Basics of Mechanical Ventilation
Objectives

To understand:

- How positive pressure ventilation helps
  - Reduce the work of breathing
  - Restore adequate gas exchange

- The basics of
  - Invasive positive pressure ventilation (IPPV)
  - Noninvasive positive pressure ventilation (NIPPV)

- The principles of bedside monitoring
  - Pressure and volume alarms
  - Flow and pressure time curves
Physiopathology of Respiratory Failure

\[ \Delta \frac{V_A}{Q} \]

\[ \uparrow \text{Resistance}_{AW} \]
\[ \downarrow \text{Compliance}_{R} \]

\[ \uparrow \text{Work of Breathing} \]
\[ \uparrow \text{VE} \]
\[ \uparrow \text{VO}_2 \]
\[ \uparrow \text{VCO}_2 \]
\[ \downarrow \text{pH} \]

\[ \text{Hypoxemia} \]
\[ \text{Hypercapnia} \]
\[ \text{Fatigue} \]
\[ \text{Neuromuscular disorders} \]

AW=Airway; R=respiratory system; VE = minute ventilation, VO2 = Oxygen consumption, VCO2=carbon dioxide production
Indications and Rationale for Initiating IPPV

- Unprotected and unstable airways (e.g., coma)
  - Intubation and IPPV allows to
    - Secure the airways
    - Reduce the risk of aspiration
    - Maintain adequate alveolar ventilation

- Hypercapnic respiratory acidosis
  - IPPV and NIPPV
    - Reduce the work of breathing and thus prevents respiratory muscle fatigue or speeds recovery when fatigue is already present
    - Maintain adequate alveolar ventilation (prevent or limit respiratory acidosis as needed)

- Hypoxic respiratory failure
  - IPPV and NIPPV help correct hypoxemia as it allows to
    - Deliver a high FiO₂ (100% if needed during IPPV)
    - Reduce shunt by maintaining flooded or collapsed alveoli open

- Others
  - Intubation to facilitate procedure (bronchoscopy), bronchial succioning
Important Pitfalls and Problems Associated with PPV

Potential detrimental effects associated with PPV

- Heart and circulation
  - Reduced venous return and afterload
  - Hypotension and reduced cardiac output
- Lungs
  - Barotrauma
  - Ventilator-induced lung injury
  - Air trapping
- Gas exchange
  - May increase dead space (compression of capillaries)
  - Shunt (e.g., unilateral lung disease - the increase in vascular resistance in the normal lung associated with PPV tends to redirect blood flow in the abnormal lung)
Important Effects of PPV on Hemodynamics

**Decreased preload**

- Positive alveolar pressure $\rightarrow$ $\uparrow$ lung volume $\rightarrow$ compression of the heart by the inflated lungs $\rightarrow$ the intramural pressure of the heart cavities rises (e.g., $\uparrow$ RAP) $\rightarrow$ venous return decreases $\rightarrow$ preload is reduced $\rightarrow$ stroke volume decreases $\rightarrow$ cardiac output and blood pressure may drop. This can be minimized with i.v. fluid, which helps restore adequate venous return and preload.

- Patients who are very sensitive to change in preload conditions (e.g., presence of hypovolemia, tamponade, PE, severe air trapping) are particularly prone to hypotension when PPV is initiated.

**Reduced afterload**

- Lung expansion increases extramural pressure (which helps pump blood out of the thorax) and thereby reduces LV afterload.

- When the cardiac performance is mainly determined by changes in afterload than in preload conditions (e.g., hypervolemic patient with systolic heart failure), PPV may be associated with an improved stroke volume. PPV is very helpful in patients with cardiogenic pulmonary edema, as it helps to reduce preload (lung congestion) and afterload. As a result stroke volume tends to increase.
Generally speaking, the effects of PPV on the cardiac chamber transmural pressures vary in parallel with:

- Airway pressure (e.g., ↑ airway pressure → ↓ venous return)
- Lung compliance (e.g., ↑ compliance → ↓ venous return)
- Chest wall stiffness (e.g., in the obese patients, a given change in airway pressure and lung volume will have more impact on the hemodynamics, given that the pressure rise around the heart is going to be higher than in patients with compliant chest wall, everything else being equal)

Note that as airway pressure increases above a certain level (e.g., high PEEP [positive end-expiratory pressure]):

- Oxygen transport start to decline despite the rising PaO₂ as cardiac output starts falling.
- Dead space also tends to increase due to compression of alveolar capillaries by high alveolar pressure, creating ventilated but poorly perfused alveolar units.

