Mechanical Ventilation during a Pandemic Or Mass Casualty Event

At the time of this writing, the world has been afflicted with SARS-CoV-2, also known as COVID-19. The spread of this coronavirus has exposed the weaknesses of our healthcare system’s ability to handle large numbers of critically ill patients. Many patients infected with COVID-19 will progress to ARDS and require mechanical ventilation. While VV ECMO is appealing, it is a very resource-intensive therapy that is not practical to implement on a wide scale. Therefore, it falls to intensivists and others to treat many patients at once with ventilators that may or may not be capable of advanced modalities.

During this kind of a pandemic, we need to remember the truism that perfect is the enemy of good. Our focus should be on saving as many lives as possible, and we won’t have the luxury of being able to titrate every ventilator perfectly. As I write this chapter, patients with acute respiratory failure are being treated by healthcare practitioners with limited background in critical care medicine. They are doing the best they can with what they have. This chapter outlines my recommendation for how to best provide care to those infected with COVID-19, but it could easily apply to an outbreak of influenza or another infectious disease, or a mass casualty situation.

The first priority is to protect the people providing care. While it may sound noble to rush to the aid of a patient, the most important resource in a pandemic is the supply of qualified healthcare workers. Personal protective equipment is mandatory for anyone caring for critically ill patients—mask, face shield, gown and gloves must be worn. Jeopardizing the health and safety of those committed to the task at hand cannot be permitted. Likewise, viral filters must be attached to all ventilators (both conventional and noninvasive), and rooms should be retrofitted to be negative-pressure if at all possible. These measures will limit the transmission of the virus to healthcare workers and other patients.

The second priority in a situation where the number of patients needing critical care is stressing the capacity of the healthcare system is to follow the K.I.S.S. principle—keep it simple, superstar. Eliminate as many decision points as possible. Follow a plan that will work for 80% of the people, so you have the time and cognitive ability to deal with the 20%
who don’t respond or get worse. The concepts underlying this are discussed further, but if you want something that can be applied quickly to a lot of patients with acute respiratory failure, here it is:

If you follow this plan, the first two steps will work for most patients. The remainder will be supported with a switch to APRV or by utilizing prone positioning. Obviously, there will need to be some adjustments made to make sure the patients receive appropriate care, but I venture that this outline will serve as an acceptable “big picture” plan for providing
mechanical ventilation in a crisis situation. This frees up the respiratory therapist to troubleshoot problems and frees up the critical care physician to devote his cognitive efforts to those who don’t respond.

Basic Concepts

Instead of rehashing all of the concepts of lung protection and the pathogenesis of ventilator-associated lung injury, let’s review just a few key concepts. This will guide treatment of patients and help streamline ventilator protocols.

- A tidal volume of 4-8 mL/kg of predicted body weight (PBW) is desirable in most patients. This is close to the normal resting tidal volume of 5-6 mL/kg PBW.
- Alveolar stretch (and potential for injury) is most closely reflected by the end-inspiratory pressure, held for 0.5-1.0 seconds. This is also known as the *plateau pressure* (P\text{PLAT}). A P\text{PLAT} higher than 30 cm H\text{2}O is associated with an increased risk of lung injury, and some studies have suggested that the P\text{PLAT} should be kept at 25 or lower.
- Oxygen is obviously necessary for life, and the FiO\text{2} often needs to be increased in the critically ill. That said, there is little if any benefit to “normalizing” the SaO\text{2} or PaO\text{2}. A PaO\text{2} of 55-80 mm Hg, which corresponds with a SaO\text{2} of 88-95%, seems to be sufficient.
- On a related note, breathing 100% oxygen does cause absorption atelectasis, which could worsen shunting (areas of lung that are perfused but not ventilated). Maintaining some nitrogen in the alveoli will help stabilize them and prevent collapse. The FiN\text{2} should be at least 0.1, and ideally 0.4-0.5 if possible. That means the FiO\text{2} should ideally be no higher than 0.6.
- PEEP is used to recruit alveoli so they can participate in gas exchange, and to keep them open at end-expiration. This helps maintain the functional residual capacity of the lung and improves oxygenation.
- Even though the term “stiff” is used to describe the lungs of a patient with ARDS, that isn’t really the problem. The problem is that the lungs are small. The usable part, at least. Some areas of the lungs are normal, and some areas are damaged. The goal is to support the patient using the normal lung and to avoid causing further damage with excessive distending pressure or tidal volumes.
- If one breath with too high of a tidal volume can cause lung injury, then it follows that the more breaths, the more potential for lung injury. Keeping the respiratory rate lower may reduce the overall impact of mechanical ventilation on the patient’s lungs. Respiratory acidosis is generally well-tolerated.

