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Low measured auto-positive end-expiratory pressure during mechanical ventilation of patients with severe asthma: Hidden auto-positive end-expiratory pressure

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Abstract

To describe the occurrence of low measured auto-end-expiratory pressure (auto-PEEP) during mechanical ventilation of patients with severe asthma.

Observational clinical study.

Medical intensive care unit of a university-affiliated county hospital.

Four mechanically ventilated patients with severe asthma who had low measured auto-PEEP despite marked increase in both peak and plateau airway pressures.

None.

Peak pressure, plateau pressure, and auto-PEEP were measured at an early time point, when airflow obstruction was most severe, and again at a later time after clinical improvement. Auto-PEEP was measured by the method of end-expiratory airway occlusion. From the early to the late time point, there was a marked decrease in peak pressure (76 plus minus 7 to 53 plus minus 6 cm H_2 O; p less than .001) and in plateau pressure (28 plus minus 2 to 18 plus minus 3 cm H_2 O; p less than .001), but only minimal change in auto-PEEP (5 plus minus 3 to 4 plus minus 3 cm H_2 O). The difference between plateau pressure and auto-PEEP decreased between the early and late time points (23 plus minus 1 to 14 plus minus 1 cm H_2 O; p less than .01), even though tidal volume was larger at the late time point. In three patients, low auto-PEEP and a large difference between plateau pressure and auto-PEEP was only seen after expiratory time was prolonged. In these three patients, prolongation of expiratory time resulted in a large decrease in measured auto-PEEP (14 plus minus 4 to 5 plus minus 4 cm H_2 O), but a much smaller change in plateau pressure (31 plus minus 3 to 29 plus minus 3 cm H_2 O).

We conclude that measured auto-PEEP may underestimate end-expiratory alveolar pressure in severe asthma, and that marked pulmonary hyperinflation may be present despite low measured auto-PEEP, especially at low respiratory rates. This phenomenon may be due to widespread airway closure that prevents accurate assessment of alveolar pressure at end-expiration.

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KEY WORDS: asthma; ventilators, mechanical; intermittent positive-pressure ventilation; positive end-expiratory pressure; airway pressure; lung compliance; alveoli; pulmonary emergencies; lungs

The elastic recoil pressure of the respiratory system is normally atmospheric (zero) at end-expiration. However, when the respiratory system fails to deflate to its relaxed volume, the increased end-expiratory lung volume is associated with

positive alveolar pressure at end-expiration [1]. The positive end-expiratory alveolar pressure that results from dynamic hyperinflation has been termed intrinsic PEEP [2,3], or auto-PEEP [1,4]. Auto-PEEP is typically measured by closing the expiratory valve at end-exhalation, allowing alveolar pressure and proximal airway pressure to equilibrate [4]. Similarly, plateau airway pressure is measured with an end-inspiratory airway occlusion and represents the elastic recoil pressure of the respiratory system at end-inspiration [5]. Plateau pressure is the sum of two pressures: the tidal elastic pressure (effective tidal volume/total respiratory system compliance) and end-expiratory alveolar pressure. In the setting of obstructive airway disease and constant tidal volume, changes in plateau pressure primarily reflect changes in end-expiratory alveolar pressure.

There are two important considerations regarding measurement of plateau pressure and auto-PEEP in asthma. First, the marked heterogeneity in airways resistance in severe asthma dictates that alveolar pressure will vary in different regions of the lung [6]. During airway occlusion, there is gas redistribution (pendelluft) and stress relaxation, and the varying pressures in different lung units equilibrate to a final, common pressure. Second, certain lung units may at times fail to communicate with the proximal airway as a result of mucous hypersecretion and increased wall thickness of the peripheral airways [7-10], and the alveolar pressure in these noncommunicating lung units will not directly contribute to the pressure measured during airway occlusion. Therefore, plateau pressure and auto-PEEP are estimations of the average end-inspiratory alveolar pressure and the average end-expiratory alveolar pressure, respectively, of lung units that are in communication with the proximal airway at the time of airway occlusion.