Other Potentially Adverse Effects of Mechanical Ventilation

Excessive airway pressure and tidal volume can lead to lung injury (ventilator-induced lung injury) and contribute to increased mortality.

Lungs of dogs ventilated for a few hours with large tidal volume demonstrate extensive hemorrhagic injury.

Other Potentially Adverse Effects of Mechanical Ventilation

In the setting of obstructive physiology (e.g., asthma and COPD), adjustment of the tidal volume and rate minute ventilation to restore a normal pH and PaCO₂ can lead to air trapping, pneumothoraces, and severe hypotension.

Upper Panel: When airway resistances are high, there is for a few breath more air going in than coming out of the lungs (dynamic hyperinflation). Subsequently, a new equilibrium is reached. The amount of air trapped can be estimated in a passive patient by discontinuing ventilation and collecting the expired volume (lower panel).

The volume of trapped gas is largely determined by:

1. The severity of airway obstruction
2. The ventilator settings (see advance course for details). Of all the settings, the imposed minutes ventilation (set rate x VT) and the most important one.
3. The time left between tidal breath for exhalation is less important if a low VT and VE are targeted.

In a *passive* subject, airway pressure represents the entire pressure ($P$) applied across the respiratory system.

The work required to deliver a tidal breath ($W_b$) = tidal volume ($V_T$) x airway pressure

The pressure ($P$) associated with the delivery of a tidal breath is defined by the simplified equation of motion of the respiratory system (lungs & chest wall):

$$P = \frac{V_T}{C_R} + \frac{V_T}{T_i} \times R_R + PEEP_{\text{total}}$$

Where $C_R$ = compliance of the respiratory system, $T_i$ = inspiratory time and $V_T/T_i$ = Flow, $R_R$ = resistance of the respiratory system and $PEEP_{\text{total}}$ = the alveolar pressure at the end of expiration = external PEEP + auto (or intrinsic) PEEP, if any. Auto PEEP = PEEP$_{\text{total}}$ – P$_{\text{extrinsic}}$ (PEEP dialed in the ventilator) adds to the inspiratory pressure one needs to generate a tidal breath.
Work of Breathing

- Work per breath is depicted as a pressure-volume area.
- Work per breath ($W_{\text{breath}}$) = $P \times$ tidal volume ($V_T$)
- $W_{\text{min}} = w_{\text{breath}} \times$ respiratory rate

The total work of breathing can be partitioned between an elastic and resistive work. By analogy, the pressure needed to inflate a balloon through a straw varies; one needs to overcome the resistance of the straw and the elasticity of the balloon.
Intrinsic PEEP and Work of Breathing

When present, intrinsic PEEP contributes to the work of breaking and can be offset by applying external PEEP.

PEEPi = intrinsic or auto PEEP; green triangle = tidal elastic work; red loop = flow resistive work; blue rectangle = work expended in offsetting intrinsic PEEP (an expiratory driver) during inflation
The Pressure and Work of Breathing can be Entirely Provided by the Ventilator (Passive Patient)
When the lung is inflated by constant flow, time and volume are linearly related. Therefore, the monitored airway pressure tracing (Paw) reflects the pressure-volume work area during inspiration. A pressure-sensing esophageal balloon reflects the average pressure change in the pleural space and therefore the work of chest wall expansion.
The Work of Breathing can be Shared Between the Ventilator and the Patient

The ventilator generates positive pressure within the airway and the patient’s inspiratory muscles generate negative pressure in the pleural space.

\[ \text{Paw} = \text{Airway pressure, Pes= esophageal pressure} \]
Relationship Between the Set Pressure Support Level and the Patient’s Breathing Effort

The changes in Pes (esophageal pressure) and in the diaphragmatic activity (EMG) associated with the increase in the level of mask pressure (Pmask = pressure support) indicate transfer of the work of breathing from the patient to the ventilator.

