setting the ventilator, and much less attention is given to the *exhalation* phase. Patients with obstructive lung disease often require 3 seconds or more to complete exhalation. In patients with airway obstruction, ventilator settings that do not allow adequate time for exhalation could lead to or worsen dynamic hyperinflation.

Patient-Ventilator Asynchrony

Mechanically-ventilated patients with obstructive lung disease who develop PEEPi have to generate a negative intrapleural pressure equal in magnitude to the level of PEEPi, in addition to the ventilator sensitivity level, before triggering occurs and a ventilator breath is initiated (Fig. 2).[18] When the inspiratory effort by the patient is less than that threshold value, the ventilator will not deliver a breath. Thus, dynamic hyperinflation leads to frequent nontriggering of breaths in patients with obstructive lung disease. Such nontriggered breaths represent wasted breathing effort on the part of the patient, and lead to patient-ventilator asynchrony.[19,20] In assist-control modes the ventilator should be set to be more responsive to the patient's breathing effort. In addition, application of external PEEP in mechanically-ventilated patients with obstructive lung disease and PEEPi may reduce nontriggered breaths by narrowing the difference between mouth pressure and alveolar pressure at end-expiration (see Fig. 1). Application of, or increase in, the level of external PEEP could reduce the elastic threshold load and work of breathing, particularly in patients with flow limitation during tidal expiration.

Increase in Airway Secretions

Acute and chronic inflammation in the lung produces excessive luminal secretions within airways.[21] The inability of most mechanically-ventilated patients to expectorate these secretions worsens the airway obstruction and pre-

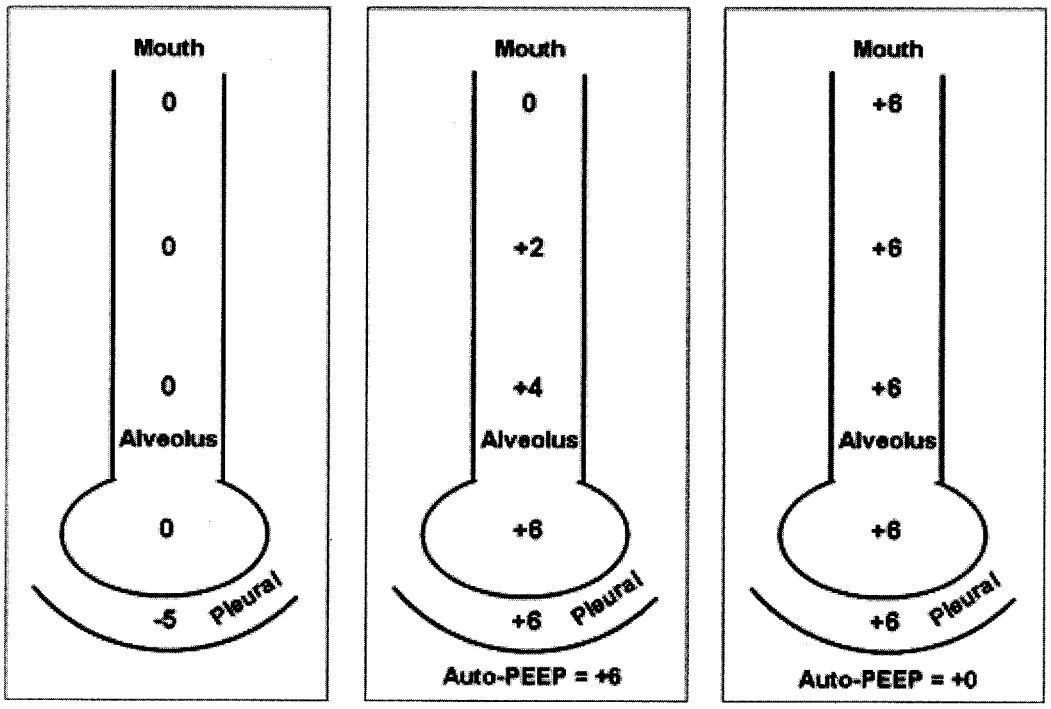

Fig. 1. Airway and alveolar pressures in patients with intrinsic positive end-expiratory pressure (auto-PEEP) and the effect of increasing extrinsic PEEP. At the end of a normal expiration (left panel), the alveolar pressure equilibrates with the airway pressure. In this example the alveolar pressure is equal to airway pressure, which in turn is equal to atmospheric pressure when external PEEP is set at zero. In the presence of dynamic hyperinflation, alveolar pressure remains higher than airway pressure at end-expiration (middle panel). The pressure within alveoli at end-expiration represents the level of auto-PEEP. The difference between alveolar pressure and airway pressure may be reduced by increasing the external PEEP (right panel). Mechanical ventilators routinely display airway pressure. Special maneuvers are needed to determine end-expiratory alveolar pressure (auto-PEEP) in mechanically-ventilated patients.

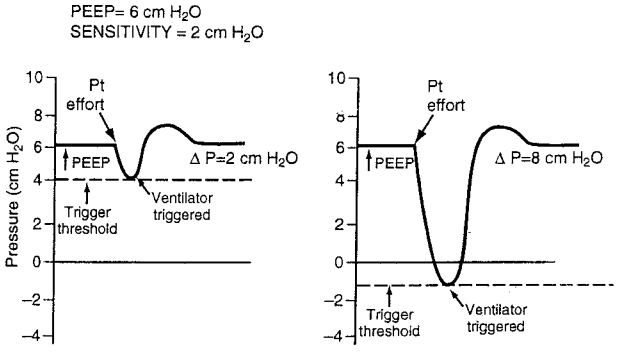

Fig. 2. Pressure-triggering in a patient with intrinsic positive end-expiratory pressure (PEEP). In the left panel, the level of external PEEP is set at 6 cm H_2O and the trigger threshold is set 2 cm H_2O below that level. The ventilator is triggered when the patient's inspiratory effort reduces the airway pressure to the set threshold level. In contrast, the right panel shows a patient with a set PEEP of zero and a similar trigger threshold of 2 cm H_2O. However, this patient has an intrinsic PEEP level of 6 cm H_2O. The patient's inspiratory effort would therefore have to generate a negative pressure of 8 cm H_2O to trigger the ventilator. Pt = patient. ΔP = change in pressure. (From Reference 18, with permission.)

disposes to development of pneumonia. Other factors, such as airway edema due to congestive heart failure or fluid overload, may also contribute to the increase in airway secretions.