Initial Ventilator Settings

Having standardized initial ventilator settings during a pandemic or mass casualty situation is helpful because it provides what should be adequate support to a large number of patients.
without having to rely on a clinician coming up with an individualized plan for each person. The hidden truth is that this is what we already do, most of the time, anyway. This assumes that the primary problem is hypoxemia, and that the underlying lung pathology is one of acute alveolar damage (e.g. ARDS).

Mode: Volume Assist-Control, with decelerating flow if possible

[There is growing evidence that pressure assist-control may be a better option for ARDS, but volume assist-control is the most commonly used mode and it's a good idea to stay with what people are comfortable with, at least to start. Decelerating flow is available on most ventilators, and the combination is known by different trade names—PRVC, VC+, CMV with Autoflow, APVcmv, etc.]

Rate: 16 breaths/minute

[This should provide a minute ventilation of about 100 mL/kg/minute, which is on the high end of the normal range. Remember that we’re not that concerned about hypercapnia.]

Tidal Volume: 6 mL/kg PBW

[This is the commonly accepted starting point for lung-protective ventilation.]

FiO₂: 50%, or 0.5

[Why not 100%? First off, most patients don’t need that much oxygen. This limits the number of adjustments that are needed. It also provides adequate alveolar nitrogen to keep alveoli open.]

PEEP: 10 cm H₂O; 15 cm H₂O for BMI > 50

[PEEP opens up alveoli and keeps them open, improving gas exchange and functional residual capacity. The more white stuff on the X-ray, the more PEEP you need. Heavier patients (BMI > 50) need more PEEP to counteract the increased restriction of their chest wall.]

These settings should work for most patients, at least to start. Of course, some adjustments will be necessary. Follow these guidelines:

- Check the P_{PLAT} every 4 hours. Lower the tidal volume as needed to keep the P_{PLAT} at 25 or less. The purpose is to avoid overdistension and injury to the healthy alveoli.
- Check the end-expiratory pressure at the same time. If the measured PEEP exceeds the set PEEP by more than 2, then “autoPEEP” is present—the patient doesn’t have enough time to exhale and the alveoli are getting overdistended. Lower the rate to 10 or 12, giving more time for the air to get out.
- Keep the SpO₂ 88-95%. This is more than enough to sustain life. There’s no need to get frequent ABGs to measure the PaO₂ as long as you feel that the pulse oximeter is
reliable, and the SpO₂ (surrogate for SaO₂) is much more important in terms of total oxygen delivery. *

- Keep the pH above 7.15, using sodium bicarbonate as needed. Otherwise, don’t worry too much about hypercapnic respiratory acidosis unless the patient has issues with intracranial hypertension or severe pulmonary hypertension. Most patients tolerate a respiratory acidosis quite well. This keeps ventilator adjustments (and respiratory therapist exposures) to a minimum. It may also prevent lung injury—every time the ventilator cycles, there’s a possibility of injury, so keeping the rate lower should be helpful.

- Troubleshoot as needed, but keep in mind that the ventilator is a means of support and will not do anything to make the patient get better any sooner. Your goal is to keep the patient alive and to not make him worse with injurious ventilator settings until he recovers from the illness.

When the Patient Gets Worse

Many patients will remain hypoxemic despite using a PEEP of 10 and a FiO₂ of 0.4-0.6. If the SpO₂ remains < 88%, escalating ventilator support may be in order. The first, and easiest, thing is to increase the PEEP and FiO₂. Increasing the PEEP to 15 and the FiO₂ to 0.8 will help in many cases, especially if the ICU staff accepts the fact that a SpO₂ of 87-88% is OK. If needed, the PEEP can be increased to 20, but be careful—a PEEP at this level can compromise hemodynamics and adversely affect gas exchange if alveoli get overdistended. If there is improvement with a PEEP higher than 15, allowing the Pplat to be 30 or less (instead of 25) will be necessary to have adequate tidal volumes.