Partitioning of the Workload Between the Ventilator and the Patient

How the work of breathing partitions between the patient and the ventilator depends on:

- Mode of ventilation (e.g., in assist control most of the work is usually done by the ventilator)
- Patient effort and synchrony with the mode of ventilation
- Specific settings of a given mode (e.g., level of pressure in PS and set rate in SIMV)
Common Modes of Ventilation

- Volume targeted ventilation (flow controlled, volume cycled)
  - AC

- Pressure targeted ventilation
  - PCV (pressure controlled, time cycled)
  - PS

- Combination modes
  - SIMV with PS and either volume or pressure-targeted mandatory cycles
Pressure and Volume Targeted Ventilation

In pressure-targeted ventilation: an airway pressure target and inspiratory time are set, while flow and tidal volume become the dependent variables.

In volume targeted ventilation (flow-controlled, volume cycled), a target volume and flow (or inspiratory time in certain ventilator) are preset and pressure and inspiratory time (or flow in the ventilator where inspiratory time is preset) become the dependent variables.

The tidal volume is the integral of the flow during inspiration = area under the curve of the flow time curve during inspiration (see next slide).
FIG. 7–3. Airway pressure (P_{aw}) and flow wave forms during pressure-controlled, time-cycled ventilation and during flow-controlled, volume-cycled ventilation delivered with constant and decelerating flow profiles.
Assist-control

Set variables
- Volume, $T_1$ or flow rate, frequency, flow profile (constant or decel)
- PEEP and $FIO_2$

Mandatory breaths
- Ventilator delivers preset volume and preset flow rate at a set back-up rate

Spontaneous breaths
- Additional cycles can be triggered by the patient but otherwise are identical to the mandatory breath.
Key set variables
- Targeted volume (or pressure target), flow rate (or inspiratory time, Ti), mandated frequency
- PEEP, FIO$_2$, pressure support

Mandatory breaths
- Ventilator delivers a fixed number of cycles with a preset volume at preset flow rate. Alternatively, a preset pressure is applied for a specified Ti

Spontaneous breaths
- Unrestricted number, aided by the selected level of pressure support
Peak Alveolar and Transpulmonary Pressures

\[ P(t) = \frac{V_T}{C_R} + \text{Flow} \times R_R + \text{PEEP tot} \]

Transpulmonary pressure is a key determinant of alveolar distension.

\[ P_{\text{transpulmonary}} = \text{Palveolar} - \text{Ppleural} \]

\[ P_{\text{plat}} = \text{Maximum Palveolar} \]
Plateau pressure tracks the highest tidal alveolar pressure, a key determinant of alveolar distension.

Plateau pressure (Pplat) is, however, only a surrogate of peak alveolar distending pressure (transpulmonary pressure = Pplat – pleural pressure).

- e.g., in a patient with a low chest wall compliance, a given Pplat is typically associated with a higher pleural pressure but less alveolar distension (smaller transpulmonary pressure) than in a patient with a compliant chest wall.

The difference between the Ppeak and Pplat tracks the resistive pressure, as dictated by the equation of motion. During an inspiratory pause, flow becomes zero, the resistive pressure is eliminated and the airway pressure drops from its peak to the plateau pressure.
Airway Resistance and Respiratory System Compliance

Under conditions of constant flow, the difference between peak and plateau airway pressures drives end-inspiratory flow.

When airflow is stopped in a passively ventilated patient by occlusion of the expiratory circuit valve at end inspiration (plateau pressure) and end expiration (total PEEP), the pressure needed to overcome the elastic recoil of the lungs and chest wall during delivery of the tidal volume is given as the difference in these values. Dividing the delivered tidal volume by this difference quantifies the respiratory system compliance.
Mean Airway Pressure

Although measured in the connecting circuit, mean airway pressure is a valid measure of the pressure applied across the lung and chest wall, averaged across both phases of the ventilatory cycle - but **only** under **passive** conditions.

