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How Accurately Can Clinicians Identify the Lower Inflection Point?

ABSTRACT & COMMENTARY

The "lung-protective" ventilatory strategy currently in vogue for managing acute respiratory distress syndrome (ARDS) is based on maintaining tidal ventilation above the lower inflection point (Pflex) on the static pressure-volume curve. However, in this study estimates of Pflex from the same data curves by six different clinicians varied by 4-9 cm H_2O for a given curve.

As data supporting the existence of ventilator-induced lung injury accumulate from both animal and human studies, clinicians are changing the way they manage patients with ARDS. The currently advocated "lung-protective strategy" for ventilatory management includes the use of tidal volumes of 5-7 mL/kg, the avoidance of alveolar (i.e., end-inflation static) pressures exceeding 35 cm H_2O, permissive hypercapnia, and the use of enough positive end-expiratory pressure (PEEP) to keep tidal ventilation above the lower inflection point (Pflex) on the static pressure-volume (P-V) curve (*See Figure*). Because Pflex is thought to represent alveolar and airway opening, ventilating the lung at pressures and volumes above this point is intended to avoid lung injury from the shear stresses created by cyclically opening and closing these structures.

In order to apply it in an individual patient, this strategy assumes that Pflex can be identified clinically on the P-V curve. O'Keefe and colleagues at Harborview Medical Center in Seattle sought to determine whether this was the case by constructing P-V curves in critically ill patients and having six clinicians attempt independently to identify Pflex in each case. The eight patients included in the study were all in the surgical-trauma ICU, and each had a known risk factor for developing ARDS. Their mean PaO_2/FIO_2 ratio was 152 torr (range, 80-320 torr), and their ARDS risk factors included sepsis (4 patients), multiple long-bone fractures (2), pulmonary contusions (2), and multiple transfusions (2). All were studied within 24 hours of onset of the risk factor.

O'Keefe et al constructed the P-V curves by gradually inflating the patients' lungs manually using a 3L calibration syringe, while simultaneously recording pressures and volumes on a commercial inline computerized respiratory monitoring system. All patients

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INSIDE

were pharmacologically paralyzed at the time of the measurements. PEEP was discontinued for 5 minutes, and measurements at 50-mL tidal volume increments were made until 15 mL/kg was delivered or static pressure reached 60 mm Hg. Three inflation procedures were done in each patient within 10 minutes, and the data were plotted onto a single curve. Direct printouts of the P-V curves were then presented to five intensivists and one respiratory therapist, who independently determined Pflex visually from the curves.

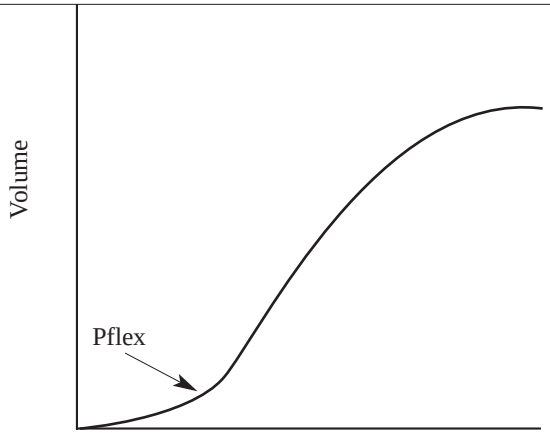
Estimated mean Pflex in these eight critically ill surgical patients ranged from 9.5-20.8 cm H_2O, with standard deviations of 1.3-3.4 cm H_2O. No individual clinician systematically determined Pflex to be significantly higher or lower than the other clinicians, but the estimated Pflex values varied substantially, with an interindividual range for each patient of 4-9 cm H_2O. O'Keefe et al conclude that estimation of Pflex by this technique is imprecise and potentially of little use in selecting minimum PEEP levels in individual patients. (O'Keefe GE, et al. *J Trauma* 1998;44[6]:1064-1068.)

COMMENT BY DAVID J. PIERSON, MD

Although current understanding of ventilator-induced lung injury supports the concept that PEEP should be set above Pflex in patients with acute lung injury, the present study casts doubt as to whether this can be done meaningfully in individual patients. I have taken informal "show-of-hands" polls of physicians and respiratory therapists attending several conferences on ARDS management, and my impression is that the majority of clinicians do not actually construct P-V curves on their patients. Such curves are not currently used routinely in patient management at the institution where this study was performed.