Increased Ventilatory Demand

Patients with obstructive lung disease may have a normal or excessive drive to breathe, due to the interaction of several factors, including hypoxemia, hyperinflation, and interstitial edema due to pneumonia or congestive heart failure (see Table 2).[22] In view of the increased load on the respiratory muscles and their reduced capacity for pressure generation, an increased demand on the muscles tends to produce muscle fatigue and ventilatory failure.[23]

Abnormalities of Gas Exchange

Several factors contribute to development of gas exchange abnormalities in patients with obstructive lung disease (Table 3). Airway obstruction produces regional hypoventilation that produces ventilation/perfusion mismatch

Table 3. Gas-Exchange Abnormalities in Patients With Obstructive Lung Disease

	Physiologic Defect	Underlying mechanisms
Hypoxemia	$\dot{V}/\dot{Q}$ mismatch	Airway narrowing, compression of normal alveoli by overdistended alveoli, loss of capillary bed
	Pulmonary hypertension	Reduced pulmonary blood flow, RV failure
	Shunt	Pneumonia, congestive heart failure
Hypercapnia	Hypoventilation	Airway narrowing, increased V_D/V_T, dynamic hyperinflation, increased elastic load, respiratory muscle weakness

$\dot{V}/\dot{Q}$ = ventilation/perfusion
RV = right ventricle
V_D/V_T = ratio of dead space volume to tidal volume

and hypoxemia.[24] In addition, concomitant pneumonia or congestive heart failure may worsen hypoxemia in these patients. Patients with acute asthma may initially compensate for hypoxemia by hyperventilation.[25] However, hypoventilation develops in the presence of severe airway narrowing and dynamic hyperinflation when the respiratory muscles are unable to bear the excessive resistive and elastic loads. Dynamic hyperinflation also places the respiratory muscles at a mechanical disadvantage.[12–14] Therefore, alveolar hypoventilation and hypercapnia are well known to develop in the most severe episodes of acute asthma. The presence of normocapnia in a patient suffering from acute asthma ("pseudonormalization") should be viewed with caution, as patients with acute asthma can progress rapidly from normocapnia to hypercapnia, acidemia, and death.[26,27] Loss of the capillary bed due to emphysema or compression of pulmonary capillaries by overinflated alveoli also tends to increase dead space, and this wasted ventilation further compromises the ability of the respiratory muscles to provide adequate ventilation.[28] Moreover, hypoxemic pulmonary vasoconstriction and right ventricular overload due to chronic vascular changes also reduce pulmonary blood flow and adversely effect gas exchange.[29] Hypoxemia and/or hypercapnia of varying severity are thus very common in ventilator-dependent patients with obstructive lung disease.[30]

Cardiovascular Dysfunction

In spontaneously breathing or ventilator-assisted patients with obstructive lung disease, cardiovascular dysfunction results from the interplay of several factors. Positive intrathoracic pressure is well known to reduce venous return and stroke volume.[31] The venous return may be further compromised by an increase in abdominal pressure due to active expiratory efforts in patients with obstructed airways. Increase in abdominal pressure also tends to collapse the inferior vena cava during expiration.[32] Hypotension may result soon after institution of mechanical ventilation in patients with airway obstruction, especially in patients who are hypovolemic.[16] Sedative administration may further compound the problem. Moreover, dynamic hyperinflation further reduces venous return, and by compressing alveolar capillaries, increases pulmonary vascular resistance, increases right ventricular afterload, and further compromises right heart function.[33,34]

Increased respiratory efforts in patients with obstructive lung disease who are spontaneously breathing or mechanically ventilated with patient-triggered modes may contribute to cardiovascular dysfunction. Vigorous inspiration against narrowed airways causes increased filling of the right heart. In the face of increased right ventricular afterload, increased filling of the right ventricle shifts the interventricular septum to the left, with a reduction in the diastolic compliance of the left ventricle.[16,35] Negative intrathoracic pressures also increase left ventricular afterload and tend to reduce stroke volume. These events may lead to increases in left ventricle end-diastolic pressures and could precipitate cardiogenic pulmonary edema.[35,36] Cardiovascular dysfunction may, therefore, worsen gas exchange and reduce blood flow to respiratory muscles, while further increasing respiratory loads and ventilatory demands.[37]

Ventilator Waveforms

Continuous displays of ventilator waveforms assist the clinician in detecting and monitoring the pathophysiologic changes described above. Table 4 lists various waveforms that are commonly available on ventilators.

The clinical examination provides valuable clues to the presence of respiratory distress in a spontaneously breathing patient or a patient receiving assisted modes of mechanical ventilation. The presence of tachypnea, air hun-

Table 4. Bedside Ventilator Waveforms That Assist in Managing Patients With Obstructive Lung Diseases

Pressure, flow, and volume waveforms
Pulmonary mechanics measurements
Flow-volume loops
Pressure-volume loops

ger, tachycardia, hypertension, or hypotension; decrease in arterial oxygen saturation; use of accessory muscles of breathing; reduction or absence of breath sounds over a region of the lung; wheezing; rib-cage abdominal asynchrony; paradoxical movement of the abdominal wall during inspiration; cyanosis; inability to trigger the ventilator; and apnea may all point to the need for urgent interventions or ventilator adjustments.[38] Ventilator waveforms can indicate abnormalities before clinical signs are evident, and they may help in distinguishing the various pathophysiological mechanisms leading to respiratory distress or patient-ventilator asynchrony. Thus, ventilator waveforms can play an important role in the clinical evaluation and management of mechanically-ventilated patients with airway obstruction.

Pressure, Flow, and Volume Waveforms

Pressure, flow, and volume waveforms are helpful in detecting the presence of dynamic hyperinflation. The persistence of flow at the end of relaxed expiration indicates that the system is above passive functional residual capacity and that flow is being driven by positive elastic recoil of the respiratory system at end-expiration (PEEPi) (Fig. 3). The presence and level of PEEPi can be confirmed by assessing the end-expiratory airway occlusion pressure (see below) and comparing it with the set level of extrinsic PEEP.

The flow and volume waveforms could be employed to directly estimate end-expiratory volume above passive functional residual capacity.[39] In a paralyzed patient, a prolonged apnea (up to 40 s) is allowed after a breath from the ventilator. The total exhaled volume is measured from the end of inspiration until there is no visually detectable change in volume (Fig. 4). The total volume of exhaled gas (called the volume at end-inspiration) is the sum of the tidal volume (V_T) and any trapped gas above functional residual capacity. By subtracting the V_T from the volume at end-inspiration one can obtain the volume at end-expiration and estimate the volume of trapped gas at end-expiration. The need for paralyzing the patient to obtain this measurement is an important problem with the routine use of this technique. Moreover, to accurately measure the volume at end-inspiration, the equipment needs to be sensitive to very low flow rates.

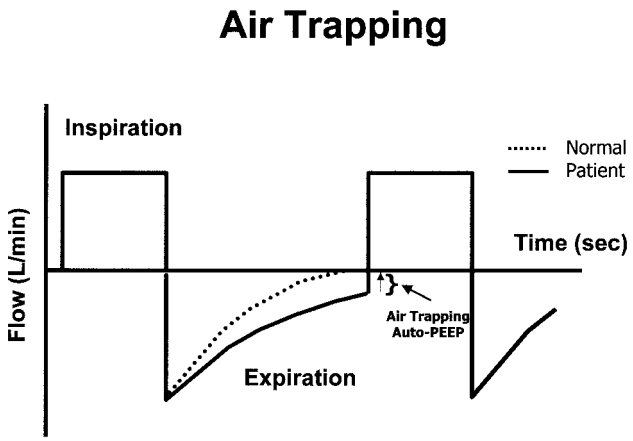

Fig. 3. Flow-time waveform showing persistence of flow at end-expiration in a patient with intrinsic positive end-expiratory pressure (auto-PEEP). In most patients with obstructive lung disease, failure to reach zero flow at the end of a relaxed expiration signifies that lung volume is above functional residual capacity and indicates dynamic hyperinflation.