In our experience, most patients with severe asthma who require mechanical ventilation have very high levels of auto-PEEP, often in the range of 10 to 20 cm H_2 O. However, approximate 2 yrs ago, we encountered an asthmatic patient with very severe airflow obstruction who had marked hyperinflation on chest roentgenogram and very high plateau

airway pressure, but surprisingly low measured auto-PEEP. To be certain that this apparent paradox was not due to measurement errors, we prospectively evaluated asthmatic patients who required mechanical ventilation to see if this phenomena would be detected in additional cases. We identified four additional asthmatic patients with severe airflow obstruction and marked hyperinflation on chest roentgenogram who demonstrated a marked increase in plateau airway pressure with low measured auto-PEEP. In three of these patients, low measured auto-PEEP was detected only after expiratory time had been prolonged by a decrease in respiratory rate. With improvement in airflow obstruction, there was a large decrease in plateau pressure and the difference between plateau pressure and auto-PEEP narrowed. In these patients, initial measurements of auto-PEEP appeared to have greatly underestimated end-expiratory alveolar pressure and the degree of pulmonary hyperinflation. We believe that the most likely explanation for the underestimation of end-expiratory alveolar pressure by measured auto-PEEP in severe asthma is widespread airway closure at end-expiration.

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MATERIALS AND METHODS

Between May 1993 and September 1994, 16 consecutive adults who underwent mechanical ventilation for severe asthma at Hennepin County Medical Center were prospectively evaluated by the authors for evidence of clinically important pulmonary hyperinflation with an inappropriately low measured auto-PEEP. Inappropriately low measured auto-PEEP was defined as an auto-PEEP of less than 10 cm H_2 O, with a difference between the plateau pressure and auto-PEEP of more than 20 cm H_2 O at a tidal volume of 8 to 10 mL/cm H_2 O, resulting in a calculated respiratory system compliance of less than 40 cm H_2 O [11,12]. Six patients met this definition of pulmonary hyperinflation with inappropriately low auto-PEEP. However, two of these six

patients were excluded from this study because auto-PEEP had been measured by an automated program of the ventilator (Adult Star, Infrasonics, San Diego, CA) that may not have ensured a sufficiently long duration of end-expiratory airway occlusion in the setting of severe airflow obstruction. The remaining four patients, in whom an initially low measured auto-PEEP was documented using prolonged end-expiratory airway occlusions, form the basis for this report. In three of the study patients, a low measured auto-PEEP with a large difference between plateau pressure and auto-PEEP was observed only after the ventilator's respiratory rate had been decreased in an attempt to reduce the severity of hyperinflation.

Of the ten patients who did not meet the criteria for hyperinflation with low measured auto-PEEP, four patients had improved rapidly and had minimal hyperinflation (plateau pressures of 15 to 20 cm H_2 O, auto-PEEP values of 2 to 4 cm H_2 O) by the time they were seen by the authors. The remaining six patients had clinically important hyperinflation with high auto-PEEP (plateau pressures of 25 to 38 cm H sub 2 O, auto-PEEP values of 12 to 20 cm H_2 O). Four of the latter six patients underwent a reduction in respiratory rate to decrease hyperinflation. Unlike the study patients, this reduction led to a parallel decrease in plateau pressure and auto-PEEP (i.e., the difference between plateau pressure and auto-PEEP did not widen when expiratory time was prolonged).

Data from the four study patients were evaluated at two different time points. The early time point was on the first day of intubation, when airflow obstruction was most severe. In the cases in which airway pressures were measured at two respiratory rates on the first day, early airway pressure data Table 1 were recorded at the (lower) respiratory rate at which an inappropriately low auto-PEEP was detected. The second time point was 1 to 6 days after intubation, when airflow obstruction had improved. All patients were initially sedated and therapeutically paralyzed and remained without spontaneous respiratory activity because of continued

paralysis or sedation when airway pressures were recorded at the later time point.

