Mechanical Ventilation Case Questions And Answers

<u>Case 1</u>
A 55 year-old man with a history of COPD presents to the emergency room with a two day history of worsening shortness of breath which came on following a recent viral infection. In the emergency room, his oxygen saturation is 88% on room air. He is working hard to breathe and is only speaking in short sentences. On exam, he has diffuse wheezes and a prolonged expiratory phase. His chest x-ray reveals changes consistent with COPD but no new focal infiltrates. An arterial blood gas (ABG) is done and shows pH 7.17, PCO_2 55, PO_2 62, HCO_3^- 25.

What are the indications for starting a patient on mechanical ventilation?
There are several primary indications for initiating mechanical ventilation including: hypercarbic respiratory failure, hypoxemic respiratory failure, to prevent or reverse atelectasis, to prevent or reverse ventilatory muscle fatigue, to permit sedation and/or neuromuscular blockade (eg. surgery), to stabilize the chest wall (eg. massive flail chest) or to ensure airway protection (eg. a patient with altered mental status and a large upper gastrointestinal bleed).

One should be aware that with hypercarbic and hypoxemic respiratory failure, there are no specific thresholds that are used to determine when to initiate mechanical ventilation. For example, you do not automatically intubate a patient when their PCO_2 rises above 60 mm Hg or their room air PO_2 falls below 55 mm Hg. Instead, you must take into account the overall clinical situation and assess whether the degree of hypercarbia or hypoxemia is putting the patient's life at risk. If they can be supported by other means, such as oxygen by face mask, you might hold off on initiating mechanical ventilation, whereas if their PO_2 continues to fall despite high levels of supplemental oxygen, mechanical ventilation would be necessary. Similarly, if a patient is hemodynamically stable, you might try oxygen by facemask for hypoxemic respiratory failure but if the patient is showing signs of sepsis with hemodynamic instability and multiorgan dysfunction, you would move more quickly to intubate the patient and stabilize their respiratory status in order that you can focus on other important issues.

What do you think about the possibility of using non-invasive positive pressure ventilation (bi-level positive airway pressure) in this patient?
There are two forms of mechanical ventilation – <u>*invasive*</u> mechanical ventilation, in which an endotracheal tube is inserted in the patient's airway, and <u>*non-invasive*</u> ventilation, in which the patient receives respiratory support through a tight fitting mask applied to their face. There are some situations in which invasive modes of mechanical ventilation are necessary and other situations in which patients can be supported by non-invasive means.

This particular patient falls into the latter category. Even though he is clearly struggling to breathe and has a rising PCO_2 and a declining pH, it is legitimate to give him a trial of non-invasive ventilation. There is now a large amount of data showing that patients who present with COPD exacerbations and hypercarbia can be successfully managed with non-invasive ventilation; this strategy is associated with a decreased need for intubation and initiation of mechanical ventilation, improved survival and shorter ICU stays when compared to managing these patients without non-invasive ventilation (eg. oxygen by face mask alone). Given the data in this regard, this patient should be given a trial of non-invasive ventilation with close follow-up of his respiratory status. If he improves, he can remain on non-invasive ventilation but if his oxygenation or hypercarbia worsens despite optimal non-invasive ventilation, or if he becomes unresponsive or uncooperative, he will require intubation and invasive mechanical ventilation.

What is the difference between bi-level positive airway pressure (BiPAP) and continuous positive airway pressure (CPAP)? What are the indications for using these different modes of non-invasive mechanical ventilation?
In CPAP therapy, a constant level of pressure is applied to the airways throughout the respiratory cycle (inhalation and exhalation). This pressure serves to stent open the large airways and prevent alveoli from collapsing, thereby avoiding atelectasis and improving oxygenation. There is no additional pressure delivered during inhalation and, therefore, no *ventilatory* support. In bi-level positive airway pressure, also referred to as non-invasive positive pressure ventilation, the expiratory pressure applied to the airways during exhalation is the same as the pressure applied in CPAP therapy. During inhalation the device imparts additional pressure (pressure support or inspiratory assist) to the airways that serves to assist the ventilatory muscles in their efforts to generate inspiratory flow to the alveoli.

These differences in the way CPAP and bi-level positive airway pressure work have a large impact on the clinical situations in which they can be employed. Because of its effect on transmural pressure and its ability to prevent upper airway collapse, CPAP is indicated for management of obstructive sleep apnea. There are also data to support its use in patients with cardiogenic pulmonary edema; the applied pressure improves oxygenation by stenting open the alveoli. CPAP also improves hemodynamics in cardiogenic pulmonary edema by decreasing afterload and pre-load, thereby improving left ventricular function. Because of the added pressure during inhalation, bi-level positive airway pressure provides a means to support ventilation and the work of breathing. There is clear data to support its use in COPD exacerbations and to support patients with other forms of ventilatory failure such as amyotrophic lateral sclerosis and muscular dystrophy. There is less clear data supporting its use in asthma exacerbations, although many clinicians will often give asthma patients a trial of this therapy during an asthma exacerbation. There are also some data to

suggest it has a role in treating oncologic patients with pneumonia. CPAP does not provide any ventilatory assistance and, therefore, should not be used in these situations.