Lung Mechanics Measurements

Measurements of respiratory mechanics in a ventilator-dependent patient can be obtained with the technique of rapid airway occlusion during constant-flow inflation.[40,41] This technique is applicable with modern ventilators that have a constant inspiratory flow. Controlled mechanical ventilation is required to accurately measure pulmonary mechanics with this technique; that is, the patient has to be relaxed and not making active inspiratory efforts. During such passive ventilation, inflation of the lung produces expansion of the thorax, and no negative intrathoracic pressure is generated by muscular effort. Deep sedation or neuromuscular paralysis may be needed to achieve such a state of relaxed passive ventilation.

Measurement of Respiratory-System Resistance and Compliance

In the rapid airway-occlusion technique, a breath-hold is performed at end-inspiration by rapidly occluding the expiratory port during controlled mechanical ventilation.[42] The airway occlusion produces an immediate drop in peak airway pressure (P_{peak}) to a lower initial pressure (P_{init}). The pressure then declines gradually to reach a plateau after 3–5 seconds (P_{plat}) (Fig. 5).[41,42] The value of P_{init} can be determined by back-extrapolation of the slope of the latter part of the airway waveform to the time of airway occlusion. This permits total or maximum resistance of the respiratory system (R_{RS}-max) to be partitioned into a minimum resistance of the respiratory system (R_{RS}-min), which reflects the ohmic resistance of the airways, and an additional effective change in respiratory resistance (ΔR_{RS}).

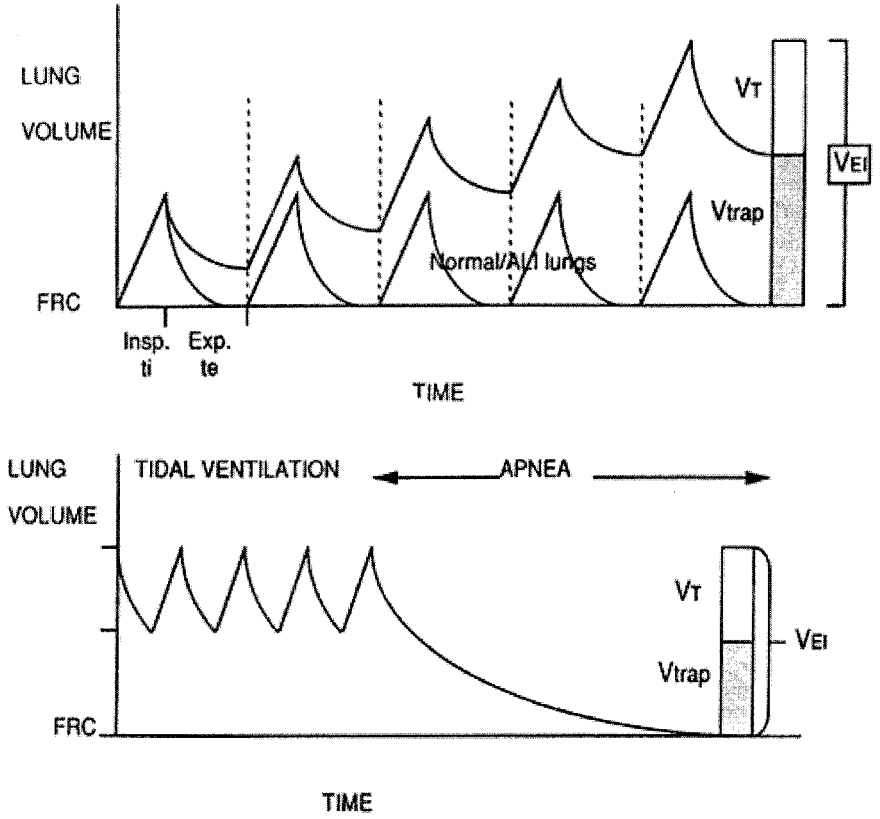

Fig. 4. In the absence of air trapping the volume of the lung at end-expiration reaches functional residual capacity (FRC). In contrast, in the presence of dynamic hyperinflation the volume of the lung at end-expiration remains higher than FRC (upper waveform in upper panel). The volume expired during a prolonged apnea could be used to determine the volume of gas trapped above FRC (lower panel). The difference between the volume at end-inspiration (V_{EI}) and tidal volume (V_T) represents the volume above FRC, or trapped gas volume (V_{trap}). ALI = acute lung injury. Insp. ti = inspiratory time. Exp. ti = expiratory time. V_T = tidal volume. (From Reference 39, with permission).

ΔR_{RS} represents 2 phenomena: time-constant inhomogeneities within the lung (pendelluft), and the viscoelastic behavior or stress relaxation of the pulmonary tissues.[43,44] Similarly, rapid occlusion of the expiratory port at end-exhalation produces an increase in airway pressure, and its plateau signifies the level of PEEPi (Fig. 6). In a passively ventilated patient, pulmonary mechanics may be calculated from these measurements as follows:

$$R_{RS}\text{-max} = (P_{peak} - P_{plat})/\text{flow} \quad (1)$$

$$R_{RS}\text{-min} = (P_{peak} - P_{init})/\text{flow} \quad (2)$$

$$\Delta R_{RS} = R_{RS}\text{-max} - R_{RS}\text{-min} \quad (3)$$

$$C_{RS} = V_T/(P_{plat} - \text{PEEPi}) \quad (4)$$

in which C_{RS} is respiratory-system compliance. Most of the increased resistance in mechanically-ventilated patients

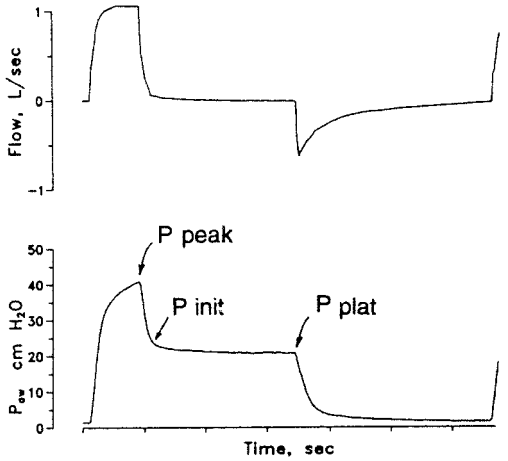

Fig. 5. Recordings of flow and airway pressure (P_{aw}) over time in a patient with chronic obstructive pulmonary disease receiving controlled mechanical ventilation. When the airway is occluded at end-inhalation, the airway pressure declines rapidly from peak inspiratory pressure (P_{peak}) to a lower initial pressure (P_{init}), followed by a gradual decrease to a plateau pressure (P_{plat}). (From Reference 43, with permission.)

