Ventilation in Obstructive Lung Disease

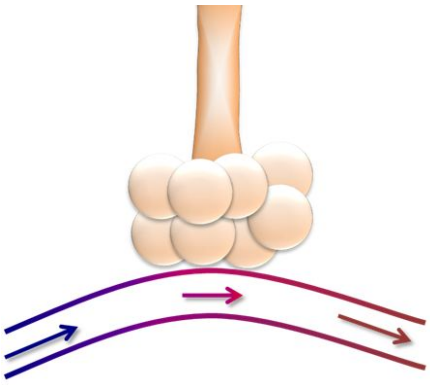
Readings adapted from Wilcox, Susan R., et al. *Mechanical Ventilation in Emergency Medicine.* Springer, 2019

Introduction

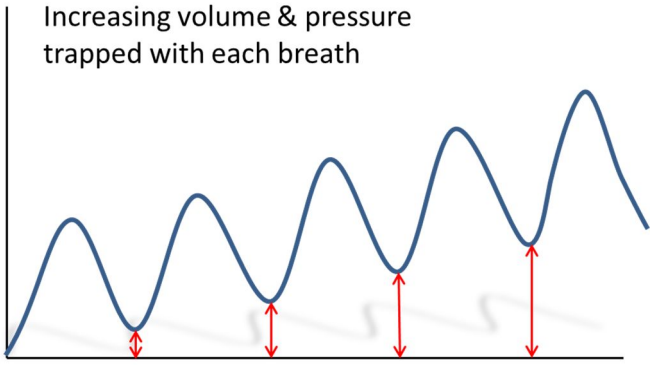
We will address two broad categories of obstructive lung disease: asthma and chronic obstructive pulmonary disease, which includes chronic bronchitis and emphysema.

Asthma

In asthma, the patient has a constriction of the bronchial smooth muscles in the airways, leading to reversible air trapping. This is indicated in the schematic. Note that the bronchial muscles do not extend into the small airways.



Intubation of an asthmatic is a dreaded complication of this illness, as asthmatics can deteriorate rapidly on the ventilator without close monitoring and active management. The goal with a ventilated asthmatic is to prevent breath-stacking or autoPEEP, and the hemodynamic instability that can result.



Before discussing the ventilator management of asthma, clinicians should note that intubation of an asthmatic should trigger even more active management with medications, rather than less. Intubated asthmatic patients should continue to receive aggressive treatment with bronchodilators, steroids, magnesium, as well as deep sedation and possibly even neuromuscular blockade in the initial hours after intubation, in an effort to relax the chest wall musculature and gain control of the situation.

Please note that neuromuscular blockade only works on skeletal muscle and therefore, will not bronchodilate smooth muscle in the airways. In addition, it is very critical to be aware of the patient's intravascular volume status, as the excess positive pressure can lead to hemodynamic collapse. Moreover, the excess pressure, including the auto-PEEP, can result in barotrauma, such as the development of a pneumothorax very quickly in this patient population.

The ventilator screen below demonstrates the effects of reactive airways disease on pulmonary mechanics. This patient had unexpected bronchospasm after being intubated. Note the elevated peak inspiratory pressure (PIP) of 45 despite the relatively low tidal

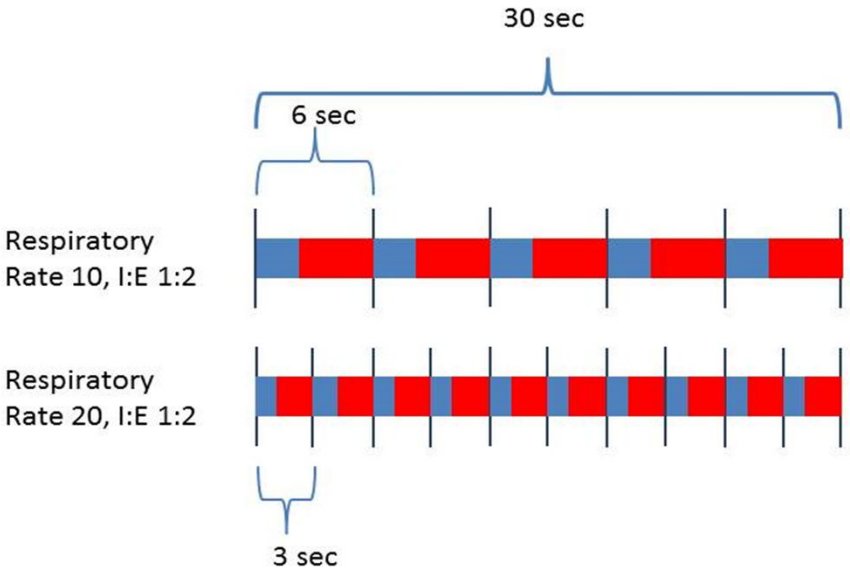
volume of 365. The patient's resistance was too high for her to even receive the full tidal volume, as the ventilator was only able to deliver 320ml before stopping.