<u>Case 2</u>
A 45 year-old. 6-foot tall man presented to the emergency room with a two day history of fever and cough productive of brown sputum. He was hemodynamically stable at the time with a blood pressure of 130/87. His chest x-ray showed a right middle lobe infiltrate and his room air ABG showed: pH 7.32, PCO_2 32, PO_2 78, HCO_3^- 18. He was started on antibiotics and admitted to the floor. Four hours later, the nurse calls because she is concerned that he is doing worse. On your arrival in the room, his blood pressure is 85/60, his pulse is 120 beats/minute and his oxygen saturation, which had been 97% on 2L oxygen by nasal cannula, is now 78% on a non-rebreather mask. The patient is obviously laboring to breathe with use of accessory muscles and is less responsive than he was on admission. He is diaphoretic and cannot talk in full sentences. On lung exam, he has crackles throughout the bilateral lung fields. You obtain a chest x-ray which shows increasing bilateral, diffuse lung opacities. An ABG is done while he is on the non-rebreather mask and shows: pH 7.17, PCO_2 45, PO_2 58, HCO_3^- 14.

What should you do now? Is there a role for CPAP or bi-level positive airway pressure in managing his hypoxemia?
Although there are certain clinical situations in which CPAP or bi-level positive airway pressure are indicated as means of respiratory support, hypoxemic respiratory failure for reasons other than cardiogenic pulmonary edema is ***not*** one of them. The data indicates that use of non-invasive ventilation in such situations is associated with worse patient outcomes. This patient is struggling to breathe; his mental status is declining and he is becoming hemodynamically unstable. Finally, he has worsening oxygenation and ventilation as evidenced by a falling P_aO_2, a rising P_aCO_2 and a worsening pH. The best option for him is intubation and mechanical ventilation.

A decision is made to intubate the patient and initiate mechanical ventilation for worsening respiratory failure. The intubation proceeds without difficulty. The tube position is confirmed and the anesthesiologist leaves the room. The respiratory therapist has secured the endotracheal tube. She turns to you and asks what settings you would like to use for the ventilator?

What information do you need to provide to the respiratory therapist?
In most cases, you need to provide the respiratory therapist five pieces of information: the mode of mechanical ventilation, the tidal volume, the respiratory rate, the inspired oxygen concentration (F_IO_2) and the level of positive end-expiratory pressure (PEEP). In rarer cases you will stipulate a peak inflation

pressure, rather than a tidal volume. This is described further in the next question.

The respiratory therapist suggests you use the volume-targeted Assist Control (AC) mode of mechanical ventilation. How does this work? How does it differ from Synchronized Intermittent Mandatory Ventilation (SIMV) or Pressure Control (PC)? Which mode is better for your patient?
In Assist Control (AC) ventilation, the clinician determines the tidal volume and the respiratory rate for the patient. The machine guarantees that the patient will receive the set number of breaths at the desired tidal volume each minute. Patients can also initiate their own breaths (i.e. take breaths above the rate set on the ventilator by the clinician); patient-initiated breaths are delivered at the full tidal volume that has been set on the machine.

In SIMV, the clinician also sets the rate and tidal volume and the machine guarantees the patient will receive the set number of breaths at the desired tidal volume each minute. Patients can also initiate their own breaths, but on the extra breaths in SIMV, the patient only gets as much tidal volume as they are capable of taking in on their own; the ventilator does not guarantee a set tidal volume for these breaths. Weak patients may draw small tidal volumes on these extra breaths while strong patients may take in larger tidal volumes.

Pressure control ventilation is quite different than either volume targeted AC or SIMV. In this mode, the clinician sets the peak inflation pressure and respiratory rate but does not specify the tidal volume. Instead, the tidal volume received by the patient varies based on the compliance of the respiratory system and the level of airway resistance. If a patient, for example, has a very compliant respiratory system (emphysema), they will receive a large tidal volume for a set pressure whereas a patient with low respiratory system compliance (ARDS) will only receive a small tidal volume.

At present, there is no evidence that a particular mode of mechanical ventilation is associated with a mortality benefit compared to the other modes. Evidence exists for improvements in short-term physiologic variables with one mode compared to another (which is largely a function of how the comparison is set up), but there are no solid data to support a preference for one mode over another. This is a subject of intense debate and the choice of mode tends to be very physician-, respiratory therapist-, and institution-dependent. In the University of Washington system, the pulmonary and critical care physicians tend to ventilate most of our patients using volume-targeted Assist Control mode and to use pressure control ventilation only in situations in which the patient has a very poor response to standard measures.

Suppose you put the patient on a volume-targeted Assist Control mode of mechanical ventilation. How do you choose the tidal volume?

The tidal volume is chosen based on the patient's weight. It is critical, however, to make sure you use the correct weight in the calculations. The size of the lungs is largely a function of a person's height. Therefore, rather than using the patient's actual weight to determine the tidal volume, you should, instead, use the patient's _**ideal**_ body weight, the value of which is derived from the patient's height. To calculate the ideal body weight in kilograms, you can use the following formulas:

    Men: [(height in inches – 60) X 2.2] + 50
    Women: [(height in inches – 60) X 2.2] + 45

Failure to use the correct weight can lead to disastrous consequences for the patient. If you were to put a 150 kg person on a tidal volume of 10 ml/kg of their _actual_ body weight, you would end up delivering tidal volumes of 1.5L and, as a result, would significantly increase the risk of barotrauma and ventilator-induced lung injury.