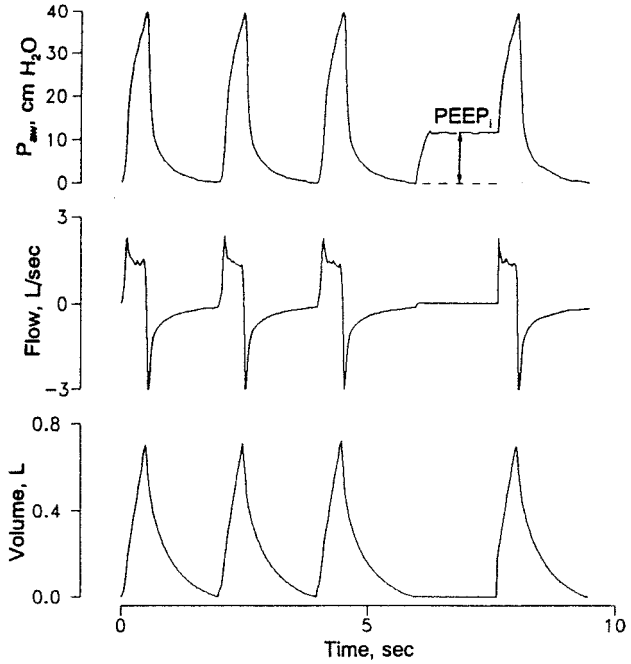

Fig. 6. Airway pressure (P_{aw}), flow, and volume waveforms from a patient receiving controlled mechanical ventilation. After the third breath, an airway occlusion was performed by rapidly occluding the expiratory port of the ventilator. During the occlusion, pressure in the airway equilibrates with alveolar pressure. In the presence of intrinsic positive end-expiratory pressure (PEEPi), airway pressure increases and the plateau value signifies the level of intrinsic PEEPi. (From Reference 8, with permission.)

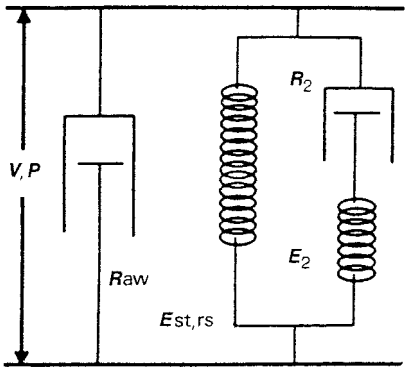

Fig. 7. Spring-and-dashpot model for interpretation of respiratory mechanics during flow interruption. The respiratory system is modeled as a standard airway resistance (R_{aw}) in parallel with a standard elastic resistance ($E_{st,rs}$), and a series spring-and-dashpot body (E_2 and R_2, respectively), which represent stress adaptation units. The distance between the 2 horizontal bars is the analogue of lung volume (V), and the tension between these bars is the analogue of pressure (P) at the airway opening. (From Reference 46, with permission.)

with obstructive lung disease originates in the lung, with minimal contribution from the chest wall resistance.[45]

Sequential changes in airway pressures after rapid airway-occlusion at constant-flow inflation are more complex than can be fully explained on the basis of the traditional single-compartment model of the respiratory system.[41] Traditionally, the elastic tissues of the alveolus and chest wall have been represented as a single alveolar compartment at the end of a conducting airway, and there is a resistive pressure drop as gas flows through the conducting airway. Milic-Emili et al proposed a spring-and-dashpot model to represent the viscoelastic behavior of thoracic tissues.[46] The model consists of 4 elements (Fig. 7). There are 2 parallel compartments in the model: a dashpot representing airway resistance (R_{aw}) and a Kelvin body. The latter consists of a spring that represents the static elastance of the respiratory system ($E_{st,rs}$), in parallel with a Maxwell body. The latter consists of a spring (E_2) and a dashpot (R_2), arranged serially. In this model the respiratory system consists of a standard R_{aw} in parallel with a standard $E_{st,rs}$, and a series spring-and-dashpot body (E_2 and R_2, respectively), which represent viscoelastic properties of the thoracic tissues (lung and chest wall).[46] The distance between the 2 horizontal bars is the analogue of lung volume, and the tension between the 2 bars is the analogue of pressure at the airway opening. Clinical studies with rapid airway-occlusions performed in anesthetized, paralyzed humans corroborate the predictions from this model and help to better explain the complex behavior of the respiratory system after rapid airway-occlusion is performed.[43–45]

Measurement of PEEPi

The rapid airway-occlusion at end-exhalation can be employed to measure PEEPi (see Fig. 6). The rapid airway-occlusion technique can also be employed to determine hyperinflation by comparing the P_{plat} between ventilator cycles at operational lung volume and breathing frequency with the values obtained following a long expiration that allows complete deflation of the system.

Patient-Ventilator Asynchrony

For mechanical ventilation to effectively unload the patient's respiratory muscles, the ventilator must provide inspiratory flow in synchrony with the patient's own respiratory rhythm. The patient's respiratory muscles continue to contribute a variable portion of the inspiratory effort during mechanical ventilation. At high levels of ventilator assistance, the contribution by the patient's respiratory efforts to the total energy expenditure during breathing is reduced. Several factors could influence the response of the ventilator to the patient's effort.[3,47] The trigger function is usually pressure or flow.[48,49] With pressure-triggering, the patient must decrease the pressure in the ventilator