APRV

If the patient is failing conventional ventilation, airway pressure release ventilation (APRV) is a reasonable next step. Most modern ventilators have an APRV mode (although they call it different things, like Bi-Vent or Bi-Level). APRV works by holding an inflation pressure for a prolonged time, usually 4-6 seconds, followed by a rapid depressurization of the circuit. The depressurization allows CO₂ to be eliminated from the alveoli, and the length of time of depressurization is short—0.5-1.0 seconds. The terminology for APRV can be confusing, but the concept is relatively simple.

- P_HIGH: The pressure that is maintained on the alveoli for the majority of the time. Think of this like you would CPAP. The pressure is usually 25-30 cm H₂O.
- T_HIGH: The time that the patient spends at P_HIGH.

* DO₂ = Cardiac Output × Hemoglobin × SaO₂ × 13.4 + [PaO₂ × 0.003]. As you can see, the vast majority of delivered oxygen is bound to hemoglobin, and so the saturation (SaO₂) is a much bigger component than the dissolved oxygen tension (PaO₂). The contribution of the PaO₂ is so small that that part of the equation is often omitted to make the math easier. So if a pulse oximeter is well-placed and functioning, so that the SpO₂ reflects the SaO₂, frequent ABGs become unnecessary.
• **P<sub>LOW</sub>**: The pressure that the ventilator “decompresses” to. This is usually zero, to permit maximal expiratory airflow, but it can be increased to 5-10 in cases of severe hypoxemia.

• **T<sub>LOW</sub>**: The time that the circuit is depressurized, or released. This is short—0.5-1.0 seconds—so that the recruited alveoli don’t collapse. It’s short enough to get the CO<sub>2</sub> out, but not any longer than that. The **T<sub>LOW</sub>** is usually adjusted to allow the peak expiratory flow to fall by about 50%. Any shorter, and CO<sub>2</sub> elimination will suffer. Longer than that (especially if flow falls to, or near, zero), and alveolar derecruitment will occur.

Ways to improve oxygenation in APRV:
- Increase the **P<sub>HIGH</sub>**, up to 35
- Increase the FiO<sub>2</sub>
- Increase the **T<sub>HIGH</sub>**, which increases the mean airway pressure
- Increase the **P<sub>LOW</sub>**, up to 10 (this will lead to a higher PaCO<sub>2</sub>, but oxygenation is more important)

Ways to improve ventilation (CO<sub>2</sub> elimination) in APRV:
- Increase the **P<sub>HIGH</sub>**—the higher the gradient between PHIGH and PLOW, the more gas is exhaled on the release
- Lower the **T<sub>HIGH</sub>**—this increases the frequency of the releases
- Increase the **T<sub>LOW</sub>**—this will allow more gas to be exhaled, but it risks alveolar derecruitment.

The primary advantage of APRV is that it increases the mean airway pressure, and oxygenation, without very high distending pressures. Holding the pressure for 4-6 seconds may recruit more alveoli without the strain of trying to inflate the lungs in a shorter time (like you do with conventional ventilation).

**Prone Positioning**

Prone positioning is a mainstay in the management of ARDS, and it has a lot of advantages. Proning helps “homogenize” the lungs by preventing regional overdistension. It also helps with secretion clearance and it takes the weight of the abdominal organs off the thorax. Prone positioning also has no physiologic cost—it doesn’t increase work of breathing, energy expenditure, or cause additional stress or strain on the lungs (unlike APRV). So, why don’t we just prone everyone? The primary reason is related to potential healthcare worker exposure. It takes 4-6 people to safely prone a patient, twice a day. This requires available workers, and there is always the risk of pathogen transmission even with personal protective equipment. For this reason, proning should be considered only if APRV fails or cannot be used (hemodynamic instability with the higher pressure, bronchopleural fistula, etc.).

If prone positioning is done, the prone period should be 16-18 hours, followed by a return to supine. Neuromuscular blockade and heavy sedation helps keep the patient from dislodging
tubes and lines, but it isn't mandatory. Proning should continue until the PaO₂/FiO₂ ratio is greater than 150 when supine.