	Early	Late
Ventilator Settings		
V_T (mL)	713 ± 132	800 ± 82
RR (breaths/min)	7 ± 4	14 ± 1.6[a]
$\dot{V}_I$ (L/min)	95 ± 5.8	105 ± 14
Airway Pressures		
Ppk (cm H_2O)	76 ± 7	53 ± 6[b]
Pplat (cm H_2O)	28 ± 2	18 ± 3[b]
AP (cm H_2O)	5 ± 3	4 ± 3

V_T, tidal volume; RR, respiratory rate; $\dot{V}_I$, inspiratory flow rate; Ppk, peak airway pressure; Pplat, plateau airway pressure; AP, auto-positive end-expiratory airway pressure (auto-PEEP).

[a] $p < .05$; [b] $p < .001$.

Table 1. Airway pressures in four automatic patients with low measured auto-PEEP (mean plus minus SD)

Ventilators used to support the patients included the Bear 2 (Bear Medical Systems, Riverside, CA) in two patients, the Servo 900C (Siemens-Elema, Solna, Sweden) in one patient, and the Adult Star (Infrasonics) in one patient. (In the latter case, auto-PEEP was measured by a manual airway occlusion technique rather than by the ventilator's automated program.) The following data were recorded at both time points: tidal volume; respiratory rate; inspiratory flow rate; peak pressure; plateau pressure; auto-PEEP; and respiratory system compliance. The latter variable was calculated as tidal volume —(plateau pressure times CF)/(plateau pressure—auto-PEEP), where CF is the ventilator circuit compliance factor (mL/cm H_2 O) used to determine the volume of gas present in the

ventilator circuit during the end-inspiratory pause (to calculate effective tidal volume).

Auto-PEEP was measured by end-expiratory airway occlusion [4]. In all cases, auto-PEEP was measured during prolonged manual occlusions at end-expiration, ensuring a final stable pressure. When the Bear 2 or Servo 900C ventilators were used, auto-PEEP was assessed by visual observation of the ventilator manometer during manual occlusion of the exhalation valve at end-expiration (Bear) or by depression of the end-expiratory pause button (Servo). With the Adult Star ventilator, we used a modification of the airway occlusion method to ensure an adequate duration of the end-expiratory occlusion. The ventilator circuit was disconnected from the endotracheal tube after a tidal breath, and was immediately replaced with a T-adaptor attached to an aneroid manometer (Boehringer, Riverside, CA). The open end of the T-adaptor was then manually occluded precisely at end-expiration, allowing auto-PEEP to be measured by observation of the manometer. In all four patients, end-expiratory airway occlusion was maintained for several seconds and was not terminated until the needle of the manometer had reached a final, stable position.

Plateau airway pressure was measured by observation of the ventilator manometer during an end-inspiratory pause. With the Servo ventilator, plateau pressure was measured by depression of the end-inspiratory pause button until a static pressure had been reached. When the Bear or Adult Star ventilator was used, a pause duration of 1.0 sec was used.

The collection and review of data used in this study were approved by the Institutional Review Board of Hennepin County Medical Center, and the need for informed consent was waived. Data analysis was performed, using the Student's t-test for paired data. Data are expressed as mean plus minus SD.

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RESULTS

Initial chest roentgenograms uniformly showed marked hyperinflation, and severe airflow obstruction was corroborated by arterial blood gases that demonstrated a $Paco_2$ of 91 plus minus 19 torr (12.1 plus minus 2.5 kPa) and a pH of 7.15 plus minus 0.07 at a time that machine-delivered minute ventilation was 8.5 plus minus 1.5 L/min. Repeat chest roentgenograms were not obtained, but repeat arterial blood gases at the later time point were improved, with a $Paco_2$ of 40 plus minus 4 torr (5.3 plus minus 0.5 kPa) and a pH of 7.44 plus minus 0.04 at a minute ventilation of 10.4 plus minus 2.6 L/min.