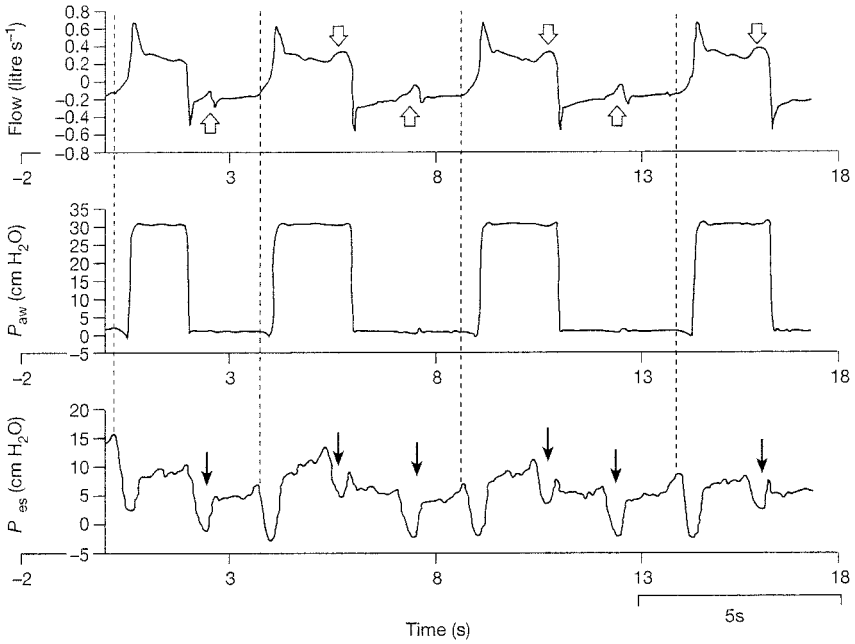

Fig. 8. Flow, airway pressure (P_{aw}), and esophageal pressure (P_{es}) waveforms from a patient with severe chronic obstructive pulmonary disease (COPD) receiving pressure support of 20 cm H_2O. The start of inspiratory efforts that trigger the ventilator are indicated by dotted vertical lines. There is a substantial time delay between the onset of the patient's inspiratory effort and the onset of flow from the ventilator. There are also several ineffective efforts, which are indicated by the black arrows. The ventilator rate is set at 12 breaths/min, but the patient is making 33 efforts/min. Note that ineffective efforts occur during both mechanical inspiration and expiration. Flow waveforms are helpful in identifying ineffective efforts, indicated by the open arrows. Ineffective efforts during mechanical inspiration cause an abrupt increase in inspiratory flow, whereas during mechanical expiration they cause an abrupt decrease in expiratory flow. (From Reference 47, with permission.)

circuit to a preset value, usually 1–2 cm H_2O. This decrease in pressure then triggers inspiratory flow. Substantial patient effort may be required during pressure-triggering, and the patient's inspiratory flow demands may not be met during the time interval required to trigger. With flow-triggering, the ventilator delivers inspiratory flow once the patient is able to generate a preset inspiratory flow.[48,49] A continuous (or bias) flow is maintained through the circuit, and the ventilator is triggered when the patient's effort creates a difference between the inspiratory and expiratory bias flow in the circuit. Flow-triggering is generally believed to require less patient effort than pressure-triggering. However, with recent improvements in pressure sensors in modern ventilators, there is minimal difference in the patient's work of breathing between flow-triggering and pressure-triggering.[50]

Autotriggering occurs when a mechanical breath is initiated without inspiratory effort by the patient.[48] Such an event may be due to random noise in the circuit, water in the circuit, circuit leaks, or cardiac oscillations.[51] The risk of such a phenomenon increases with greater sensitivity of the triggering system.

Inability of a patient effort to trigger the ventilator or delay in triggering is called ineffective triggering and is especially common when dynamic hyperinflation develops in mechanically-ventilated patients. In the presence of PEEPi, the patient has to employ additional inspiratory-muscle effort to decrease alveolar pressure below the external PEEP level in order to trigger the ventilator (see Fig. 2).[19,52] The presence of PEEPi therefore delays onset of effective inspiratory effort and ventilator triggering. When the triggering is considerably delayed, the mandatory breaths may be completely out of phase with the patient's breaths (Fig. 8).[47] Such asynchrony can be corrected by adjusting the ventilator settings or by reducing the patient's respiratory drive. The inability of a patient effort to trigger a breath occurs with surprising frequency during high levels of ventilator support provided by pressure support or assist-control ventilation modes (Fig. 9).[19,53] There is a proportional increase in the frequency of ineffective triggering as the level of ventilator assistance is increased. For example, with high levels of pressure support, Leung et al found that as many as one third of all patient efforts may fail to trigger the ventilator.[19] Ineffective triggering occurred mainly due to premature inspiratory efforts before elastic recoil pressure had fallen to a level that could be overcome by the patient's inspiratory efforts.[19] With ineffective triggering, careful examination of ventilator waveforms reveals patient efforts that are not recognized by the ventilator (see Figs. 8 and 9). Adjustment of the

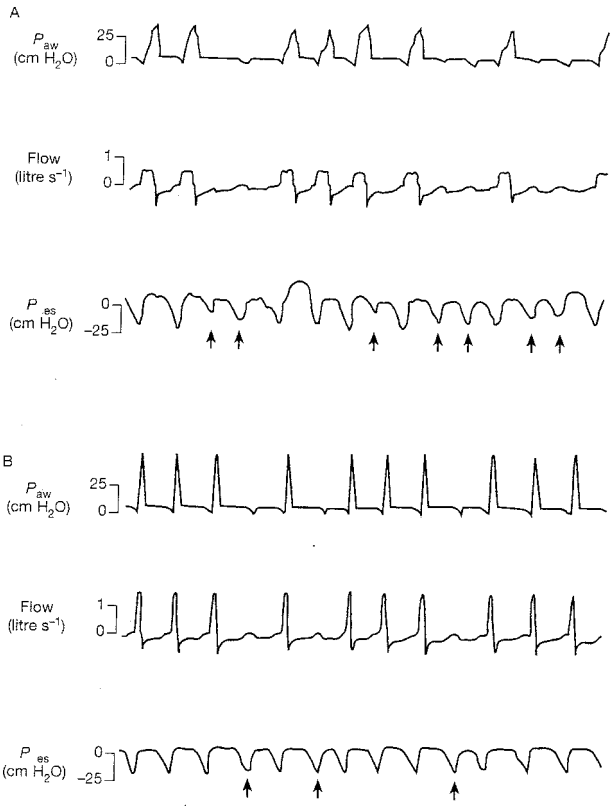

Fig. 9. Ventilator waveforms showing the effect of inspiratory flow on ineffective patient efforts that do not trigger the ventilator. These airway pressure (P_{aw}), flow, and esophageal pressure (P_{es}) waveforms are from a patient with chronic obstructive pulmonary disease receiving assist-control ventilation, with a constant tidal volume of 0.55 L. Ineffective efforts are indicated by arrows. A: Several ineffective efforts are seen with an inspiratory flow rate of 30 L/min. B: The inspiratory flow rate was increased to 90 L/min. By decreasing the inspiratory time and allowing more time for exhalation, dynamic hyperinflation was reduced and the number of ineffective efforts decreased. The ventilator rate increased as the patient was able to more effectively trigger the ventilator. In patients with dynamic hyperinflation causing ineffective triggering, an increase in the inspiratory flow rate could substantially reduce wasted patient effort. (From Reference 47, with permission.)

ventilator settings to achieve better patient-ventilator interaction could help to more effectively unload the patient's respiratory muscles during mechanical ventilation (see Fig. 9).

Flow-Volume Loops

Figure 10A shows a normal flow-volume loop. The inspiratory limb of the curve reflects the setting of inspiratory flow. The expiratory limb shows a smooth decrease in expiratory flow. Analysis of the flow-volume loop may be helpful for identifying reduced expiratory flow, flow limitation during expiration, and the presence of excessive secretions.

In patients with reduced expiratory flow due to airway narrowing, the expiratory limb of the flow-volume loop shows a curvilinear pattern (see Fig. 10B). When expiratory flow persists at end-exhalation, the expiratory limb of the flow-volume loop appears truncated, which indicates failure of the respiratory system to achieve its resting equilibrium position (dynamic hyperinflation).

Flow limitation during expiration can be detected by applying negative pressure at the airway opening during expiration.[54] In the absence of expiratory flow limitation, the application of negative pressure at the mouth increases expiratory flow. In contrast, in patients with expiratory flow limitation, flow does not increase when negative pressure is applied (Fig. 11).