**Therapies That Are Less Preferred**

Inhaled nitric oxide should not be used simply for hypoxemia. There is no data supporting its use in ARDS. If there is significant right ventricular dysfunction and cardiogenic shock, then inhaled NO might be useful.

High frequency oscillatory ventilation can be used, but it is not preferred for several reasons:
- Availability of the HFOV units is limited.
- Monitoring the patient is difficult, particularly when respiratory isolation measures are in place—there are no alarms on the HFOV unit to let you know when the patient has a problem.
- APRV improves oxygenation by increasing the mean airway pressure, which is the same thing that HFOV does; APRV is easier to use and doesn't require special equipment.
- Clinical trials have not shown a benefit from HFOV in ARDS, regardless of severity.

**Other Things**

Don't forget about the other things that go into caring for the critically ill. While the focus right now is respiratory care, we need to make sure that we're treating the patient as a whole.
- Nutrition should be provided early on, by the enteral route. Aim for about 25 kcal/kg/day of a commercially available formula, but don't stress if the patient doesn't tolerate full feeding. Just get something in the gut.
- Diuresis will help with oxygenation. Try to keep the patient within 5% of his normal body weight, and avoid excessive fluid boluses. If hemodynamics are tenuous, the combination of albumin/furosemide can be helpful.†
- DVT prophylaxis—enoxaparin, or heparin if there's renal dysfunction.
- Early mobilization once the respiratory and hemodynamic status of the patient has improved.
- Tracheostomy once the FiO₂ is down to 0.5 or less, the PEEP is down to 10 or less, and the patient is afebrile.

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† 12.5 grams of 25% albumin IV q6h, with each dose of albumin followed by 20-40 mg of IV furosemide. Alternatively, you could use a furosemide infusion along with the q6h albumin.
COVID-19 MECHANICAL VENTILATION PLAN‡

The purpose of this plan is to provide standardized ventilator settings for patients with presumed or confirmed coronavirus infection. Having a standardized plan should reduce the number of times that healthcare workers have to adjust ventilator settings and help prevent unnecessary exposure to respiratory secretions.

This plan is not intended to replace clinical judgment. Changing the ventilator mode or settings may be necessary for an individual patient, at the attending physician’s discretion.

INITIAL VENTILATOR SETTINGS:

Mode: PRVC§
Rate: 16
VT: 6 mL/kg
FiO₂: 0.5
PEEP: 10

Goal SpO₂ is 88-95%. There is no reason to increase the FiO₂ to get a SpO₂ higher than this range. Routine ABGs are not necessary and should only be ordered if it will lead to a significant change in strategy.

Goal pH is > 7.15. Otherwise, hypercapnia is acceptable.

Measure the end-inspiratory pressure q4h. If the Pplat is > 25, lower the VT until the Pplat is 25.

Measure the end-expiratory pressure q4h. If the measured PEEP is > 2 over the set PEEP, lower the ventilator rate to 10-12 and increase the VT to 8 mL/kg.

For hypoxemia (SpO₂ < 88%) despite the above settings, increase the PEEP to 15 and the FiO₂ up to 0.8.

For hypoxemia (SpO₂ < 88%) despite a PEEP of 15 and FiO₂ of 0.8, begin APRV:

- P_HIGH 30
- P_LOW 0-5, depending on degree of hypoxemia
- T_HIGH 5.0 sec
- T_LOW 0.8 sec—adjust for a drop in peak expiratory flow of 50%
- FiO₂ 0.8

Prone positioning (16 hours prone, 8 hours supine) should be initiated if APRV cannot be used successfully. Inhaled nitric oxide should not be used for hypoxemia. It is reserved for the treatment of right ventricular failure in cardiogenic shock.