Ventilator settings and airway pressures at the early and later time points are given in Table 1. Peak pressure and plateau pressure were significantly higher at the early vs. the later time point, but measured auto-PEEP at the two time points was similar. The difference between plateau pressure and auto-PEEP was much larger at the early time point (23 plus minus 1 vs. 14 plus minus 1 cm H_2 O; p less than .01) Figure 1, even though tidal volume was slightly larger at the later time point. Accordingly, respiratory system compliance was significantly lower at the early time point (27 plus minus 5 vs. 51 plus minus 9 mL/cm H_2 O; p less than .01).

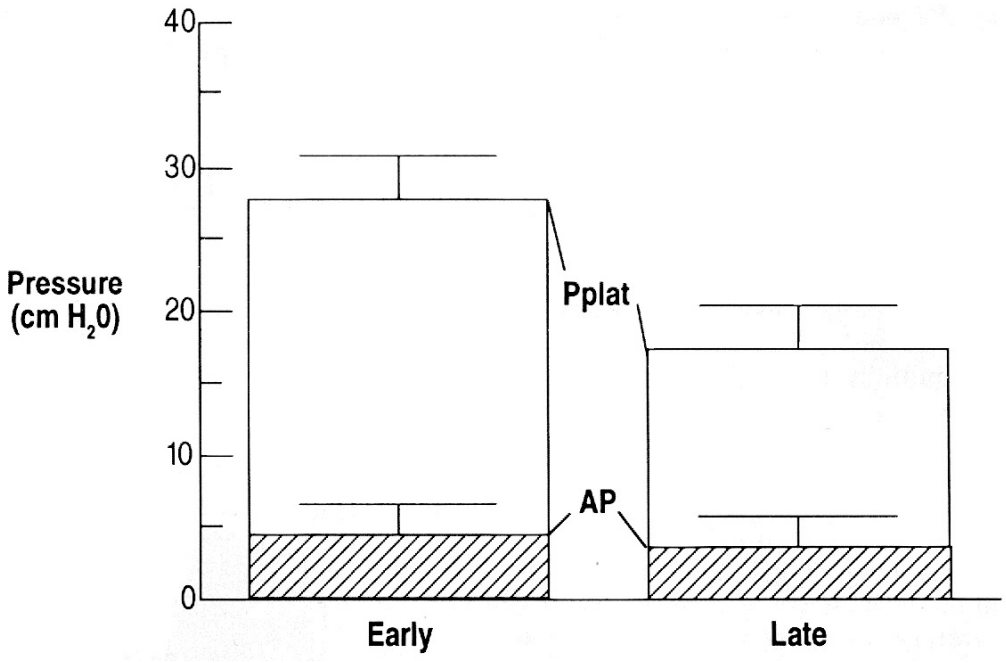

Figure 1. Plateau airway pressure (Pplat) and auto-positive end-expiratory pressure (AP) in four mechanically ventilated patients with asthma measured initially when airflow obstruction was most severe (Early) and after clinical improvement (Late). Data are expressed as mean plus minus SD.

In three cases, airway pressures at the early time point were measured before and after expiratory time was prolonged by a reduction in respiratory rate from 16 plus minus 2 to 5 plus minus 2 breaths/min. Prolongation of expiratory time resulted in a measured decrease in auto-PEEP (14 plus minus 4 vs. 5 plus minus 4 cm H_2 O; p less than .001). However, there was minimal difference in peak pressure (79 plus minus 7 vs. 81 plus minus 8 cm H_2 O) or plateau pressure (31 plus minus 3 vs. 29 plus minus 3 cm H_2 O) at the two respiratory rates. The effects of prolongation in expiratory time on plateau pressure and measured auto-PEEP for individual patients are shown in Figure 2.