Checking the plateau pressure (Pplat) confirmed that this was a resistance problem, rather than a pure compliance problem. Her PIP was 39 at the time the inspiratory hold was performed, but her Pplat was only 28. The delta between 39 and 28 indicates a significant resistance component.

This patient was treated with continuous bronchodilators with rapid improvement in the bronchospasm. Her PIP returned to normal within minutes.

Four ventilator maneuvers increase expiratory time, namely, decreasing the respiratory rate, decreasing the I:E ratio, decreasing the inspiratory time, or increasing the inspiratory flow. Of these, decreasing the respiratory rate is the most effective means to allow more time to exhale.

The figure shows a schematic of 30 seconds with two patients, set with the same I:E ratio of 1:2. The first patient has a rate of 10 breaths per minute, allowing 6 seconds per breath cycle. The second patient has only 3 seconds per breath cycle, given the respiratory rate of 20. The blue represents inspiration, the red the time for exhalation. Note that even with the same I:E, the lower rate offers a substantially longer time to exhale.



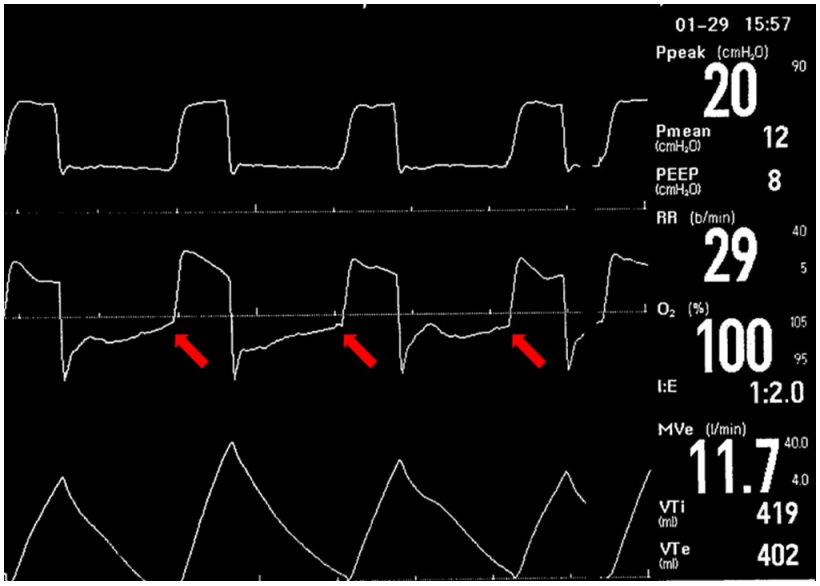
In looking further at this diagram, one can imagine the effects of changing the I:E ratio, the inspiratory flow, or the I time. Fig 9.3 shows a hypothetical example of the effects of these changes in a patient on volume control. In a given patient, the exact values will vary, but the purpose of the illustration is to show the relationship among the parameters of I:E, inspiratory time, and inspiratory flow.

Expressed in I:E ratio		Expressed in inspiratory time	Expressed in inspiratory flow
1:3		0.75 sec	90 L/min
1:2		1 sec	70 L/min
1:1		1.5 sec	50 L/min

In addition to a slow respiratory rate, a low I;E ratio, a short inspiratory time and/or a fast inspiratory flow rate, asthmatics should also be ventilated with low tidal volumes.