The flow-volume curve can provide an indication of excessive secretions more reliably than clinical examination. The most reliable indicator of the presence of excessive secretions in the airways is the presence of a sawtooth pattern on both the inspiratory and expiratory flow-volume curves (Fig. 12). In 35 ventilator-dependent patients, the presence of this abnormality suggested a high probability of the presence of secretions, whereas the absence of such a pattern suggested that secretions were absent.[55]

Pressure-Volume Loops

The use of pressure-volume curves has been advocated for optimizing ventilator settings in patients with acute lung injury.[56] In patients with obstructive lung disease, pressure-volume loops may be helpful in detecting inadequate trigger sensitivity (Fig. 13A). Changes in the shape of the curve may occur with alterations in the respiratory compliance (see Figs. 13B and 13C). The presence of overdistention may be detected. Likewise, inadequate inspiratory flow or active patient inspiratory efforts may also be seen in the pressure-volume loop (Fig. 14).

Effects of Treatment

Bronchodilator Therapy

Bronchodilators are commonly employed for treatment of ventilator-supported patients with asthma or COPD.[57] Bronchodilators help by reversing bronchoconstriction, decreasing airway resistance, and reducing dynamic hyperinflation and PEEPi.[43,58] As a result, the work of breathing decreases and the patient's sensation of dyspnea is relieved. Lung mechanics determined by the rapid airway-occlusion technique could be employed to determine the effect of bronchodilator therapy.[43,58] Changes in the flow-

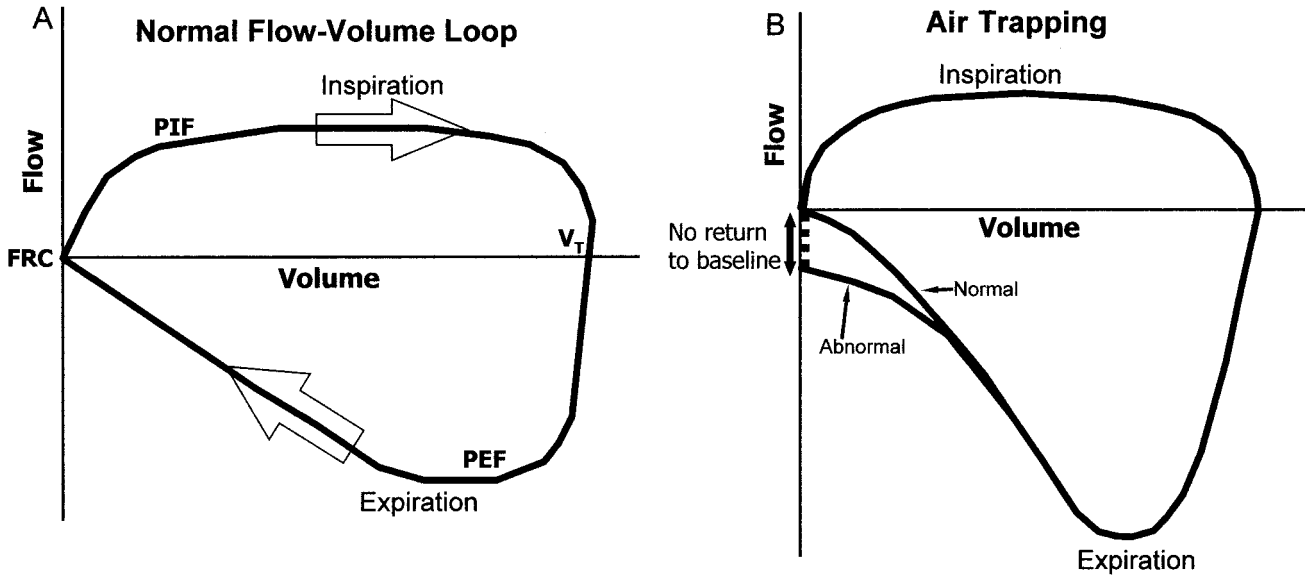

Fig. 10. Flow-volume loops display inspiratory and expiratory flow at various lung volumes. A: Normal flow-volume loop. B: Flow-volume loop from a patient with intrinsic positive end-expiratory pressure (air trapping), the expiratory limb of the loop appears truncated. PIF = peak inspiratory flow. FRC = functional residual capacity. V_T = tidal volume. PEF = peak expiratory flow.

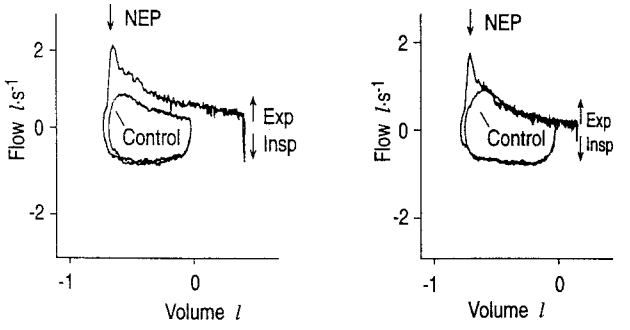

Fig. 11. Flow-volume curves from a patient with chronic obstructive pulmonary disease who had no flow limitation during expiration (left panel), and from one with expiratory flow limitation (right panel). Following a control breath, negative expiratory pressure (NEP) is applied to the airway opening at the onset of expiration. In the absence of expiratory flow limitation, the application of NEP increases flow over the entire duration of expiration, compared to the control breath (left panel). In contrast, in the patient with expiratory flow limitation, a transient increase in flow occurs when NEP is applied at the onset of expiration, but the flows during the remainder of expiration are similar to those during the control breath (right panel). Exp = expiratory. Insp = inspiratory. (From Reference 54, with permission.)

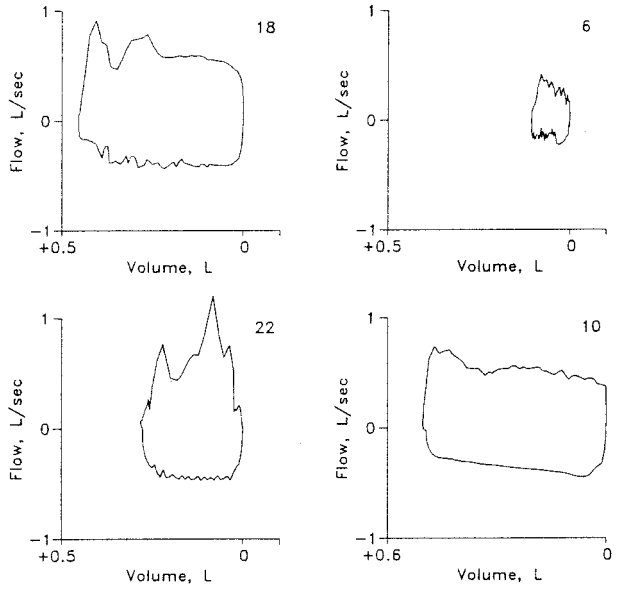

Fig. 12. Flow-volume curves indicating presence of airway secretions. In 4 different patients, a sawtooth pattern is observed in both inspiratory and expiratory limbs of the flow-volume curves. The numbers to the right of each panel represent the number of each patient in the study. (From Reference 55, with permission.)

volume loop also indicate a bronchodilator effect (Fig. 15).