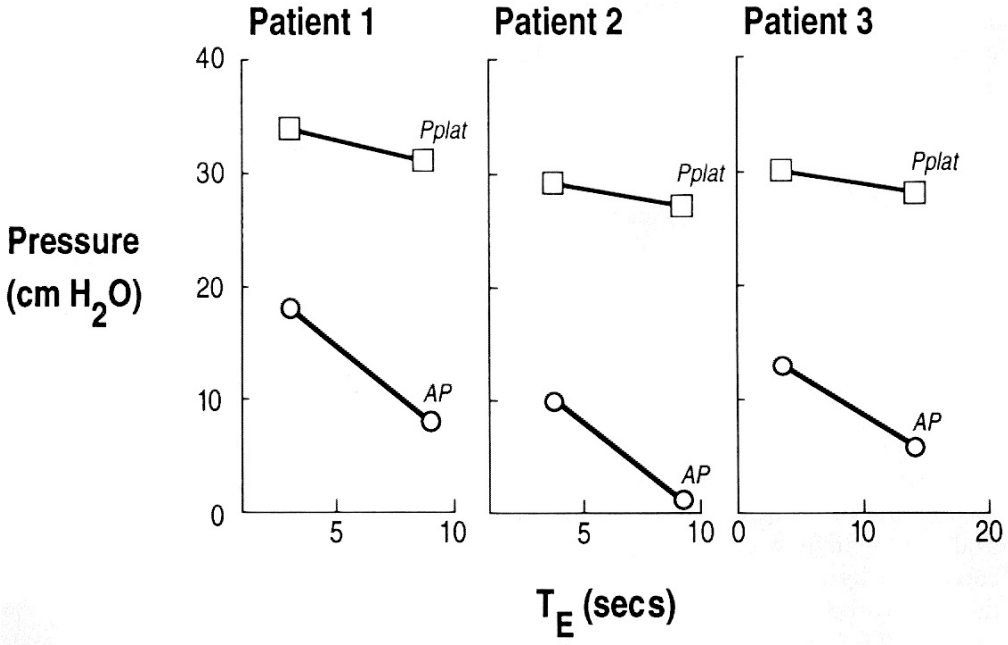

Figure 2. Effect of prolongation of expiratory time (TE) on plateau pressure (Pplat) and auto-positive end-expiratory pressure (AP) in three patients with severe asthma. In each patient, prolongation of expiratory time was associated with a much larger decrease in measured auto-positive end-expiratory pressure than in plateau pressure.

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DISCUSSION

When patients with severe airflow obstruction are mechanically ventilated, pulmonary hyperinflation is indicated by an increase in auto-PEEP and a parallel increase in plateau airway pressure [13,14]. The asthmatic patients described in this study initially had a marked increase in plateau pressure, indicating clinically important hyperinflation, although

measured auto-PEEP was surprisingly low. With improvement in airflow obstruction, plateau pressure decreased and the difference between plateau pressure and auto-PEEP narrowed. Initially, the low measured auto-PEEP appeared to have underestimated end-expiratory alveolar pressure and the severity of pulmonary hyperinflation. There are three possible explanations for the high plateau pressure and low measured auto-PEEP values in our patients with severe asthma: a) technical errors in measuring auto-PEEP or plateau pressure (or both); b) a reduction in compliance of the lungs or chest wall; or c) widespread airway closure at end-expiration that prevented accurate assessment of end-expiratory alveolar pressure.

Technical errors can lead to inaccurate assessment of the elastic recoil pressure of the respiratory system during an airway occlusion. If airway occlusions are too brief to allow equilibration of alveolar and proximal airway pressure, measured auto-PEEP could be erroneously low and plateau pressure erroneously high. In each of our four patients, auto-PEEP was measured with prolonged manual airway occlusions that ensured sufficient time for equilibration of pressure. Plateau pressure was measured with a 1.0-sec airway occlusion in three patients and with an even longer occlusion in the other patient. In normal individuals, postocclusion airway pressure rapidly reaches a static value. In the setting of airflow obstruction, there is greater time-constant inhomogeneity and a longer time is required to reach a final static pressure. However, the patients in this study showed minimal differences (less than equals 1 cm H_2 O) in plateau pressures as measured with a 1- and 2-sec occlusion. (The 2-sec occlusion represents the maximal duration of occlusion allowed by the ventilator.) Therefore, the high plateau pressure recorded on the first day of mechanical ventilation in our study was unlikely to have significantly overestimated the end-inspiratory elastic recoil pressure of the respiratory system. Another technical consideration is the site of end-expiratory airway occlusion. One study [15] found that measured auto-PEEP may be higher when the airway is occluded at the endotracheal tube instead of distally at the exhalation valve. In

three of our patients, a conventional distal occlusion was used. However, this factor does not explain the measurement of low measured auto-PEEP with a large difference between plateau pressure and auto-PEEP only after expiratory time was prolonged Figure 2.