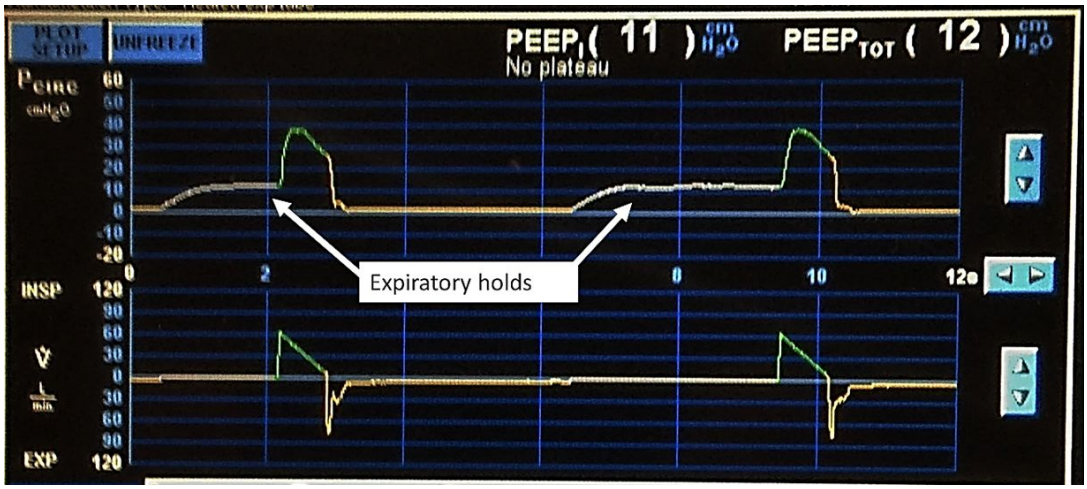
Considering that the larger the tidal volume, the more the patient has to exhale, this is fairly intuitive.

In monitoring an intubated asthmatic, looking for air trapping is key. In the vent tracing below, note that the flow tracing, in the middle, does not return to the baseline before the next breath. (Red arrows) This represents that the patient is still exhaling when the next breath is given, leading to air trapping. Seeing this pattern on the ventilator can be an early clue that the patient is air trapping. If you were caring for this patient, how would you address this air trapping?



In this patient, you could first decrease the respiratory rate, or increase sedation if the patient is over-breathing. The I:E ratio is only 1:2, so changing the I time to make a ratio of 1:3 or 1:4 is also appropriate. Also continued treatment with bronchodilators to decrease the bronchospasm associated with this disease will also mitigate the excess auto-PEEP.

Recall that to quantify the pressure exerted by air trapping, one should check for autoPEEP by checking an expiratory hold button on the mechanical ventilator. In this tracing, what is the autoPEEP, or the intrinsic PEEP? What is the total PEEP?



The intrinsic PEEP is 11, and the total PEEP is 12. This indicates that the patient was only set on 1 of PEEP (an unusual - and not recommended - setting, used in this circumstance for demonstration purposes only.)

Thus, to set the ventilator for an asthmatic, select a low tidal volume of 6-8 mL/kg of predicted body weight. The respiratory rate should be low, less than 20 breaths per minute, and often around 10. The I:E ratio should be changed to 1:3 or less. PEEP should be set at 5 cmH2O. The FiO2 should be down-titrated as tolerated. These patients continue to receive heavy sedation, possibly NMB if required, continuous bronchodilators, and close monitoring for breath stacking and autoPEEP. AutoPEEP should be monitored periodically or after any ventilator change with an expiratory hold. Arterial blood gases (ABGs) should be checked to ensure that the patient is being adequately ventilated.

Permissive hypercapnia is the concept of tolerating a $PaCO_2$ > 40 mm Hg and a pH > 7.20 to 7.25 for the sake of achieving another goal. In the case of asthma, the goal is to allow time to exhale and prevent air-trapping. Permissive hypercapnia is a reasonable strategy, especially early in ventilating the asthmatic.

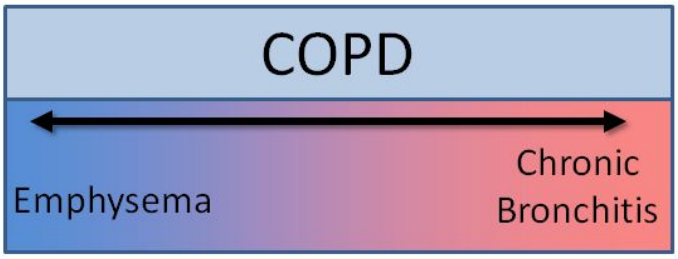
Initial Ventilator Settings in Asthma

Tidal Volume	6-8 ml/kg PBW
Respiratory Rate	6 - 14 breaths/minute, allowing for permissive hypercapnia
PEEP	~ 5 cmH_2O
FiO_2	Decrease as tolerated, $SpO_2 \geq 92\%$

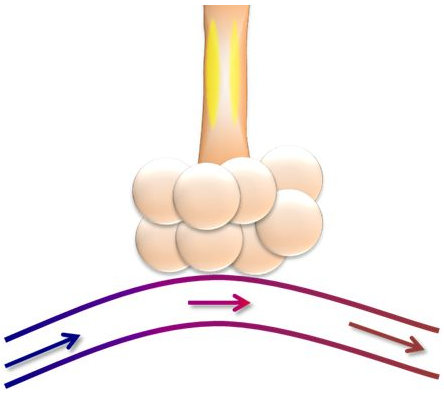
The following ventilator screen demonstrates these settings. The patient is set at 6ml/kg at 350 mls, with a respiratory rate of 14, a PEEP of 5, and a FiO_2 40%. Note, however, that the patient is not synchronous with the ventilator and is taking large tidal volumes. This can be a very dangerous situation, leading to worsening air-trapping and possibly hemodynamic compromise. This patient needs to be deeply sedated and neuromuscular blockade administered if needed. Additionally, the patient should continue to receive bronchodilators and all other appropriate medical treatments.