Figure

Pressure-Volume Curve in ARDS



Hypothetical static pressure-volume curve of the respiratory system in a patient with ARDS. The lower inflection point (Pflex), above which compliance increases, is believed to represent the opening of collapsed alveoli and small airways.

I think there are three reasons for this. First, the P-V curves used in lectures and to illustrate data in published papers tend to look like the hypothetical curve in the figure. However, curves from actual patients are often less smooth, and it is usually a judgment call as to where the slope begins to increase. Even the strongest advocates of performing individual P-V curves report a certain percentage of patients in whom a distinct Pflex cannot be identified. Second, construction of P-V curves by the technique described in this study is cumbersome and fairly labor-intensive. Admittedly, attempts are being made to speed and simplify the process, but such streamlined techniques are not currently available to the clinician. Finally, to construct a reproducible P-V curve in the ICU requires that the patient be completely motionless, which for practical purposes means pharmacologically paralyzed. While short-term use of muscle relaxants is necessary for certain procedures and may be justi-

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fiable for the purpose discussed here, there are numerous compelling reasons for avoiding their use in critically ill patients whenever possible.

Most studies on patients with medical illnesses predisposing to ARDS have determined Pflex to be in the range of 8-10 cm H_2O. Accordingly, it makes sense to use at least about 10 cm H_2O of PEEP in such patients when ARDS develops. I think it is clinically acceptable to make this assumption, rather than attempting to construct P-V curves in all patients, when managing ARDS according to the current "lung-protective" strategy. It is important to realize, however, that different patient groups may have different requirements for PEEP, fluid support, and other aspects of management, as illustrated by the considerably higher mean Pflex values determined in the critically ill surgical-trauma patients included in this study. ❖

Potential problems with the clinical use of pressure-volume curves in determining PEEP levels in patients with ARDS include:

a. the requirement that the patient be paralyzed.
b. the inability to identify a lower inflection point on some curves.
c. imprecision in identifying Pflex.
d. the cumbersomeness and time requirements for making the measurements.
e. All of the above

Most patients with ARDS secondary to medical illnesses have lower inflection points in the range of:

a. 2-4 cm H_2O.
b. 5-7 cm H_2O.
c. 8-10 cm H_2O.
d. 12-15 cm H_2O.
e. 18-20 cm H_2O.

Antibiotic Delay Increases Mortality in Ventilator-Associated Pneumonia

ABSTRACT & COMMENTARY

Synopsis: *Delay in initiating or adding appropriate antibiotic coverage while awaiting results of bronchoalveolar lavage cultures is associated with higher mortality in patients developing pneumonia during mechanical ventilation. Gram-negative organisms resistant to third-generation cephalosporins and methicillin-resistant S. aureus were the most commonly missed pathogens in pre-BAL antibiotic coverage.*

Source: Kollef ME, Ward S. *Chest* 1998;113(2): 412-420.

Kollef and ward identified 130 consecutive patients during a recent year who underwent fiberoptic bronchoscopy with bronchoalveolar lavage (BAL) in evaluation of presumed ventilator-associated pneumonia (VAP) in a university teaching hospital. The effect of BAL culture results on antibiotic management was examined. Forty-one patients had not received any antibiotics in the 48 hours preceding BAL, 17 had been started on antibiotics during the previous 48 hours, and the remaining 72 had been receiving antibiotics for at least 48 hours prior to the BAL. Sixty patients had culture results that indicated a potential pathogen accounting for VAP. These infected patients were classified as receiving appropriate antibiotics if the presumed pathogen was sensitive to the prescribed antibiotics, or inappropriate if there was a pathogen that was not covered.