A second possible explanation for the initially large difference between plateau pressure and auto-PEEP is decrease in lung or chest wall compliance. None of our patients had roentgenographic evidence of a parenchymal infiltrative process that would reduce lung compliance, and the use of paralyzing agents ensured that respiratory muscles were passive and that chest wall compliance was not decreased. While pulmonary hyperinflation may have caused end-inspiratory lung volume to approach total lung capacity, the initial decrease in calculated respiratory system compliance was not solely due to tidal ventilation on the upper, flatter portion of the respiratory system's curvilinear pressure-volume curve [16]. In three of our patients, a large difference between plateau pressure and auto-PEEP, and low calculated respiratory system compliance, was only seen after expiratory time was prolonged. A prolonged expiratory time should have decreased lung volume and caused tidal ventilation to occur on a lower, steeper portion of the pressure-volume curve [16]. Although calculated respiratory system compliance was initially low, this finding could be explained by an underestimation of end-expiratory alveolar pressure by measured auto-PEEP. Rossi and associates [17] demonstrated that failure to account for the presence of auto-PEEP results in an underestimation of respiratory system compliance. Similarly, if measured auto-PEEP had underestimated alveolar pressure at end-expiration, the calculated compliance of the respiratory system would be falsely low.

We believe that the most likely explanation for the initially low measured auto-PEEP, and the large difference between plateau pressure and auto-PEEP, is widespread airway closure at end-expiration. During an airway occlusion, the pressure recorded in the proximal airway reflects only the alveolar pressure in communicating lung units. If a large percentage of conducting airways was closed at endexhalation, and the

alveolar pressure in these noncommunicating lung units was greater than alveolar pressure in communicating units, then the pressure measured during an end-expiratory airway occlusion (measured auto-PEEP) would be lower than the end-expiratory alveolar pressure in most of the lung Figure 3. Even though end-expiratory airway closure in asthma could cause measured auto-PEEP to be misleadingly low, it would not necessarily affect the utility of the plateau pressure as an indicator of lung hyperinflation. Airways that are closed at the end of expiration could remain closed throughout the ventilatory cycle, especially if the airway was occluded by a tenacious mucous plug, with atelectasis prevented by collateral ventilation [18]. Alternatively, occluded airways could reopen during a positive-pressure inspiration, creating a "ball-valve" effect [8,10], when the pressure in the airway exceeded the critical opening pressure of the small airways [19]. Both processes could occur in different regions of the lung, or in the same lung unit with different ventilatory cycles. If airways opened during inspiration, then the resultant high end-inspiratory alveolar pressure in the affected units would be reflected in the end-inspiratory occlusion pressure (plateau pressure). If airways remained closed throughout the ventilatory cycle, overdistention of the remaining communicating lung units might still result in an increase in plateau pressure. Therefore, the high plateau pressure and low measured auto-PEEP that were initially seen in our patients would be consistent with widespread airway closure at end-exhalation.