Heliox

Heliox 80/20 is a mixture of 80% helium and 20% oxygen. Heliox 80/20 is about one third less dense than air. Since airway resistance to turbulent flows is related to gas density for a given driving pressure,[59] heliox 80/20 reduces airway resistance and work of breathing in mechanically-ventilated patients.[60] Moreover, heliox improves delivery of aerosolized drugs, including bronchodilators, to the lower airway of mechanically-ventilated patients.[61,62]

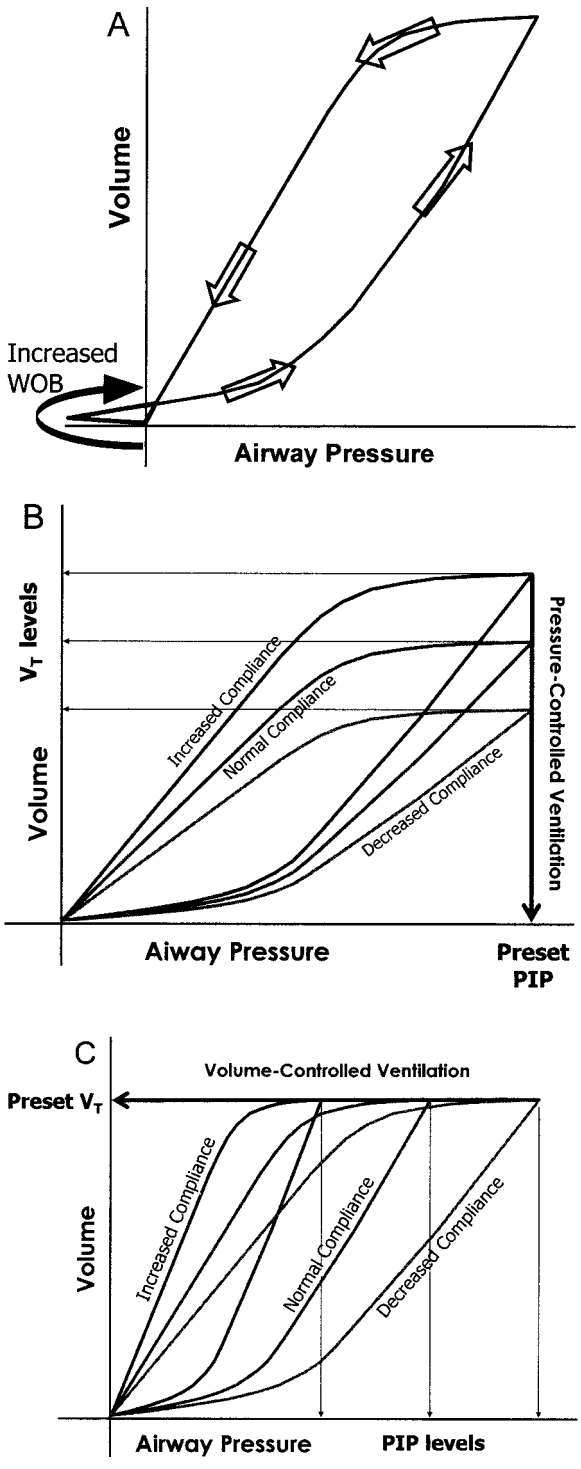

Fig. 13. A: Pressure-volume loop showing excessive patient effort due to inadequate trigger sensitivity. The shape of the pressure-volume curve could indicate changes in respiratory-system compliance. The shape of the pressure-volume curve during volume-controlled ventilation (B) differs from that during pressure-controlled ventilation (C). Panels B and C show alterations in the shape of the pressure-volume curve with alterations in respiratory-system compliance. WOB = work of breathing. V_T = tidal volume. PIP = peak inspiratory pressure.

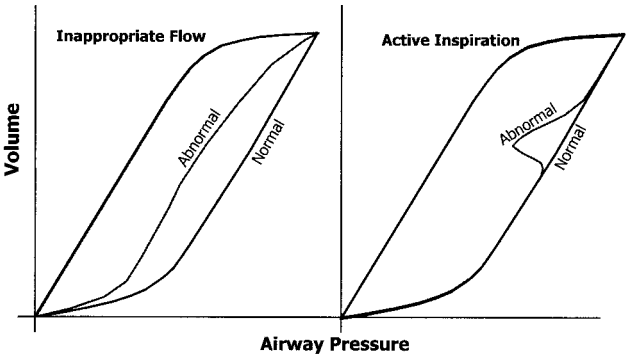

Fig. 14. Pressure-volume loops may also be helpful in detecting an inappropriately low inspiratory flow setting (left) and active patient efforts during inspiration (right).

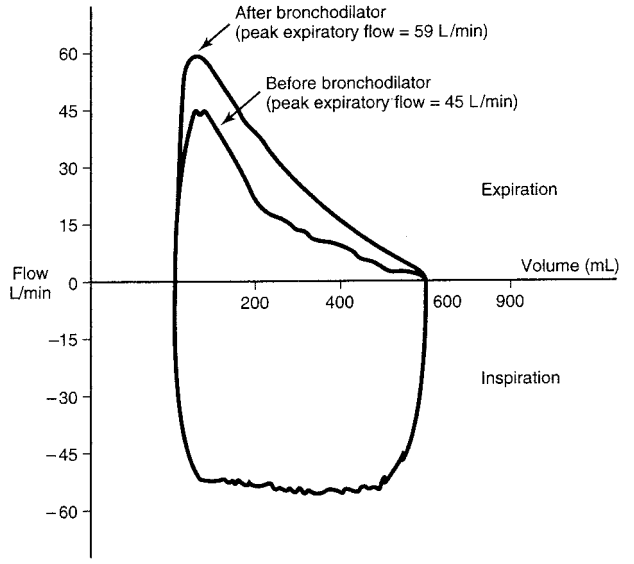

Fig. 15. Flow-volume loops showing a response to bronchodilator administration. The expiratory limb of the curve is concave in patients with expiratory flow limitation. Administration of a bronchodilator aerosol leads to improvement in expiratory flow. (From Reference 18, with permission.)

Summary

A substantial proportion of patients who receive mechanical ventilation have underlying obstructive lung disease, especially COPD. The underlying pathophysiologic mechanisms in patients with obstructive lung disease lead to several challenging problems during mechanical ventilation. Pressure, volume, and flow waveforms, as well as flow-volume, pressure-flow, and pressure-volume waveforms are now routinely available at the bedside. The availability of these waveform displays allows the clinician to recognize changes in a patient's condition at the bedside before clinical signs become overt. Experienced clinicians are well aware of the diagnostic utility of ventilator wave-

forms, and recognition of abnormal patterns could lead to early intervention and appropriate adjustments in ventilator settings. Monitoring of respiratory mechanics, especially airway resistance and PEEPi, and recognition of other abnormalities in ventilator waveforms could be very useful in the diagnosis and management of mechanically-ventilated patients with obstructive lung diseases.