Changes in mean airway pressure are produced by changes in minute ventilation, PEEP, and I:E ratio.

Mean airway pressures affect pleural pressure and lung distention.

Therefore, changes in mean airway pressure during passive inflation may influence:

- Arterial oxygenation
- Cardiac output
Pressure Controlled Ventilation

Key set variables:
- Pressure, $T_1$, and frequency
- PEEP and FIO$_2$

Mandatory breaths
- Ventilator generates a predetermined pressure for a preset time

Spontaneous breaths
- PCV-AC mode: same as mandatory breaths
- PCV-SIMV mode: unsupported or PS

Important caveat
- It is important to understand that in pressure-controlled ventilation the relation between the set rate and minute ventilation is complex. Above a certain frequency (e.g., when intrinsic PEEP is created due to a reduced expiratory time), the driving pressure starts to drop--and so does the delivered tidal volume.
- A pneumothorax or other adverse change in the mechanics of the respiratory system will not trigger a high alarm pressure but a low tidal volume alarm instead.
Pressure Support

Pressure = set variable.

Mandatory breaths: none.

Spontaneous breaths
  • Ventilator provides a preset pressure assist, which terminates when flow drops to a specified fraction (typically 25%) of its maximum.
  • Patient effort determines size of breath and flow rate.
PCV: Key Parameter to Monitor is $V_T$

What Causes a Decreased $V_T$ During PCV?

- **Change in mechanics**
  - $\uparrow$ airway resistance: e.g., bronchospasm
  - $\downarrow$ respiratory system compliance.
    e.g., pulmonary edema, pneumothorax

- **AutoPEEP $\uparrow$**
  - $\uparrow$ expiratory resistance
  - $\downarrow$ expiratory time
    e.g., $\uparrow$ rate

- **Inspiratory time $\downarrow$**
  - e.g., $\uparrow$ rate if I:E ratio constant
Auto-PEEP (Intrinsic PEEP, PEEPi)

Note that Auto-PEEP is not equivalent to air trapping. Active expiratory muscle contraction is an often under appreciated contributor (left panel) to positive pressure at the end of expiration.

Suspecting and Measuring AutoPEEP

AutoPEEP is commonly measured by performing a pause at the end of expiration. In a passive patient, flow interruption is associated with pressure equilibration through the entire system. In such conditions, proximal airway pressure tracks the mean alveolar pressure caused by dynamic hyperinflation.

Suspect AutoPEEP if flow at the end of expiration does not return to the zero baseline.
Interim Summary and Key Points

- Mechanical ventilation helps to improve respiratory gas exchange and can provide complete or partial work of breathing assistance.
- Safe and effective implementation of mechanical ventilation requires understanding the equation of motion for the respiratory system.
- Monitoring dynamic and static airway pressures and flows yields vital information for interpreting the mechanics of the respiratory system and for adjusting machine settings for optimal performance.
Mechanical Ventilation and Gas Exchange

Respiratory acidosis
Hypoxemia
Hypercapnic Acidosis

Determinants of PaCO₂
- PACO₂ = 0.863 x VCO₂/VA
- VA = VE (1-VD/VT)

Causes of hypercapnia
- Inadequate minute ventilation (VE)
- Dead space ventilation ↑ (VD/VT)
- CO₂ production ↑ (VCO₂)

Corrective measures for respiratory acidosis
- When appropriate, increase the minute ventilation (e.g., the rate or the tidal volume)
Mechanism for Arterial Hypoxemia

- Reduced FiO₂ (e.g., toxic fumes, altitude)
- Hypoventilation
- Impaired diffusion
- Ventilation/perfusion (Vₐ/Q) mismatching
  - High Vₐ/Q \( (\infty = \text{Shunt}) \)
  - Low Vₐ/Q \( (0 = \text{Dead-Space}) \)
- Shunting
  - If significant shunting is present, the FIO₂ requirement is typically > 60%
The relationship between PaCO$_2$ and minute ventilation is not linear. In patients with hypoventilation, small changes in minute ventilation may have large effect on the PaCO$_2$. 
Shunting: Effects of FiO\textsubscript{2} on PaO\textsubscript{2}

Note that as the % of shunt rises, increasing the FiO\textsubscript{2} has less and less impact on PaO\textsubscript{2}.