Once you know the ideal body weight, you can choose the tidal volume for the patient. The majority of patients are placed on a tidal volume corresponding to 8-10 ml/kg of their ideal body weight. In order to decrease the risk of air-trapping and barotrauma, patients intubated for COPD or asthma exacerbations are often placed on 6-8 ml/kg of their ideal body weight. Patients who develop ARDS are placed on 4-6 ml/kg of their ideal body weight but are not typically started at these low levels. Instead, they are started on 8-10 ml/kg and the tidal volume is gradually decreased over a period of time. Finally, many ventilator-dependent patients with spinal cord injuries are maintained on higher tidal volumes ranging from 12 ml/kg to as high as 20 ml/kg of their ideal body weight. Proponents of this practice argue that it improves patient comfort and prevents mucous plugging and atelectasis which predispose to pulmonary complications in this patient population. The data supporting this practice is actually quite limited and there is considerable debate as to the safety and efficacy of such high tidal volumes in these patients.

The patient in this case is 6-foot tall (72 inches). Using the formula above, his ideal body weight is about 76kg. Therefore, you would place him on a tidal volume between 600 and 750 ml, which corresponds to between 8 and 10 ml/kg.

What respiratory rate should you choose for the patient?
Contrary to the popular, but erroneous, practice of choosing the ubiquitous respiratory rate of "12" because "that is what I've always seen other people do," the respiratory rate should be selected based on an assessment of the patient's minute ventilation requirements. For example, a patient with a normal bicarbonate of 24 who was intubated for a procedure may only need 6-8 liters/minute of ventilation whereas a patient in severe sepsis with a bicarbonate of 10 may require upwards of 20-25 liters/minute of ventilation to maintain an

adequate acid-base status. Once you derive an estimate of the minute ventilation (V_E) needs of the patient, you can use your previously determined tidal volume and simple division to calculate an appropriate respiratory rate (RR = V_E/tidal volume)

This practice is particularly important in the period immediately following intubation. Most patients receive paralytic agents for intubation and, as a result, have no ability to mount respiratory efforts for 15 to 60 minutes following the procedure. They are dependent on you to choose the correct rate and give them an adequate amount of minute ventilation. Failure to do so will lead to increasing respiratory acidosis and worsening pH. As the paralytic agent wears off and sedative needs decrease, the patients will often set the respiratory rate on their own in an effort to match their minute ventilation needs.

What should the F_IO_2 and PEEP be set at?
In the majority of cases patients are initially placed on an F_IO_2 of 1.0 and a PEEP of 5 cm H_2O.If the initial ABG drawn 30 to 60 minutes after intubation reveals an adequate P_aO_2 (above 65 mm Hg) the F_IO_2 can be turned down to a lower level. Further adjustments are made in the F_IO_2 based on the patient's oxygen saturation and blood gases are not necessary for every change in this parameter. It is not uncommon to hear comments about maintaining patients on too high an inspired oxygen concentration for too long a period of time, as there is concern about provoking oxygen toxicity in the lungs. This concern is largely based on the results of laboratory studies in animal and normal human volunteers and concrete evidence of its occurrence in critically ill ICU patients is lacking. If a patient requires a high F_IO_2 in order to maintain adequate oxygenation then they should receive those high levels for as long as necessary.

The PEEP is typically not decreased below 5 cm H_2O but can be increased as necessary (up to 15 to 20 cm H_2O) to support oxygenation in patients with severe hypoxemia (discussed further below). It tends to be a more effective tool in diffuse, rather than focal, lung processes such as ARDS.

Case 3
A patient is admitted to the ICU with severe necrotizing pancreatitis. A few hours after admission, he developed increasing oxygen requirements and was intubated for hypoxemic respiratory failure. Initially, his oxygen saturation improved to the mid-90% range on an F_IO_2 of 0.5, but in the past 2 hours, the nurse has had to increase the F_IO_2 back to 0.7 and his SaO_2 is still in the lower 90% range. The patient remains on a PEEP of 5 cm H_2O. The nurse drew an ABG which shows pH 7.35, pCO_2 38, PO_2 60, HCO_3^- 22 on an F_IO_2 of 0.8. The patient's repeat chest x-ray is shown below. An echocardiogram performed earlier in the day revealed normal left ventricular function.

How do you explain his worsening oxygenation status?

The patient's chest x-ray shows diffuse bilateral opacities in a pattern consistent with pulmonary edema. The ratio of the P_aO_2 to F_IO_2, referred to as the P/F ratio, is 75. He also has an echocardiogram showing evidence of normal left ventricular function. Based on the combination of the x-ray findings, the low P/F ratio and the evidence of normal left ventricular function, this patient should be classified as having developed the Acute Respiratory Distress Syndrome (ARDS). Pancreatitis is one of several known precipitating factors for this syndrome. Other known precipitants include pneumonia or other forms of severe infection, trauma, severe burns, aspiration of gastric contents, drug overdose and a variety of other processes.

What can you do to improve his oxygenation?

There are two initial options for improving oxygenation for people on mechanical ventilation: raising the F_IO_2 and increasing the PEEP (positive end-expiratory pressure). PEEP helps keep the alveoli open and prevents small airway closure. It is very effective in diffuse processes such as ARDS, pulmonary edema or diffuse alveolar hemorrhage. It is generally not effective and may actually worsen oxygenation in focal processes such as lobar pneumonia. In these cases, the PEEP gets preferentially applied to the more compliant normal lung and does not open alveoli in the diseased portions. The normal alveoli may then become over-distended leading to compression of the capillaries running between the alveoli. As a result, blood flow to these normal regions is decreased and blood is shunted

to the abnormal areas of the lung where poor ventilation/perfusion (V/Q) matching leads to worsening hypoxemia. In focal processes, increasing the F_IO_2 may be the best option for improving oxygenation.