COPD

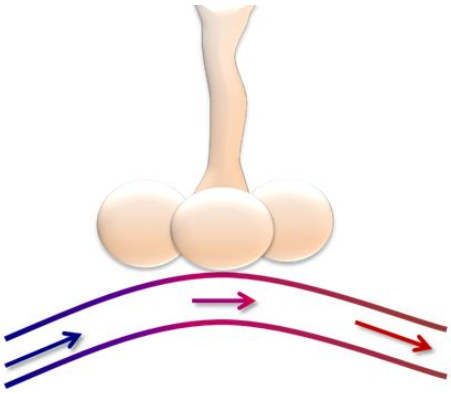
There are two types of obstructive lung disease falling under the umbrella of COPD, namely, chronic bronchitis and emphysema. While some patients may have one or the other, many will exist on the continuum.



Chronic bronchitis can resemble the asthmatic schematic above, with the notable exception that muscles hypertrophy and are not entirely reversible. Additionally, chronic bronchitis is associated with increased mucous production.



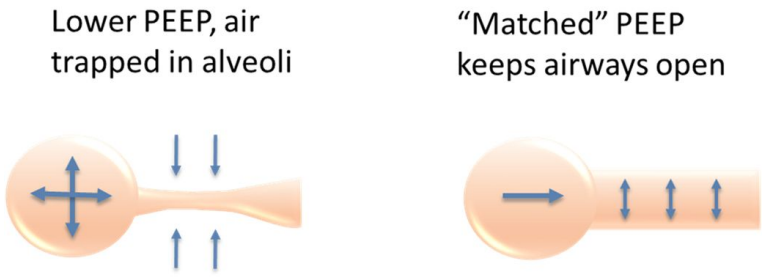
Emphysema is a disease of parenchymal destruction. Not only is there loss of alveoli, resulting in decreased surface area, or decreased diffusion area (leading to an increased DLCO), but the small airways can become floppy due to the loss of other tissues holding them open.



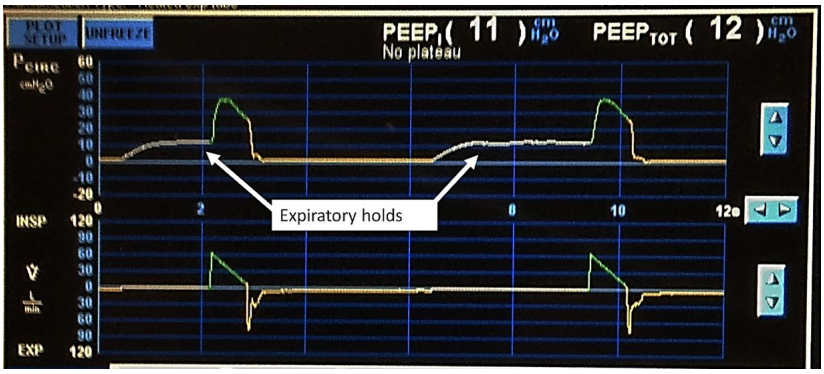
Understanding the pathophysiology of COPD is important for considering how to best ventilate these patients. It should be noted, however, that most patients with COPD have some mixing of elements of chronic bronchitis and emphysema. These conditions exist on a spectrum rather than a dichotomy.

Most patients with COPD are now managed with BPAP, with improved outcomes over intubation. However, on occasion, a patient with COPD is not a candidate for BPAP or fails to improve with a trial of BPAP, mandating intubation and invasive mechanical ventilation. Many of the principles that apply in mechanical ventilation for asthma also apply in COPD. Both are obstructive diseases, and in both processes, the patients require adequate time to exhale. Therefore, low tidal volumes, low rates, and low I:E ratios are appropriate. However, a key difference involves the role of PEEP.