Under such conditions, reducing the shunt fraction is key to the ability to improve gas exchange, and this typically requires PPV and PEEP.
Key Factors Determining the Effects of Shunting on Arterial $O_2$ Saturation

- Note that inpatients with shunt physiology a reduction in arterial $O_2$ saturation may be due to a change in:
  - the intraparenchymal shunt fraction (e.g., atelectasis)
  - or
  - the $SvO_2$ (e.g., drop in cardiac output).

A sudden drop in $O_2$ saturation in patients with ARDS warrants a thorough assessment of the respiratory and cardiovascular system.

$SvO_2$

Shunt fraction
In normal lungs, the inspiratory resistive pressure is similar to the expiratory resistive pressure (light shaded area under the airway pressure-time curve) so that mean airway pressure (Paw) can be used to track mean alveolar pressure (Palv).

Lung regions with shunt tend to distribute preferentially in the dependent regions. Tidal ventilation helps open collapsed regions, and PEEP helps to maintain those regions open throughout expiration and to reduce shunt. Note that level of PEEP required to achieve such varies along the gravitational axis.
Effect of PEEP-induced Alveolar Recruitment on PaO₂

Approach to MV

Is MV indicated? 

YES

NO

Contraindication to NIPPV? 

YES

NO

NIPPV

Success?

YES

Invasive MV

NO

Conservative treatment and periodic reassessment

NO

NO
Noninvasive Ventilation

- Ventilatory support provided without invasive airway control
  - No tracheostomy
  - No ETT
Key Differences Between NIPPV and IPPV

**Advantages of NIPPV**

- Allows the patients to maintain normal functions
  - Speech
  - Eating
- Helps avoid the risks and complications related to:
  - Intubation
  - Sedation
  → Less ventilator-associated pneumonia

**Disadvantages of NIPPV**

- Less airway pressure is tolerated
- Does not protect against aspiration
- No access to airway for suctioning
Clinical Use of NIPPV in Intensive Care

- Decompensated COPD (Hypercapnic Respiratory Failure)
- Cardiogenic pulmonary edema
- Hypoxic respiratory failure

Other possible indications
- Weaning (post-extubation)
- Obesity hypoventilation syndrome
- Patients deemed not to be intubated
- Post-surgery
- Asthma

Adapted from: Am J Respir Crit Care Med. 2001;163:283-291.
Contraindications to NIPPV

- Cardiac or respiratory arrest
- Nonrespiratory organ failure
- Severe encephalopathy (e.g., GCS < 10)
- Severe upper gastrointestinal bleeding
- Hemodynamic instability or unstable cardiac arrhythmia
- Facial surgery, trauma, or deformity
- Upper airway obstruction
- Inability to cooperate/protect the airway
- Inability to clear respiratory secretions
- High risk for aspiration

Adapted from: Am J Respir Crit Care Med. 2001;163:283-291.
**Initiating NIPPV**

**Initial settings:**
- Spontaneous trigger mode with backup rate
- Start with low pressures
  - IPAP 8 - 12 cmH₂O
  - PEEP 3 - 5 cmH₂O
- Adjust inspired O₂ to keep O₂ sat > 90%
- Increase IPAP gradually up to 20 cm H₂O (as tolerated) to:
  - alleviate dyspnea
  - decrease respiratory rate
  - increase tidal volume
  - establish patient-ventilator synchrony
Success and Failure Criteria for NPPV

- Improvements in pH and PCO₂ occurring within 2 hours predict the eventual success of NPPV.

- If stabilization or improvement has not been achieved during this time period, the patient should be considered an NPPV failure and intubation must be strongly considered.