It is important to remember that although increasing the PEEP may improve oxygenation in certain cases, it is not an entirely benign intervention. High levels of PEEP ($\geq$ 15 H_2O) can impair venous return and, therefore, decrease cardiac output and blood pressure. Ironically, even though the P_aO_2 may be improved on the higher level of PEEP, a drop in cardiac output will actually impair oxygen delivery to the tissues. High levels of PEEP also increase the risk of barotrauma. Given the risk of such problems, the clinician must always assess whether the increased level of PEEP is providing benefit to the patient. If the P_aO_2 fails to improve on higher levels of PEEP, then the PEEP should be returned to lower levels in order to avoid the complications described above.

It is also important to remember that while we tend to focus our attention on the P_aO_2 and the S_aO_2, our main concern is whether we are actually able to deliver enough oxygen to the tissues. Although the P_aO_2 and the S_aO_2 contribute to oxygen delivery (DO_2), other factors, including cardiac output and the hemoglobin concentration play a larger role, as you can see in the following equation:

$$DO_2 = \text{Cardiac Output} \times [(Hb \times 1.34 \times S_aO_2) + 0.003 \times P_aO_2]$$

In the severely hypoxemic patient, improving the P_aO_2 often has only a small effect on overall oxygen delivery; you can have a larger impact by transfusing blood if they are markedly anemic or by improving cardiac output by, for example, administering IV fluids. Considering these other interventions would be an important part of management for any severely hypoxemic patient.

What other changes should you consider making in the ventilator settings?
Because this patient has developed ARDS, he should be put on what is referred to as a "lung-protective" ventilation strategy, in which the tidal volume is incrementally decreased to 6 ml/kg of his ideal body weight. The goal is to bring the static or plateau pressure below 30 cm H_2O (the concept of static pressure is described further below in another case). If this goal is not reached when the patient's tidal volume is at 6 ml/kg then the tidal volume can be reduced to as low as 4 ml/kg in an effort to reach the target plateau pressure. This strategy is based on the results of the ARDSnet study which demonstrated that patients with ARDS who were ventilated according to this protocol had improved survival when compared to people with ARDS who were ventilated on tidal volumes of 10-12 ml/kg. The mortality benefit is present even if the plateau pressure was below 30 cm H_2O before the tidal volume was decreased to 6 ml/kg.

If his oxygen saturation fails to improve despite being on high levels of support (eg. F_IO_2 of 1.0 and 20 cm H_2O of PEEP), what other options do you have for improving his oxygenation?

Several strategies can be used in patients with what is referred to as "refractory hypoxemia". They all share a common feature: they can improve oxygenation but have not been shown to improve patient outcomes and, in particular, mortality. In some cases, patients are placed in the prone position using a specially designed bed that facilitates this change in patient position. The theory behind this intervention is that many patients develop atelectasis in dependent lung zones. If blood flow continues to these areas, shunt physiology develops and contributes to hypoxemia. By rotating the person to the prone position, this dependent atelectasis is relieved and there are improvements in ventilation-perfusion matching and, as a result, gas exchange.

Another strategy involves using inhaled pulmonary vasodilator medications (nitric oxide or prostacyclin). If given intravenously, these medications would cause diffuse pulmonary vasodilation which, in turn, would increase blood flow to poorly ventilated areas and worsen oxygenation. By using an inhaled form, however, the medication is delivered to and vasodilation occurs only in those areas of the lung that receive adequate ventilation. This helps improve matching of pulmonary blood flow and ventilation and improves gas exchange. Some clinicians employ what are referred to as recruitment maneuvers in which the lung is held in an inflated position for an extended period of time in an effort to reduce atelectasis and open up previously close regions of the lung.

In other cases, patients are given a bolus and/or drip of a paralytic medication such as vecuronium. The goal of the paralytic agent is to eliminate any muscular activity on the part of the patient and, thereby, decrease oxygen consumption. It also eliminates any patient respiratory effort which might be contributing to dysynchrony with the ventilator and either ineffective ventilation or increased oxygen consumption. Finally, some practitioners place patients on alternative modes of mechanical ventilation such as high frequency oscillatory ventilation.

These strategies are associated with risks and, in the case of prone ventilation and inhaled therapies, are associated with very high cost. In the absence of data supporting a mortality benefit, they should only be used in rare circumstances.

Case 4
A 63 year-old woman was intubated four days ago for respiratory failure secondary to sepsis from a presumed pneumonia. She is on appropriate antibiotics, is now off pressors, and her WBC count has declined to the normal range. During your pre-rounding, you note that her F_IO_2 is down to 0.4 and she is on a PEEP of 5 cm H_2O. On these settings, the ABG shows pH 7.36, pCO_2 46, PO_2 75, HCO_3^- 26. She has a weak cough and continues with copious secretions, requiring suctioning every 30 to 60 minutes.

At what point do you start considering whether your patient is ready to come off the ventilator?
There are several conditions that should be satisfied before you can consider separating your patient from the ventilator. First, the underlying process that put them on the ventilator should be better or improving. Second, the patient should be able to maintain adequate oxygenation on minimal support (eg. P_aO_2 > 80 mm Hg on an F_IO_2 of 0.5 and PEEP < 8.0 cm H_2O). Finally, the patient should be able to maintain an adequate acid-base balance without requiring high levels of minute ventilation (>12 liters/minute) in order to do so. If higher levels of minute ventilation are required, the ventilatory demands on the patient may be high and they are at risk for tiring out once off they are taken off the ventilator.