Based on the BAL results and clinical course, the antibiotic regimen was unchanged in 51 patients, antibiotics were started or changed in 51, and antibiotics were discontinued in 28. The groups were similar in admitting diagnoses, age, sex, premorbid lifestyle, and severity of illness (APACHE II). Overall, 40% of the studied patients expired. The mortality of the patients who were continued on current treatment was 33%, while those in the group requiring a change experienced a mortality of 61%. Patients having antibiotics stopped following a negative BAL culture died at a rate of 14%. Patients with resistant organisms identified died at a rate of 59%, while those with sensitive organisms experienced a 31% mortality. Death specifically from VAP was 24% in those requiring a change in therapy, 7.8% in those continued on treatment, and only 3.6% in patients in whom antibiotics were stopped (sterile BAL).

The BAL fluid from most patients requiring a change in antibiotic regimen grew gram-negative organisms resistant to a previously administered cephalosporin (72% of changes) or methicillin-resistant *Staphylococcus aureus* (MRSA) (27%). Patients requiring a change in therapy were more likely to have bacteremia, septic shock, or an empyema. There was no difference in the duration of mechanical ventilation or in overall hospital stay. In this study, no additional hospital days could be attributed to the development of VAP. The clinical recommendation of Kollef and Ward is to treat VAP empirically with appropriate antibiotics rather than wait for BAL culture results. Antibiotic coverage in patients already receiving antibiotics should include treatment for resistant gram-negative bacteria and MRSA.

COMMENT BY CHARLES G. DURBIN, Jr., MD, FCCM

This is an important study identifying that the development of VAP in patients already on antibiotic treat-

ment results in a worse outcome. As expected, resistant organisms predominated and were associated with a worse outcome. Unfortunately, the recommendations of Kollef and Ward remain speculative. Patients with VAP requiring an antibiotic change were indeed more ill than those not receiving antibiotics or those receiving the appropriate antibiotics at the time of culture. Because those not receiving and not started on antibiotics were included with those not requiring a change, these "healthy" patients (not having VAP) contaminated the unchanged group, making the difference reach significance. The antibiotic treatment recommendations (cover for resistant organisms while awaiting BAL results) make sense but are not proven in this study. ❖

Resistant organisms in BAL culture:

a. are most likely with hemodynamic changes.
b. are more likely with multiple lobe processes.
c. are more likely in patients previously treated with antibiotics.
d. occur most often in patients without lobar infiltrates.
e. occur most often in patients with complicating empyema.

VAP patients with BAL culture results suggesting that antibiotic coverage is inadequate:

a. should have all antibiotics stopped for 48 hours and a repeat BAL performed.
b. should have antibiotics added to broaden coverage.
c. experience a high mortality.
d. have a significantly longer length of stay than others with VAP.
e. are older and more likely male.

Special Feature

Ventilatory Support in Patients with COPD: An Update

By Mark T. Gladwin, MD

In the rapidly changing field of mechanical ventilation, how best to manage patients with chronic obstructive pulmonary disease (COPD) or acute severe asthma remains one of the most active and important topics. In this brief review, using a question-and-answer format, I discuss several aspects of ventilatory support in patients with severe COPD, emphasizing recent developments.[1]

Noninvasive Ventilation for Acute Exacerbations

- Can noninvasive positive-pressure ventilation (NPPV) be used safely and effectively in COPD patients with severe respiratory acidosis (for example, arterial PCO_2 > 70 mm Hg and pH < 7.20)?
- How much inspiratory positive pressure is needed to achieve adequate unloading of the ventilatory muscles?
- Is it necessary to apply positive end-expiratory pressure (PEEP) to counteract the effects of air trapping and auto-PEEP in order to achieve clinical success?

When initiating NPPV, inspiratory and expiratory pressures are typically set at low levels for patient comfort and acceptance, and gradually raised to levels between 8 and 20 cm H_2O inspiratory pressure and between 0 and 6 cm H_2O expiratory pressure. Two recent studies of NPPV in patients with severe exacerbations of COPD used different levels of pressure support with equally positive results. Kramer and colleagues[2] used an average inspiratory pressure of 11 cm H_2O delivered by nasal mask with an average expiratory pressure of 3 cm H_2O, while Brochard and colleagues[3] used an inspiratory pressure of 20 cm H_2O but no PEEP, delivered via face mask. Thus, the application of PEEP (called expiratory positive airway pressure, EPAP, on some ventilators) may not be necessary in all cases. Gastric distention with air is considered unlikely with pressure support levels less than 25 cm H_2O.[4]