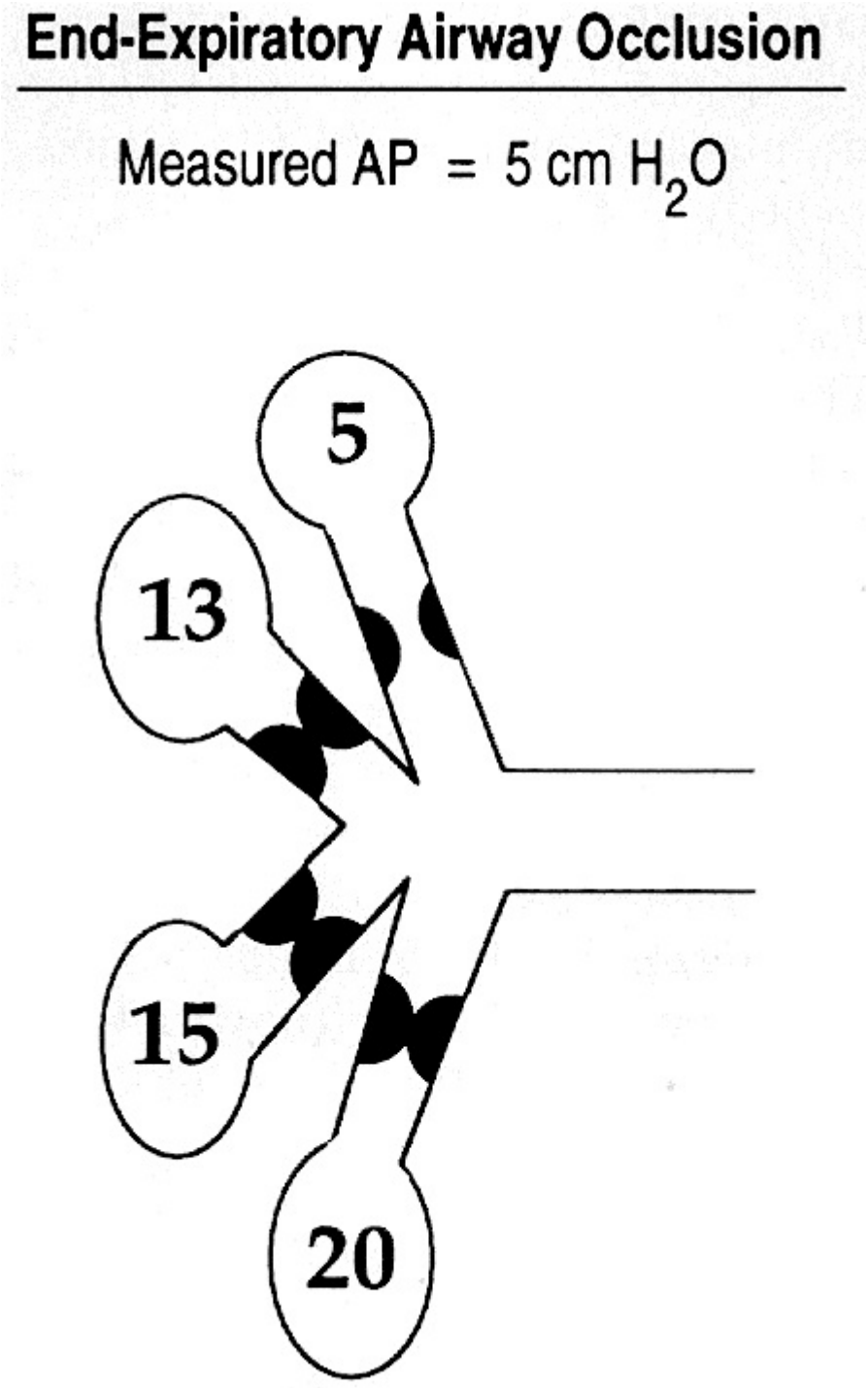

Figure 3. A hypothetical model to explain underestimation of end-expiratory alveolar pressure by measured auto-positive end-expiratory pressure (AP) in severe asthma. Alveolar pressure (cm H2 O) in four different lung units is shown. At end-expiration, only the upper lung unit is in communication with the central airway, causing the airway occlusion pressure (measured auto-positive end-expiratory pressure) to be much lower than end-expiratory alveolar pressure in the other hyperinflated, but noncommunicating, lung units.

Airway narrowing in asthma is due to a combination of infiltration with inflammatory cells, bronchial vascular congestion and edema, connective tissue deposition, thickening of the basement membrane, muscle hypertrophy, mucous gland and goblet cell prominence, epithelial hyperplasia, and intraluminal mucous and cellular debris [7].