How do you determine if the patient is capable of being separated from the ventilator?
In the past, clinicians used to look at several different variables, referred to as "weaning parameters" in an effort to assess readiness for separation from the ventilator. For example, if a patient's vital capacity was > 10 ml/kg, then the patient was likely to tolerate being off the ventilator. None of the different parameters that were used had perfect predictive ability and this approach has since been supplanted by a different strategy utilizing a trial of spontaneous breathing. If patients meet certain criteria on the respiratory therapist's morning rounds, they are placed on CPAP, t-piece or a low level of pressure support and are monitored for a period of 30-120 minutes while they breathe on their own. An arterial blood gas is usually drawn at the end of this period. A successful trial is one in which the patient looks comfortable, maintains a good respiratory rate (< 25 breaths/minute), takes in sufficient tidal volumes (> 5 ml/kg) and maintains stable vital signs including heart rate, blood pressure and S_aO_2. The arterial blood gas should also show relatively stable oxygenation and no evidence of increasing P_aCO_2. Patients can fail the trial for a variety of different reasons including vital sign instability, worsening oxygenation, hypercarbia or other signs of insufficient ventilatory capacity. Patients who pass their trial of spontaneous breathing are deemed ready to be separated from the ventilator.

Suppose your patient demonstrates that she can be separated from the ventilator. Should she be extubated?

When a patient passes a spontaneous breathing trial, they are ready to be separated from the ventilator. In other words, they no longer need the ventilatory or oxygen support of the machine at their bedside. It is important to remember, however, that the decision to separate a patient from the ventilator is distinct from the decision to remove the endotracheal tube. Some patients can be separated from the ventilator but still require an endotracheal tube. In order to qualify for extubation, patients should be free of upper airway problems, should be able to protect against aspiration of gastric or oral contents and should be able to cough and clear secretions without a need for frequent suctioning. In this case, the patient has a weak cough and copious secretions, problems that would lead you to predict that she might decompensate if extubated. As a result, the endotracheal tube should remain in place until these issues resolve.

Case 5
A 65 year-old man was admitted to the ICU with pneumonia and was intubated when he developed progressive hypoxemia. He has been on the ventilator for 5 days and has generally been tolerating this therapy well. The nurse calls you because he has all of a sudden become severely agitated and appears to be fighting the ventilator. She asks if she can increase the infusion rates on his midazolam and fentanyl drips for sedation.

What should you do next?
This patient has suddenly become agitated while breathing on the ventilator. In some cases, the agitation is due to inadequate sedation. In many other cases, however, there is a new potentially serious problem that has developed. The problem, however, may not be obvious because the patient is intubated and, therefore, cannot talk to you and tell you what is wrong. It is important to consider and exclude such problems, particularly when there is an acute change in the patient's status. Some important items to consider on the differential diagnosis include a pneumothorax, mucous plug in the endotracheal tube, myocardial infarction, pulmonary embolism, unrecognized disconnection from the ventilator or other ventilator problem, auto-PEEP (hyperinflation and increased intrathoracic pressure) causing difficulty triggering the ventilator, worsening oxygenation and delirium. The first step in the evaluation is to disconnect the patient from the ventilator and manually ventilate ("bag") them. If they improve, the problem is proximal to the endotracheal tube (i.e. the machine or the ventilator tubing). If they are still having problems the airways should be suctioned and a thorough assessment (some history, exam, ABG, CXR and possibly an EKG and laboratory studies) should be performed. Further testing such as a CT pulmonary angiogram might be warranted based on what you find on your initial evaluation. You should always consider the differential diagnosis noted above and exclude life threatening issues before assuming the problem is inadequate sedation.

Case 6

A 65 year-old woman is intubated emergently for a severe COPD exacerbation. She underwent a rapid sequence intubation using succinylcholine for paralysis and etomidate for sedation. Shortly after intubation, she becomes hypotensive with her blood pressure dropping from 145/85 prior to intubation to 95/60 post-intubation. On exam, she has a very prolonged expiratory phase and diffuse wheezing.

What is the differential diagnosis for this patient's hypotension?
This patient decompensated immediately following intubation, a situation with a short differential diagnosis. Potential diagnoses include: esophageal intubation (the endotracheal tube can become dislodged after an initial correct placement and slide into the esophagus); mainstem bronchus intubation; problems with the ventilator circuit, tension pneumothorax, blood pressure effects of sedative medications; severe auto-PEEP (air-trapping and hyperinflation in patients with airflow obstruction leading to increased intrathoracic pressure, decreased venous return and impaired cardiac output); decreased venous return due to positive pressure ventilation and/or volume depleted state in a patient with a weak, pre-load dependent right heart (eg. severe pulmonary hypertension).

What can you do to sort through this differential and identify the etiology of the problem?
A thorough physical exam may reveal the source of the problem. Unilateral absence of breath sounds and elevated neck veins are suggestive of tension pneumothorax while unilateral absence of breath sounds, normal neck veins and tracheal deviation toward the quiet side of the chest suggest mainstem intubation. Worsening oxygen saturation and decreased/absent breath sounds bilaterally suggests esophageal intubation. A chest x-ray may also reveal the presence of a pneumothorax or mainstem intubation while a review of the medication record may point to a role for the sedative medications. To look for the presence of auto-PEEP, if the person is not making spontaneous respiratory efforts you can perform an expiratory pause of the ventilator and examine the difference between the total PEEP and the set-PEEP. On ventilators with graphical displays, you can also examine the flow versus time curves. In normal patients, expiratory flow returns to zero prior to the next breath. The patient is able to exhale fully and there is no longer expiratory airflow by the time the next breath is due. In patients with auto-PEEP, expiratory flow does not return to the zero line before the next breath is delivered. As a result of the airflow obstruction, expiratory airflow is slowed and continues up until the next breath is delivered. Observing this expiratory airflow pattern on the graphic display confirms the presence of auto-PEEP but does not tell you the magnitude of the problem.