- Other criteria for a failed NPPV trial include: worsened encephalopathy or agitation, inability to clear secretions, inability to tolerate any available mask, hemodynamic instability, worsened oxygenation.
Conclusions

- A good understanding of respiratory physiology is required for the judicious mechanical ventilation.
- Unless contraindicated, NIPPV is becoming the first modality to try in many settings.
- Monitoring key variables such as $P_{\text{plateau}}$ and auto-PEEP is mandatory to safe and effective practice.
29-year-old patient (weight 120 kg, height 170 cm)

ARDS secondary to bilateral pneumonia

Ventilator settings: AC with $V_T$ 800 ml and back-up rate 10/min, PEEP 5 cmH$_2$O, FIO$_2$ 80 %

Measured variables: rate 25, $V_E$ = 20 l/min, Ppeak 40 cm H$_2$O, Pplat 35 cm H$_2$O

ABG: pH 7.40, PaO$_2$ 55 mmHg, PaCO$_2$ 38 mmHg, O$_2$ saturation 85%
Question 1

What mechanism best explains the patient’s hypoxemia?

1. V/Q mismatch
2. Shunt
3. Abnormal diffusion
4. Inadequate oxygen delivery and high tissue extraction
If in a ventilated patient, FIO₂ > 60% is needed, shunt is certainly the main cause for the hypoxemia (correct response: 2).

As a rule, increasing the FIO₂ will compensate for Vₐ/Q mismatching but not for shunt. When Vₐ/Q mismatching is present, hypoxemia typically corrects with an FIO₂ < 60%.

Altered diffusion is rarely a clinically relevant issue.

Increasing the ventilation rate will not exert a significant impact on oxygenation unless it contributes to air trapping and auto-PEEP.
Which of the following ventilatory setting changes is the next best step to reduce shunt and increase the PaO$_2$/FIO$_2$ ratio (a bedside index of oxygen exchange)?

1. Increase PEEP to 10 cmH$_2$O
2. Increase the FIO$_2$ to 100%
3. Add an inspiratory pause of 1 second
4. Increase respiratory rate to 30/min
Interventions that target mean airway pressure are the most helpful. They help recruit flooded or collapsed alveoli and maintain the recruited alveoli open for gas exchange (reduced shunt).

Increasing PEEP is the first intervention to consider; extending the inspiratory time and I:E ratio is a secondary option to raise mean airway pressure.

In the presence of shunt, increasing the FIO$_2$ would reduce the ratio.

Although increasing rate may affect mean Paw, its impact is overall minor. Rate adjustment is mainly used to control minute ventilation and its consequences on:

- air trapping
- PaCO$_2$ and pH
Question 3

- PEEP is increased to 10 cmH₂O
- PaO₂ is now 85 mmHg, Pplat is 45, and Ppeak 50 cmH₂O
- The high pressure alarm is now triggered.

Your next step is:
1. Reset the alarm pressure to 55 cmH₂O.
2. Disconnect the patient from the ventilator and start manual ventilation (bagging).
3. Order a stat chest x-ray to assess for a pneumothorax.
4. Reduce the tidal volume slowly until the alarm turns off.
5. None of the above
The correct answer is 5. Option 1 does not address the issue of the excessive tidal volume and airway pressure.

The rise in airway pressure that triggered the alarm was predictable following the increase in PEEP level. There is thus no need for 2 and 3.

Tidal volume is never titrated to an arbitrary set alarm pressure.

Pplat, which tracks alveolar pressure and the risk of developing ventilator-induced lung injury (VILI), is an easy and accessible bedside parameter used to assess the risk of alveolar overdistension. In this patient, it is the high Pplat associated with the choice of an excessive tidal volume that puts the patient at risk of VILI.

Patients with ARDS have reduced aerated lung volume (“baby lungs”) and need to ventilated with small tidal volumes: e.g., 6 ml/kg predicted ideal body weight. This patient is clearly ventilated with an excessive tidal volume for his size (ideal or predicted body weight). The tidal volume must be reduced. A tidal volume and PEEP combination associated with a Pplat of less than 25 cmH₂O is generally considered safe. Concerns regarding the risk of overdistension and VILI is significant when Pplat is > 30 cmH₂O.