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Discussion

MacIntyre: I have a comment about heliox. You showed that it reduces peak pressure. Part of that, I think, is heliox's effect on airflow resistance. A major role of heliox, though, may be that it allows the emptying of air trapping, which during volume-controlled ventilation would also drop the peak pressure. That may be its major effect—reducing air trapping in intrinsic PEEP.

Let me ask you a more practical question that I get asked in talking about intrinsic PEEP and managing patients with obstructive disease, who have a lot of intrinsic PEEP. We talk about reducing the minute ventilation, and we talk about giving albuterol, as you described, trying to reduce the intrinsic PEEP, but the issue of the inspiratory-expiratory ratio is always a confusing one. The argument would be, "I will shorten the inspiratory time a lot to reduce intrinsic PEEP," but to do that and keep the tidal volume the same, the flow is going to have to go up and so the peak pressure will go up.

My question is, how bad is it to raise the peak pressure—not the plateau pressure, but the peak pressure—as a price to pay for shortening the inspiratory time and maybe giving more expiratory time to allow more emptying of trapped air?

Dhand: Like all your questions, that one is difficult to answer. But I think the effects of reducing the inspiratory time can be difficult to predict. With a patient who has a lot of air trapping it would be helpful if we could get some idea of the plateau pressure. So, if you raise the peak pressure, there may not be a corresponding change in the plateau pressure, When you shorten the inspiration and lengthen the expiration, you've reduced the intrinsic PEEP somewhat. As a result, the plateau pressure will tend to decrease to some extent as well. So you might have a higher peak pressure, but you might have actually lowered the plateau pressure, and so the lungs may not actually see the higher pressure that is being recorded by the ventilator.

On the other hand, reducing the inspiratory time may lead to an increase in the respiratory rate and make matters worse. Then the only solution would be to sedate the patient and try to control the minute ventilation. Also, we know from the study by Leather-

man and Ravenscraft,[1] and Lluís Blanch also showed in his presentation to this conference yesterday,[2] there may be hidden auto-PEEP in patients with airflow obstruction. Initially, as you give them therapy, the intrinsic PEEP might look like it's getting worse, because now the lung units that were not communicating earlier have started communicating and you now see the higher levels of intrinsic PEEP. Again, you have to assess how your patient is doing, and assess their pulmonary mechanics and hemodynamics. But it can be tricky. I completely agree with you.

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MacIntyre: In your practice, do you try shortening inspiratory time as one strategy?

Dhand: Exactly. That's a common strategy to use, and I would especially recommend it with patients who have auto-PEEP and ventilator nontriggering. Another occasion when the inspiratory time is an issue is during mechanical ventilation with some of the Siemens ventilators that don't have adequate inspiratory flow during volume-control ventilation. So our respiratory therapists have solved that problem by never using the volume-control modes with Siemens ventilators; they just use pressure-control. When the flow rate is inadequate, the patient fights the ventilator, and increasing the inspiratory flow rate might reduce their respiratory drive and reduce the patient-ventilator asynchrony. However, decreasing the inspiratory time tends to increase respiratory drive. That's why I think that the effects of shortening the inspiratory time can be difficult to predict.

Nilsestuen: You showed a pressure-volume loop that had the characteristic "figure 8" associated with the trigger effort. I've been frustrated with a lot of the newer ventilators, because with the algorithms they use to plot those curves, the "figure 8" is completely gone, and it doesn't show. At the end of expiration, the graph refreshes and you can no longer see the trigger effort.

The Bear 1000 ventilator used to have very nice pressure-volume loops. In addition the graphics package had a feature that allowed freeze-framing a control breath and then it would plot the current or live image on top. It was a wonderful way of comparing pressure-volume loops before and after changes in the ventilator settings, such as after bronchodilator treatments. It was a *great* graphics option. None of the newer ventilators have picked up on that concept, and I wish they would.

Durbin: Regarding Neil MacIntyre's question and your last answer, the operating room gives us an opportunity to test the experiment of rapid lung inflations. Relatively healthy patients with obstructive disease frequently undergo surgery. The tendency of many of the residents I deal with is to use slow inflations because they don't want to overpressurize the lung; that's exactly the opposite of the strategy we just discussed. So I spend a lot of time cranking up the driving pressure on these anesthesia ventilators, which are basically pressure-driven, constant-flow, volume ventilators. That shortens the inspiratory time and raises the peak airway pressure, but it does not change plateau pressure or tidal volume. The expiratory time increases and gas trapping should be less.

My question, though, relates to the exhaled-CO_2 curve, which is another common monitor in the operating room for judging the adequacy of volume ventilation. How does gas trapping affect end- tidal CO_2 and the shape of the CO_2 excretion curve? Can I use CO_2 monitoring as a monitor of either too-short exhalation and gas trapping, or adequacy of ventilation?

Dhand: No, I have never used that.

Thompson: We've looked at the slope of phase 3 of the volumetric CO_2 waveform,[1] and it does correlate quite well with changes in volume. It's not something that you could use at the bedside to observe the changes.

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Durbin: Also, the CO_2 monitor used in the operating room gives a time-versus-CO_2 waveform rather than a volume-versus-CO_2 waveform, which makes it even more confusing, I think.

Blanch: I think the capabilities and utility of the capnographic indices at the bedside need further research. Now we have fast CO_2 analyzers that plot CO_2 as a function of exhaled volume. That is a major advantage that should allow monitoring the effects of bronchodilators on the CO_2 slope, assessment of physiologic dead space to predict outcome, and the effect of other treatments in patients receiving mechanical ventilation. Capnography is a very interesting monitoring tool because it's noninvasive.

Hess: I have an observation about the slides in which you showed the measurement of auto-PEEP. The set PEEP on the ventilator was always at zero. I think that's an important point.

Something that I often see clinically is a patient with obstructive lung disease and the PEEP on the ventilator is set at 10 cm H_2O, which is nicely counterbalancing the auto-PEEP, and the respiratory therapist does the auto-PEEP measurement and says that there is no auto-PEEP. Everyone thinks that things are fine. If you took away the PEEP applied by the ventilator you would measure 10 cm H_2O of auto-PEEP, which would give a completely different picture of what's happening clinically.

Dhand: Exactly. I tell our therapists that you measure it on whatever PEEP you have set and if you don't see auto-PEEP, then you need to reduce it and recheck it, because you will see the auto-PEEP only if it is higher than your set PEEP. If it is lower—if you have set a PEEP of 10 cm H_2O but the auto-PEEP is 8 cm H_2O—then you are not going to see it.

Hess: I think that's very important.

Dhand: Yes, that's a very important concern. The presence of auto-PEEP will influence the ventilator triggering, and that might become an issue when the set PEEP levels are lowered.

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