How should you manage the most likely source of the problem?
Given that the patient was intubated for a severe COPD exacerbation and likely has a lot of airflow obstruction, the most likely explanation for her hypotension is auto-PEEP. This is a problem commonly seen in patients with severe asthma or COPD in which they trap air on exhalation and become progressively hyperinflated. This hyperinflation leads to increased intrathoracic pressure, which decreases venous return, impairs cardiac output and, as a result, leads to decreased blood pressure. Patients can even go into cardiac arrest (usually a pulseless electrical activity arrest) from this phenomenon. Several strategies are used to prevent or manage this problem. First, the minute ventilation can be decreased by lowering the respiratory rate or tidal volume. If you put less air into the lungs each minute, the patient has to exhale less air and, therefore, there is less potential for air-trapping. Second, you can provide more time for the patient to exhale. This is done by increasing the flow rate on inhalation and thereby decreasing the ratio of inspiratory time to expiratory time (I:E ratio). Finally, you can use bronchodilators and steroids to facilitate bronchodilation, decrease airway inflammation and promote exhalation. If a patient becomes bradycardic or pulseless, you should disconnect them from the ventilator and let their chest deflate as the trapped air escapes.

A key aspect of managing auto-PEEP is anticipating situations in which it might occur. Patients intubated during severe asthma and COPD exacerbations are prime candidates for this problem and you should always be on the alert for the problem in these situations.

Case 7
You are called to the bedside of a patient because the nurse is concerned that the ventilator's pressure alarm is now going off. He was admitted for a COPD exacerbation and was intubated earlier in the day when he failed a trial of non-invasive ventilation. Earlier in the evening, the peak pressure was 45 cm H_2O while the static pressure was 25 cm H_2O. At the time she calls you, the peak pressure has risen to 60 cm H_2O and the static pressure is now 40 mm Hg. His heart rate has increased from 90 beats/minute to 110 beats/minute while his blood pressure has fallen from 110/85 to 90/70. The physical exam is noteworthy for diminished breath sounds on the left side of the chest.

What do "static" pressures represent on the ventilator?
The static or "plateau" pressure is representative of the compliance of the respiratory system (lung, chest wall and abdomen). In essence, it is telling you how much pressure is necessary to inflate the alveoli with each breath. Any problem which causes a fall in the compliance of the respiratory system will cause static pressures to rise. Examples of such problems include the onset of ARDS or pulmonary edema, large pleural effusions, pneumothorax, abdominal distention, or circumferential chest wall burns. The ventilator does not display this

pressure with every breath. Instead, you must use an inspiratory pause maneuver to see this value.

What do "peak" pressures represent on the ventilator?
The peak pressure is representative of the resistance in the system from the ventilator tubing all the way down to the segmental bronchi. Anything that affects the resistance of these tubes (mucous plugging, bronchospasm, blood clots, and kinked endotracheal tube) will cause the peak pressure to rise. The machine displays the peak pressure with every breath. It is important to know that, while some of the same factors contribute to both peak and static airway pressures, a number of things that affect peak pressure are external to the patient and do not necessarily reflect a change in the compliance of the patient's lungs.

Where do you think the problem lies with this particular patient?
There are two basic patterns of abnormalities that arise when there are pressure problems in mechanical ventilation: 1) the peak pressure rises but the static pressure remains unchanged. This situation suggests there is a resistance problem in the system; 2) the peak pressure rises but the static pressure rises as well. This situation suggests that the problem involves a change in the compliance of the respiratory system. In the case described above, both the peak and the static pressures have increased, suggesting this patient has a new "compliance" problem. Something has happened to the lungs, chest wall or abdomen to lower the compliance of the system. In a patient with COPD who develops unilateral diminished breath sounds, tachycardia and hypotension while on mechanical ventilation, you should be highly suspicious that the patient has developed a tension pneumothorax as the cause of the altered compliance.

What management steps should you institute at this point?
Whenever the static pressures change acutely and there is a change in lung compliance, you should obtain a chest x-ray to look for evidence of a pneumothorax, new pleural effusions, worsening edema or ARDS. You should also examine the patient's abdomen for evidence of over-distention, which might impair downward movement of the diaphragm and impair respiratory system compliance. If the patient becomes hemodynamically unstable and you have a high suspicion for a tension pneumothorax, you should place a large bore needle into the second intercostal space along the mid-clavicular line to decompress the pneumothorax before the chest x-ray is performed. Patient's who develop pneumothorax while on the ventilator will ultimately require tube thoracostomy (chest tube placement).

Case 8
At 11:00PM, you are called to the bedside of a 55 year-old man who was intubated one day prior for airway protection during a large upper

gastrointestinal hemorrhage due to esophageal varices. He has self-extubated and is lying in bed with the endotracheal tube in his hand. At the time this happened, he had been on an FIO$_2$ of 0.4 and a PEEP of 5 cm H$_2$O. His P$_a$O$_2$ earlier in the day was 100 mm Hg. The team had been planning to do a spontaneous breathing trial in the morning. At present, his oxygen saturation is 94% on 6L oxygen by nasal cannula. The nurse wants to know if you want to re-intubate him.