The application of modest amounts of PEEP will reduce the inspiratory work of spontaneous breathing associated with dynamic hyperinflation and auto-PEEP. This is supported by a trial of continuous positive airway pressure (CPAP) without additional inspiratory pressure in patients with COPD and acute ventilatory failure, which demonstrated improvements in dyspnea, inspiratory effort, and arterial blood gas values.[5]

Numerous uncontrolled studies, as well as case series using historical controls, have consistently demonstrated an improvement in ventilatory failure with NPPV. Recent prospective randomized trials have demonstrated similar positive results. Brochard et al[3] randomized 85 patients with severe COPD and acute ventilatory failure to noninvasive inspiratory positive airway pressure of 20 cm H_2O or to standard therapy without NPPV. Intubation rates in the two patient groups were 26% and 74%, respectively, mortality 9% vs. 29%, complications 16% vs. 45%, and hospital length of stay 23 ± 17 days vs. 35 ± 33 days. Kramer et al[2] studied 31 patients with ventilatory failure, and in the subgroup with COPD the rate of intubation was 9% with NPPV as compared to 67% in the standard treatment group.

With the advent of NPPV, mandatory criteria for endotracheal intubation are becoming more difficult to identify. While apnea or agonal respiration, uncontrolled agitation, uncorrectable life-threatening hypoxia, hemodynamic instability or serious dysrhythmia, and a high

risk for aspiration remain relative exclusion criteria for NPPV and, thus, indications for endotracheal intubation, a falling arterial pH secondary to ventilatory failure and rising $PaCO_2$ is less so. Thus, while a pH of 7.25 or less has historically been considered a reasonable "line in the sand" beyond which intubation was necessary, this may now be a manageable degree of ventilatory failure with properly applied NPPV. Indeed, in the Brochard study,[3] the average arterial pH in patients in whom intubation was successfully avoided was as low as 7.28 ± 0.1, with average $PaCO_2$ 70 ± 12 mmHg.

The level of consciousness acceptable for noninvasive ventilation is likewise controversial. While patients with severe obtundation have frequently been excluded from studies of NPPV, other authors have documented success even in patients with this finding.[6]

Dynamic Hyperinflation and Auto-PEEP

- How serious a problem is auto-PEEP in hospitalized patients with COPD?
- What is the difference between static and dynamic auto-PEEP?
- What is the best way to quantitate dynamic hyperinflation?
- Does externally applied PEEP help to maintain the patency of collapsible airways in patients with severe COPD?

No concept in the management of acute exacerbations of COPD is more important than the concept of dynamic hyperinflation and auto-PEEP.[1,7,8] Whenever there is insufficient emptying of alveoli during exhalation, as occurs with rapid respiratory rates in persons with normal airways or in persons with airway obstruction due to emphysema or reactive airways disease, the alveoli and the lung will be overdistended at end-exhalation. This elevation of end-expiratory resting lung volume (functional residual capacity, FRC) above normal is termed "dynamic hyperinflation" and is accompanied by an elevated net static recoil pressure of the respiratory system. This pressure has been called auto-PEEP, occult-PEEP, or intrinsic-PEEP because it cannot be measured on the ventilator manometer (because the manometer communicates with atmospheric pressure during exhalation) and is produced intrinsically by the patient and not set by the ventilator.

Methods for quantitating dynamic hyperinflation and auto-PEEP include the following:

- Directly measure the excess gas trapped at end-exhalation.[9] Measure expired volume during a 30- to 50-second period of apnea following a mechanically delivered breath. Subtraction of the delivered tidal volume from the total expired volume will give the volume of trapped air. This technique requires a completely relaxed patient without inspiratory effort.
- Apply the end-expiratory occlusion technique. Manually occlude the ventilator's expiratory port at end-expiration, or with some ventilators activate an end-expiratory pause button on the ventilator control panel. An occlusion of about one second allows time for the trapped air to equilibrate across most obstructed airways. The pressure measured on the ventilator's pressure manometer during this maneuver is the auto-PEEP. This technique is only applicable in patients who are not actively attempting to breathe.
- Directly measure esophageal pressure (which is equivalent to pleural pressure) using an esophageal balloon and pressure transducer.
- Monitor inspiratory flow, peak inspiratory pressure, or end-inspiratory plateau pressure while increasing dialed-in PEEP by increments of 3 cm H_2O. As long as flow, peak inspiratory pressure, or plateau pressure does not change, the set (external) PEEP is below the level of auto-PEEP. If flow decreases or peak inspiratory or plateau pressure goes up with the addition of external PEEP, the auto-PEEP level has been exceeded. Only peak inspiratory pressure can be measured if patients are actively attempting to breathe.