Remember, however, that the actual distending alveolar pressure is the transpulmonary pressure (Pplat- Ppleural). Higher Pplat can be accepted in a patient with low chest wall compliance, as less alveolar distension will be present for the same Pplat, everything else being equal.
Case 2

67-year-old female (weight 50 kg) with severe emphysema is admitted for COPD decompensation. She failed NIPPV and required sedation, paralysis, and intubation.

Soon after intubation and initiation of mechanical ventilation, she became hypotensive (BP dropped from 170/95 to 80/60). She has cold extremities, distended neck veins, midline trachea, distant heart sounds, and symmetrical breath sounds with prolonged expiratory phase.

Ventilatory settings are: assist control, tidal volume 500ml, rate 15/min, PEEP 5 cmH$_2$O, FIO$_2$ 1.0 (100%)

ABG: pH 7.20, PaO$_2$ 250 mmHg, PaCO$_2$ 77 mmHg

Measured variables: rate 15/min, VE = 7.5 l/min, Ppeak 45 cmH$_2$O, Pplat 30 cmH$_2$O
The next step in this patient’s management should be:

1. Order a stat echocardiogram to assess for tamponade.
2. Order a stat AngioCT to assess for pulmonary embolism.
3. Measure AutoPEEP, disconnect the patient briefly from the ventilator, then resume ventilation with a lower tidal volume and rate and administer intravenous fluid.
4. Start the patient on intravenous dopamine and adjust the ventilator to normalize the PaCO₂.
The correct answer is 3. Remember that gas trapping is your key concern in the ventilated patient with obstructive physiology.

A quick look at the expiratory flow tracing and performing an expiratory pause maneuver demonstrated that the patient has developed severe dynamic hyperinflation and intrinsic PEEP (15 cmH₂O).

Brief disconnection (1 - 2 minutes) from the ventilator while continuously monitoring oxygen saturation is safe in this condition and allows for the lung to empty, intrinsic PEEP to decrease--thus restoring venous return, preload, stroke volume, and BP. The restoration of BP following ventilator disconnection is not specific for air trapping. Therefore, intrinsic PEEP needs to be measured to confirm the diagnosis.

It is also important to consider the possibility of a tension pneumothorax in this patient. The symmetrical chest and midline trachea did not suggest this possibility here.

Also notice that Pplat was elevated (due to gas trapping), but in contrast to a patient with stiff lungs (ARDS), there is a large difference between Ppeak and Pplat because airway resistance is markedly elevated in patients with COPD.
You change the ventilator’s tidal volume to 300 ml and the rate to 15/min. After 1 liter of physiologic saline is infused, the BP is now 100/70 mmHg and heart rate is 120/min. ABG: pH 7.30, PaO₂ 250 mmHg, PaCO₂ 60 mmHg. Measured variables: rate 20/min, VE = 6.0 l/min, Ppeak 37 cmH₂O, Pplat 25 cmH₂O, Intrinsic PEEP is now 7 cmH₂O (total PEEP=12 cmH₂O).

The best next step is to:
1. Continue with bronchodilators and tolerate the current mild respiratory acidosis.
2. Increase the rate to normalize PaCO₂.
3. Increase the tidal volume but only to normalize the pH.
4. Ask for another ABG since you do not believe the drop in PaCO₂--minute ventilation declined.
The best next step is continue with bronchodilator and tolerate the current mild respiratory acidosis (RA).

- The patient has no contraindications to mild RA (history of an acute or chronic central nervous system problem, that may be worsened by the increase in intracranial pressure associated with RA, heart failure, cardiac ischemia, or arrhythmia.
- Although less than present initially, dynamic hyperinflation is still an issue (high Pplat and relatively low BP). Thus, increasing the minute ventilation to normalize the pH or PaCO₂ will make this worse.
- The reduction in PaCO₂ is due to less air trapping, with improved venous return and reduced dead space ventilation. Hyperinflation tends to compress capillaries and thus promote ventilation of unperfused alveolar units (dead space).
References and Suggested Readings