What should you do?
This patient has experienced what is referred to as an "unplanned extubation." There are two ways in which this can occur: In an "accidental" extubation, the endotracheal tube becomes dislodged by patient movement, transferring the patient between beds or other activities at the bedside. In "self-extubation" the patient pulls the endotracheal tube out by themselves.

In cases of unplanned extubation, it is not always necessary to reintubate the patient. The limited data available on this clinical situation suggests that when unplanned extubation occurs in a patient who the team was already considering separating from the ventilator (eg. planning for spontaneous breathing trials), the majority of these patients remain off the ventilator. If they initially appear stable following the unplanned extubation, you can observe them and not automatically reintubate them. The likelihood of them remaining off the ventilator is increased if their P/F ratio is > 200, their minute ventilation needs were low prior to the event and it was a self-extubation. In situations in which you were not considering separating the patient from the ventilator because they were too sick, or oxygen requirements or minute ventilation needs are high, the overwhelming majority of these patients require reintubation. They should not be observed and should, instead, be reintubated immediately.

In this particular case, the team had been considering doing a spontaneous breathing trial and the patient had low oxygen requirements. He is someone you could observe for a period of time rather than reintubating immediately. If you were concerned about on-going gastrointestinal bleeding and aspiration risk, however, you might need to reintubate him for that reason.

You decide not to reintubate the patient because his clinical status appears stable. Four hours later, you are called back to the bedside because the patient is laboring to breathe and his oxygen saturation has fallen into the upper 80% range on a venturi mask set with an F$_I$O$_2$ of 0.5. You obtain an ABG and it shows pH 7.30, PCO$_2$ 47, PO$_2$ 60 and bicarbonate 25.

Should you reintubate the patient or can you give him a trial of non-invasive ventilation?
This patient is "failing" extubation, as he has a rising PCO$_2$ and declining PO$_2$ within only 4 hours of his unplanned extubation. He should not be given a trial of non-invasive ventilation and should, instead, be reintubated. The data on

extubation failure demonstrates that patients who fail extubation and receive a trial of non-invasive ventilation end up being reintubated at the same rate as those who receive standard care (early reintubation). All the non-invasive ventilation appears to do in these cases is delay reintubation to a point when the patient may, in fact, be sicker and the intubation process may carry more risk.

Case 9

A 30 year-old woman has been intubated for 2 days following a motor vehicle accident. On morning rounds, she passed her spontaneous breathing trial and a decision was made to extubate her. She remains volume overloaded. The respiratory therapist removes the endotracheal tube and shortly afterwards, she is noted to have stridor. You are called to the room and find her struggling to breathe. She has audible stridor and suprasternal retractions on exam. Her oxygen saturation on 4L oxygen by nasal cannula is 94%.

What is the differential diagnosis for her problem?
This patient has developed post-extubation stridor. This is most likely due to laryngeal edema related to her volume overload but could also be due to laryngospasm, dislocation of the arytenoid structures or, in rare cases, bilateral vocal cord paralysis. Stridor is usually apparent immediately upon extubation but, in some cases, may develop over a period of hours following extubation.

How should you manage the patient? Does she need to be reintubated?
Management of the problem is guided by the patient's clinical appearance. Patients who appear to be in extremis or have other signs of severe respiratory distress should be reintubated immediately, although you should be aware that reintubation may be challenging due to the airway edema. Patients who appear more clinically stable can be managed with a different approach. They are often given dexamethasone and racemic epinephrine to help decrease the airway edema, although you must recognize that the data supporting these practices is poor and largely derived from the pediatric patient population. In addition, patients can be placed on a mixture of helium and oxygen (heliox) administered through a face mask. For a similar inspired oxygen concentration, a mixture of helium and oxygen is less dense than a mixture of nitrogen and oxygen and, as a result, has better flow properties through the edematous, narrowed airway. Administering this gas mixture can significantly decrease the patient's work of breathing until the airway edema resolves. Heliox cannot be used, however, in patients who require high inspired oxygen concentrations ($F_IO_2 > 0.4$) because the density of such gas mixtures is increased and the favorable flow properties of the gas mixture are eliminated. Laryngeal edema will typically resolve over 24 to 48 hours. If the patient has stridor on subsequent attempts at extubation or clinical suspicion for the other possible causes is high enough, Otolaryngology should be consulted to evaluate the laryngeal structures for evidence of the other problems noted above.

In cases where you suspect prior to extubation that a patient may have laryngeal edema and post-extubation stridor, what can you do to minimize the risk of this problem?

There is some evidence to suggest that if you suspect a patient may have laryngeal edema following extubation, you can decrease the risk of this problem by administering several doses of corticosteroids prior to extubation. Consensus does not exist regarding this practice, however and it is not uniformly done across institutions. There are also problems associated with correctly identifying which patients will require this intervention, a not insignificant issue in light of the desire to avoid unnecessary use of corticosteroids.

Case 10

A 57 year-old woman was intubated two weeks ago for respiratory failure resulting from ARDS due to urosepsis. About 5 days ago, her oxygen requirements declined such that she is now on an F_IO_2 of 0.4 with a PEEP of 5 cm H_2O. She has been doing poorly, however, on her spontaneous breathing trials and has not been able to be separated from the ventilator. On her latest spontaneous breathing trial this morning, her tidal volumes were between 125 and 150 ml and her respiratory rates rose to 35 after only 10 minutes of spontaneous breathing.

What items should you consider on the differential diagnosis for a patient who cannot be liberated from the mechanical ventilator?