Static PEEP refers to the measurement of PEEP under static conditions and dynamic PEEP under dynamic conditions. A static condition occurs when there is no inspiratory effort and measures the pressure or volume of gas emptying from the lungs. During the end-expiratory occlusion method, a prolonged occlusion allows for emptying of even the most obstructed regions of the lung (that is, those with the longest time constants). This also occurs with the direct measurement of total trapped gas. These two static measurements will more accurately reflect the entire volume or pressure of trapped gas (as there is time for almost complete emptying), and will thus more accurately represent the detrimental effects of auto-PEEP on intrathoracic pressure and venous return than will more rapid approximations.

Dynamic measurements can be made with an esophageal balloon measuring intrathoracic pressure and the simultaneous measurement of airflow. When auto-PEEP is present and the patient attempts to breathe, the intrathoracic (esophageal) pressure will fall while initially no flow will occur. Eventually the pressure drops below the level of auto-PEEP and flow begins. The amount of negative intrathoracic pressue required to initiate flow is the dynamic auto-PEEP.

Understanding this mechanism leads to further observations. First, airflow should occur when the intrathoracic pressure exceeds the auto-PEEP of the lung units with the least auto-PEEP. This value may be substantial-

ly less than the level of auto-PEEP measured under static conditions and can be measured in a spontaneously breathing patient. Furthermore, this measurement more accurately reflects the elevated work of breathing associated with auto-PEEP because this is the minimal threshold (hurdle) that must be overcome to initiate inspiratory flow. Titrating PEEP in increments of 3 cm H_2O until flow occurs or peak or plateau inspiratory pressures increase will also reflect the lowest auto-PEEP of any lung unit and is a dynamic measurement.

Auto-PEEP likely contributes to deaths from pulseless electrical activity (PEA) in hospitalized patients. The patient with COPD is at greatest risk for this complication immediately after intubation. The urgency of the situation often leads to overzealous manual ventilation prior to connection to the ventilator. Large tidal volumes and rapid respiratory rates increase lung volume and shorten expiratory time, preventing adequate exhalation and potentiating dynamic hyperinflation. In the setting of non-elective emergency intubation, the reduction in cardiac output associated with dynamic hyperinflation is often compounded by volume depletion and sedation.

Hypotension occurs in 25% of all emergency intubations, and more frequently in patients with COPD and baseline hypercapnia. The transition from manual to mechanical ventilation carries the iatrogenic hazards of attempting to apply physiological tidal volumes and respiratory rates and ill-advised attempts to drive $PaCO_2$ to the normal range—interventions that critically reduce expiratory time.

It is instructive to consider case reports of patients with COPD who develop PEA and do not respond to aggressive resuscitation.[10,11] In several cases, after resuscitative efforts had been discontinued, the arterial line began to pick up a pulse. Discontinuation of mechanical ventilation allowed exhalation to occur and intrathoracic pressure to decrease, resulting in enhanced venous return and restoration of cardiac output. A recent review of 89 in-hospital cardiac arrests,[12] 35 of which involved PEA, revealed no discernible etiology in 18, and 13 (74%) of these patients were subsequently found to have COPD by history, pulmonary function testing, or at autopsy. By contrast, only 11% of the remaining patients had COPD. This suggests that unrecognized auto-PEEP may be a common cause of PEA.