In addition, loss of surfactant in small airways greatly increases the airway's surface tension [20]. These factors, together with even a relatively modest degree of smooth muscle shortening [21], may markedly increase the likelihood of small airway closure in severe asthma. Widespread airway closure in severe asthma is supported by several lines of evidence. Failure of post mortem lungs to collapse is a characteristic finding in fatal asthma [7,22]. Woolcock and colleagues [8,9] measured lung volumes by helium dilution and plethysmography during the course of severe exacerbations of asthma and concluded that airway closure was likely. A similar conclusion was reached by Tuxen and associates [10] in a study of mechanically ventilated patients with severe asthma. These investigators assessed the relative contributions of dynamic hyperinflation and gas trapped behind obstructed airways to increased end-expiratory lung volume. The volume of gas due to dynamic hyperinflation was measured by subtracting the delivered tidal volume from the total volume of gas passively exhaled during a prolonged apnea. The amount of residual trapped gas was estimated by subtracting the predicted functional residual capacity from postapnea lung volume. (Postapnea lung volume was estimated by computer analysis of posterior-anterior and lateral roentgenograms.) These investigators [10] concluded that most of the increased end-expiratory lung volume in severe asthma was due to trapped gas that could not be passively exhaled, regardless of the duration of expiratory time.

In three of our patients, both plateau pressure and measured auto-PEEP were initially increased, and the difference between plateau pressure and auto-PEEP was appropriate for the delivered tidal volume. When expiratory time was prolonged by a reduction in respiratory rate, there was a large decrease in measured auto-PEEP, but minimal change in plateau pressure. This marked increase in the difference between plateau pressure and auto-PEEP likely reflected a greater degree of airway closure at the longer expiratory time. Tuxen and associates [10] suggested that, during mechanical ventilation for severe asthma, most lung units likely communicate with the central airways by the end of inspiration, but then

progressively close off during expiration. Prolongation of expiratory time may permit more airways to close during expiration.

Prolongation of expiratory time is commonly used to reduce the magnitude of lung hyperinflation, thereby decreasing end-inspiratory lung volume and the risk of barotrauma [10,23]. However, prolongation of expiratory time is ineffective in reducing the amount of gas trapped behind occluded airways. Because measured auto-PEEP reflects only the end-expiratory alveolar pressure in communicating lung units, it is unlikely that patients who have minimal measured auto-PEEP will benefit from prolongation of expiratory time. For patients with asthma who have high plateau pressure and low measured auto-PEEP, a reduction in end-inspiratory lung volume may be better accomplished by reducing tidal volume than by decreasing respiratory rate.

Underestimation of end-expiratory alveolar pressure by measured auto-PEEP in asthma could lead to an inaccurate assessment of the degree of pulmonary hyperinflation. Clinically, we use plateau pressure as the primary indicator of the severity of hyperinflation during mechanical ventilation of patients with severe asthma. In our patients, plateau pressure was high initially and decreased markedly over time, paralleling clinical improvement in airflow obstruction Figure 1. Thus, plateau pressure may serve as a useful indicator of the severity of hyperinflation, even when measured auto-PEEP does not.

In summary, measured auto-PEEP may underestimate the degree of pulmonary hyperinflation and end-expiratory alveolar pressure in some mechanically ventilated asthmatic patients, especially when expiratory time is prolonged. This phenomenon may be due to airway closure before the completion of exhalation, which in effect seals off alveoli under positive pressure from the pressure recording site in the proximal airway. Underestimation of end-expiratory alveolar pressure by measured auto-PEEP should be suspected when a patient with severe asthma is found to have an unexpectedly large difference between plateau pressure and auto-PEEP (i.e., a low calculated respiratory system compliance). In these

patients, changes in the severity of pulmonary hyperinflation in asthma may be better evaluated by serial determinations of plateau pressure (at constant tidal volume) than by measurement of auto-PEEP. In severe asthma associated with high plateau pressure and minimal auto-PEEP, a reduction in respiratory rate or an increase in peak inspiratory flow rate may be of limited benefit for decreasing the severity of hyperinflation, and a reduction in end-inspiratory lung volume is best accomplished by decreasing tidal volume.

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