Patients with COPD are at high risk of developing autoPEEP. Due to their obstructive disease, they require additional time to exhale. However, the mechanism of obstruction can differ between asthma and COPD, especially COPD with emphysematous changes as illustrated above. With the destruction of parenchyma, the small airways can collapse with exhalation, trapping air behind. In this circumstance, this trapped air leads to autoPEEP. Increasing the set PEEP, to match the autoPEEP, is not necessarily an intuitive solution. However, as illustrated by the diagram below, increasing the PEEP to prevent collapse of these small airways can allow the patient to exhale more fully.



Reexamine the tracing of the ventilator screen from the Asthma section, imaging that this patient has COPD. If this patient has 11 of autoPEEP, or intrinsic PEEP, what PEEP would you select?



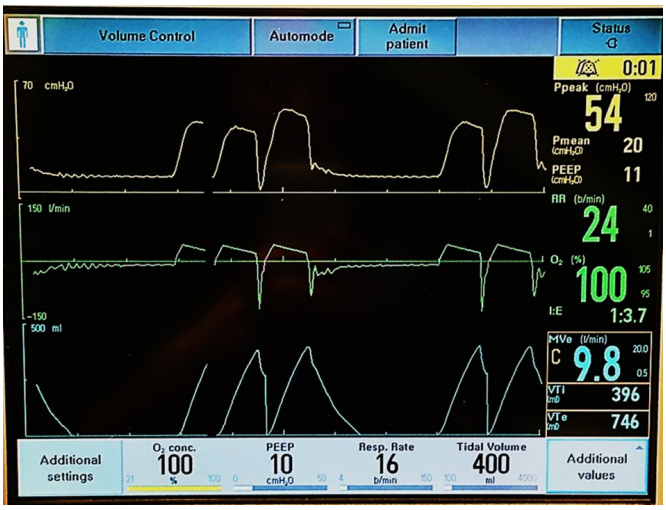
To match the autoPEEP, 11cm H_2O would be an appropriate PEEP selection.

Lastly, patients with COPD are often chronically hypoxemic. Indications of chronic hypoxemia physical exam findings of chronic hypoxemia can be demonstrated with nail clubbing. Additionally, can include an elevated hemoglobin level on the CBC, indicating the patient's compensation for their chronic lung disease. Because these patients are baseline hypoxemic, and ventilation is often a relatively greater issue for them than hypoxemia, the oxygen saturation for a patient with COPD should be targeted at 88-92% in most circumstances. This is increasingly important as more data demonstrating the risks of hyperoxia continue to accumulate.

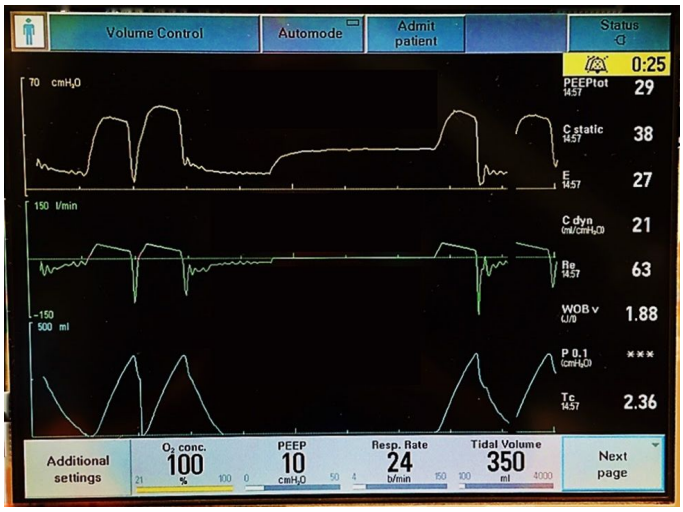
Initial Ventilator Settings in COPD

Tidal Volume	6-8 ml/kg PBW
Respiratory Rate	6 - 20 breaths/minute, allowing for permissive hypercapnia
PEEP	5 - 15 cm H_2O - may need to match autoPEEP for patients with significant emphysematous physiology
FiO_2	Decrease as tolerated, SpO_2 target 88-92%

This ventilator screen demonstrates a patient with COPD with severe dyssynchrony. The PIP is 54, indicating severe pathology. The irregular waveforms indicate the dyssynchrony. The patient is set at a respiratory rate of 16 but is breathing at 24.



An expiratory hold was performed and demonstrated a total PEEP of 29, with a set PEEP of 10. This indicates a high autoPEEP of 19. Therefore, this is a very high-risk situation. This patient was deeply sedated, NMB administered, and the ETT was disconnected from the ventilator to allow the patient to exhale.



Once sedated and relaxed, the patient was placed back on the ventilator at a rate of 12, with frequent expiratory holds to check the autoPEEP.