Application of external PEEP in the presence of auto-PEEP will reduce the patient's work of spontaneous breathing[13] but can also increase dynamic hyperinflation. External PEEP should only be applied to the spontaneously breathing patient to decrease work of breathing. The impact of auto-PEEP on mechanical work of breathing, both on and off the ventilator, is significant. With dynamic hyperinflation, at end-expiration there will be a significant pressure gradient between the (positive) alveolar pressure and (zero) pressure in the airway distal to the critical closure point. The process of inspiration involves reversing this pressure gradient, such that alveolar pressure is less than airway pressure and flow reverses. If end-expiratory alveolar pressure (that is, auto-PEEP) remains, for example, at 10 cm H_2O, intrapleural pressures must exceed -10 cm H_2O in order to drop alveolar pressure to less than atmospheric and initiate inspiratory flow. Therefore, auto-PEEP represents an inspiratory threshold load that must be overcome. Furthermore, in the presence of dynamic hyperinflation, breathing occurs at higher lung volumes, such that inspiration occurs at a higher portion of the pressure-volume curve, where there is a greater inward elastic recoil of the overexpanded chest wall.

The mechanically ventilated patient with dynamic hyperinflation must first overcome this inspiratory threshold load, and, depending on the triggering mechanism of the ventilator, further reverse flow (flow-triggered) or lower the airway pressure (pressure triggered) to initiate the delivery of a breath. This can be detected at the bedside by observing chest wall expansion and accessory muscle activity that do not trigger a ventilated breath. Airway and esophageal pressure tracings reveal negative inspiratory excursions that do not trigger a breath, alternating with those that do. Carefully applying extrinsic PEEP increases airway pressure and reduces the pressure gradient required to reverse flow on inspiration. Applied PEEP can be titrated to a level at which every inspiratory effort triggers a breath, resulting in sharp reductions in the patient's work of breathing.

Patients with emphysema, characterized by compliant or floppy airways, poorly tethered by damaged elastic fibers, develop an early "equal pressure point" as the positive extramural pressure exceeds the elastic recoil forces of the airway and the positive intramural airway pressure. Theoretically, the application of extrinsic PEEP less than the original level of auto-PEEP should serve to prevent this early dynamic airway closure at the equal pressure point by maintaining a positive airway pressure that counterbalances the positive extramural pressure that surrounds the airways.

Unfortunately, this concept that applied PEEP thus "stents" open airways and facilitates lung emptying is not supported by clinical studies. Application of external PEEP rarely leads to actual reductions in lung volume. As the level of applied PEEP approaches that of auto-PEEP, lung volume actually increases. This is in keeping with the waterfall analogy of auto-PEEP,[1] where a waterfall represents the critical airways narrowing, the

water above the behind the dam represents the auto-PEEP, and the water beneath the waterfall represents the pressure at the airway. As the downstream water level rises above that of the waterfall, the water backs up behind the waterfall. In fact, the greatest risk of applied PEEP in the COPD patient is inducing further hyperinflation with cardiovascular compromise.

Progressive increments of PEEP compared with 0 PEEP induce hemodynamic impairment when levels of applied PEEP exceed 80-85% of measured auto-PEEP. It is therefore reasonable to judiciously apply PEEP to about 80% of the measured auto-PEEP level, to reduce work of breathing associated with patient-initiated mechanical ventilation. However, there is no rationale for its use during controlled mechanical ventilation when there is no patient inspiratory effort (such as in the paralyzed patient), or in an attempt to "stent" open airways to reduce lung volume.

Identifying clinical assessments that actually reflect alveolar distention and limit the magnitude of this distention should prevent complications of dynamic hyperinflation. Plateau pressure is measured by occluding the proximal airway at end inspiration, allowing the peak proximal airway pressure to equilibrate across airways with high resistance. Peak inspiratory pressure falls to a lower post-occlusion pressure that more accurately reflects the elastic recoil of the respiratory system and thus alveolar distention. In patients with COPD and asthma, peak airway pressure is highly dependent on inspiratory airflow resistance and peak inspiratory flow, while plateau pressure is independent of flow-resistive properties unless the pause time is inadequate to allow complete equilibration or the airways are completely obstructed.

It has been recommended that plateau pressures be kept below 35 cm of water, although this remains theoretical. Alternatively, limiting end-expiratory volume (the excess volume exhaled in addition to tidal volume during a period of apnea) to less than 1.4 liters[14] may be relevant to patients with asthma and COPD, in that hypotension and barotrauma are uncommon in patients with severe asthma, when VEI is less than this. Unfortunately, the measurement of VEI requires a 30- to 60-second complete exhalation, which clinically can only be achieved with deep sedation, usually with neuromuscular blockade.