‡ This can be printed out and posted by each patient’s bed, if you choose.
§ PRVC is on the Servo ventilator. If you’re using another ventilator, choose Volume Assist-Control with decelerating flow (also known as CMV with Autoflow; VC++; APVcmv; etc.)
Initial APRV Settings
P-high 30 cm H2O
P-low 0 cm H2O
T-high 5.0 sec
T-low 0.8 sec [Adjust for a drop in peak expiratory flow of 50%]
FiO2 80%

Ensure ETT is in place
Rule out tube obstruction
Rule out pneumothorax

SpO2 > 88%

In Preferred Order:
1. Increase P-high by 1-2, to a max of 35
2. Increase T-high by 1.0 sec
3. Increase P-low by 1-2, to a max of 10
4. Increase FiO2 up to 100%

Lower FiO2 (and P-low, if > 0) as tolerated for SpO2 88-94%

pH > 7.15
PaCO2 < 70

Decrease T-high by 0.5 - 1.0 sec, to a min of 3.0

Begin "Drop and Spread" Weaning when:
1. FiO2 is 50% or lower
2. P-low has been weaned to 0
3. pH is > 7.24
4. Patient is breathing spontaneously

Lower P-high for SpO2 88-94%

Increase T-high for pH > 7.24 and PaCO2 < 60

PATIENT DETERIORATES

P-high 12 and T-high is > 9 sec
FiO2 < 50%

Daily Spontaneous Breathing Trial
PSV with PS 7, CPAP 5

FAIL

PASS

EXTUBATE

Notes:
In cases of morbid obesity or abdominal compartment syndrome, increasing the P-high to a max of 40 may be necessary
Hypotension that occurs after starting APRV is often due to hypovolemia--make sure the patient has an adequate intravascular volume
Prone Positioning Checklist

Indications for Prone Positioning
Hypoxemic respiratory failure with the following features:
- PaO₂/FiO₂ ratio < 150 despite high PEEP or APRV
- Diffuse bilateral lung infiltrates
- Dorsal consolidation on CT (if available)

Contraindications for Prone Positioning
- Prohibitive risk of pathogen exposure to ICU staff
- Unstable cervical spine
- Significant long bone fractures
- Anatomical or treatment considerations that preclude proning

Minimum Necessary Personnel
1 respiratory therapist to control the airway and ventilator
4 turners (may be RN, MD, PCT, RRT, or student)
1 supervisor, who should not be involved in the proning process itself

Turning Process

A. Prepare the patient
- Apply lubricant to eyes and tape eyelids closed
- Remove any jewelry from the patient’s head or neck
- Remove any bite blocks
- Bolus necessary analgesia/sedation/neuromuscular blocker
- Confirm SpO₂ and ETCO₂ monitors are in place and functional

B. Position the personnel
- Two turners on either side of the patient (four total)
- Respiratory therapist at the head of the patient to manage the head, airway, and face pillow
- If available, one person to manage ventilator tubing and provide backup
- Supervisor at the foot of the bed

C. Pad the patient (if going from SUPINE to PRONE)
- Foam face pillow, making sure the endotracheal tube is not kinked (it may be necessary to cut out some of the foam padding)
- Two pillows each on the chest, lower pelvis, and shins
- Place a sheet over the patient (head to toe) and wrap snugly, bundling the pillows to the patient

D. Disconnect
- Central lines (after necessary boluses)
- Arterial lines
- Hemodialysis lines
- Cardiac monitor leads
E. Turn the patient—Supervisor should read each step aloud, with verbal confirmation by the team members

1. Supervisor confirms that the airway and ventilator tubing are under control by the respiratory therapist

2. Supervisor confirms that all lines and leads have been disconnected (SpO₂ and ETCO₂ monitors may be left in place, unless it interferes with the turning process)

3. On the supervisor’s count, the team will turn the patient onto his left side, keeping the pillows tight against the body using the sheet

4. Supervisor confirms that nothing needs to be repositioned

5. On the supervisor’s count, the team will turn the patient to the PRONE or SUPINE position, ensuring that the pillows and face pad are kept in the proper position

6. Respiratory therapist confirms to the supervisor that the endotracheal tube is at the proper depth and that the tube is not obstructed, with an appropriate ETCO₂ waveform

7. If PRONE, turners confirm to the supervisor that the patient is appropriately padded and that arms and legs are positioned comfortably

8. If SUPINE, turners remove padding

9. Reattach cardiac monitor leads, arterial line, and restart infusions

Prone position should be maintained for 16 hours, followed by 8 hours in the supine position. Eye and mouth care is essential. Tube feeding in the prone position is permissible if the tube is post-pyloric; otherwise, hold tube feeding while prone and increase the rate of feeding while supine.