When a person is repeatedly failing spontaneous breathing trials it is not sufficient to simply put them back on the ventilator and repeat spontaneous breathing trials day after day. You must consider why they are failing these trials and search for reversible causes whose elimination might facilitate separation from the ventilator. The main reason that most patients fail spontaneous breathing trials is that their primary process has not improved sufficiently. Beyond this, there are several broad categories of other problems that contribute to persistent ventilatory failure and inability to separate from the ventilator. The first category includes neurologic issues such as an insufficient or absent respiratory drive, the lingering effects of sedative or narcotic medications due to accumulation in body stores and anxiety. The second broad category involves problems of neuromuscular competence. The problems that fall into this category leave the patient too weak to do the work of breathing on their own and include issues such as critical illness polyneuropathy/myopathy, insufficient nutritional status, electrolyte abnormalities (hypokalemia, hypophosphatemia, hypomagnesemia), hypothyroidism, and lingering effects of neuromuscular blocking agents. The final broad category relates to problems in which the demands being placed on the patient are too high. In other words, the patient is being asked to do too much work in order to pass their spontaneous breathing trials. This category includes items such as high airway resistance due to COPD or airway secretions, poor respiratory system compliance stemming from ARDS,

pulmonary edema, auto-PEEP, large pleural effusions, or abdominal distention and, finally, high minute ventilation needs which may result from untreated infection, fevers, overfeeding, excessive dead-space or untreated metabolic acidosis.

What diagnostic steps can you consider to help you sort through this differential?
There are several simple measures that can be undertaken as part of the initial work-up including a physical exam to search for evidence of polyneuropathy, abdominal distention or volume overload, reviewing the patient's medication history and fluid balances, and ordering pertinent laboratory studies (potassium, phosphate, magnesium, TSH). Review of the patient's imaging studies is also useful as this may reveal the existence of persistent edema or large pleural effusions.

If your review of the pertinent patient information reveals that they are failing their spontaneous breathing trials because their minute ventilation needs are too high, you can consider ordering a study referred to as a "metabolic cart." In this test, special equipment is brought to the bedside by a respiratory therapist who then collects exhaled gases and arterial blood gases from the patient. This information is used to determine the rate of CO_2 production by the patient as well as to calculate the amount of their minute ventilation that goes to clearing their dead-space (dead-space fraction). Normal CO_2 production is about 250 ml/min. Patients with higher rates than this will have to breathe more (i.e. higher minute ventilation) in order to clear all of this CO_2 from their body and prevent rising arterial CO_2 levels and worsening pH. A normal dead-space fraction is about 0.25-0.35. If the dead-space fraction is too high (eg. > 0.60-0.65) the patient will also have to maintain a high minute ventilation just to maintain sufficient alveolar ventilation, prevent hypercarbic respiratory failure and preserve their acid-base balance.

If you identify that the cause of their problems is excessive CO_2 production, you can then search for the underlying cause of that problem. For example, the patient may be receiving too much nutritional support or may have an unrecognized metabolic acidosis. If you determine that the problem is excessive dead-space ventilation, you can search for causes of that problem such as pulmonary embolism or dynamic hyperinflation. However, in many cases, the high dead-space is due to the underlying lung disease (eg. slowly resolving ARDS) and there is not much you can do to fix the problem besides give the patient time to improve.

What can you do to help her get off the ventilator?
The most important thing to do in these cases is a systematic search for reversible causes of failure to separate from the ventilator and to treat those causes. For example, if your patient is volume-overloaded, they may benefit from

diuresis. The patient with large pleural effusions may benefit from drainage of those effusions.

In the absence of easily reversible causes, patients usually need time for their underlying problem to resolve or for them to regain strength. These patients should receive daily spontaneous breathing trials using either T-piece of pressure support ventilation. SIMV should not be used for this purpose. Patients should be given one trial per day and in between trials should be put back on a sufficient level of respiratory support (either Assist Control or pressure support ventilation) to prevent tachypnea and maintain patient comfort. The goal is to allow the patient to rest and to avoid ventilatory muscle fatigue. A good way to think of this is as follows: Suppose a person just ran a marathon. You would not ask that person to then run a 10 km race as a follow-up. You would let them rest. Similarly, the patient who tires out during spontaneous breathing should not be asked to do any more work. Their ventilatory muscles need the rest.

Over time, you will gain a sense of whether the patient is close to getting off the ventilator or whether they will need a lot more time. For patients in whom it is clear they will need a significant period of time to get off the ventilator, consideration can be given to transferring the patient to a long-term acute care facility that specializes in the care of such patients.

At what point do you consider placing a tracheostomy tube in this patient?
There are many different indications for placing a tracheostomy tube in a patient. For example, some patients need them because they have lost the ability to protect their airway.

Patients who cannot be separated from the ventilator for prolonged periods of time eventually require tracheostomy placement in order to reduce the risk of complications from long-term use of an endotracheal tube. Tracheostomy tubes also offer the benefits of decreased sedation needs, increased patient comfort, increased chances for the patient to eat or speak, ease of patient transfer and ease of taking the patient on and off the ventilator without the need for reintubation and its associated risks if they fail a period of spontaneous breathing.

There is considerable debate as to when the tracheostomy tube should be placed. Some centers advocate placing the tracheostomy tube within the first several days of a severe illness such as ARDS while other centers prefer to wait longer periods of time (eg. 3 or 4 weeks or even longer) before deciding to pursue this option. At this time, there are no good data to help resolve this argument and the decision is very institution and physician-dependent.