Administration of Bronchodilators to Mechanically Ventilated Patients

- Are nebulizers more effective than metered-dose inhalers (MDIs)?
- Where in the ventilator circuit should the aerosol generator be placed for optimal delivery of drugs to the patient?
- What steps can the clinician take to maximize bronchodilator delivery during mechanical ventilation?

Beta-adrenergic and anticholinergic bronchodilators can be effectively delivered to the mechanically ventilated patient via small-volume nebulizers (SVN) or MDIs. These medications, when dosed and delivered effectively, will reduce airway resistance (measured by peak and plateau inspiratory pressures) and dynamic hyperinflation.

Delivery of bronchodilator aerosols can be improved by a number of measures under control of the clinician.[1,15,16] First, medication should be delivered at a distance from the endotracheal tube in the inspiratory limb of the ventilator circuit to avoid impaction and deposition of larger aerosol particles. Second, humidification of the ventilator circuit reduces aerosol delivery by one-third to one-half, likely by increasing particle size, resulting in particle impaction in the circuit. Finally, the ventilator settings will affect delivery. In a lung model, delivery was enhanced by spontaneous breathing (CPAP mode), larger tidal volumes, and an increase in duty cycle (total inspiratory time/duration of total breathing cycle). Some authors recommend a breath hold after an MDI actuation to increase duty cycle. This recommendation is reasonable provided dynamic hyperinflation is modest and the clinician is cognizant that increases in duty cycle will increase dynamic hyperinflation.

There has been considerable debate about whether nebulizers are more effective than metered-dose inhalers in ventilated patients. Numerous studies support the efficacy of MDIs provided spacer devices are used, the MDI is actuated immediately prior to inspiratory flow with a 30-second to 1-minute pause between actuations, and the above general recommendations are followed. Using these techniques, four puffs (90 mcg/puff) of albuterol reduced airway resistance to the same degree as eight and 16 puffs, without the rise in heart rate that occurred with the higher doses.[17] Advantages of MDIs over nebulizers are lower cost, freedom from contamination, and ease of dosing.

Inspiratory Pressure and Work of Breathing During Weaning

- What is the effect of the endotracheal tube on work of breathing?
- Is it necessary to provide pressure support during trials of spontaneous breathing?

This remains an unclear and controversial area. Recent work by Straus and colleagues[18] suggests that the work of breathing through a size 7- to 9-mm internal diameter endotracheal tube is the same during a T-piece trial as it is following extubation. Using the acoustic reflection method, they measured, in 14 successfully extubated patients, the resistance of the endotracheal tube and of the supraglottic airway during a two-hour wean-

ing T-piece trial and again following extubation. While endotracheal tube resistance was greater than supraglottic airway resistance, work of breathing was the same with and without the endotracheal tube. Straus et al conclude that a two-hour trial of spontaneous breathing through an endotracheal tube, without any ventilator support, is similar to the work of breathing following extubation. Therefore, pressure support would likely provide excessive support. Application of continuous positive airway pressure (CPAP) at 5 cm H_2O would increase functional residual capacity (assuming that auto-PEEP did not exceed this level) but would not provide excessive support. Flow triggering would reduce the work imposed by dead space in the ventilator and tubing.

The advantage of a trial of spontaneous breathing without any positive inspiratory pressure is further supported by the results of two much-discussed weaning studies,[19,20] demonstrating that even difficult to wean patients may be successfully weaned using two-hour trials of spontaneous breathing via either a T-piece or CPAP of 5 cm H_2O. ❖

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Aerosol delivery to an intubated patient is increased by which of the following?

a. Placement of the delivery device as close to the endotracheal tube as possible.
b. Ensuring that the inspiratory circuit is completely humidified during delivery.
c. Use of large tidal volumes.
d. Shortening inspiratory time.
e. Use of a small-volume nebulizer instead of a metered-dose inhaler.

In Future Issues:

Are Aortic and Radial Artery Pressures the Same on Vasopressors?