Basic Physics of Mechanical Ventilation:

A ventilator is just a sophisticated leaf blower.
- It is essentially a FLOW DELIVERY MECHANISM.
- Inside, there is a precisely controlled turbine. It spins and generates a flow.

The CONTROL variables:
- FLOW
- VOLUME
- PRESSURE

If you specify an inspiratory time, you can only really change one other variable- either flow, volume or pressure. The others will be dependent on lung compliance and lung resistance.

They are all related.

A ventilator can be set to “control” one of these variables. A “controlled” variable will be treated as the main goal of the ventilators’ work. Its waveform will not change on the monitor, irrespective of lung compliance or resistance. This can be dangerous, as the ventilator will then mindlessly pursue that “guaranteed” variable. So if you set the pressure as a controlled variable, you bet the ventilator is going to deliver a breath with precisely that much volume. All the other variables may go to hell- but the volume will be perfect.

Clearly, limits must be set to ensure the other variables don’t get ignored by the idiot leaf-blower.

These “limits” are the limit phase variables. Unlike control variables (of which you can use only one) there can be dual limits set on a mode of ventilation. For example, one can have a volume-controlled pressure-limited mode, where a volume limit is targeted, and a pressure limit is also set (so that the ventilator does not exceed a certain pressure while mindlessly pursuing a guaranteed volume)

The PHASE variables:
Each breath has four phases: the initiation phase, inspiratory phase, plateau phase and the expiratory phase. Each phase has a variable which controls how it starts, how it proceeds, and how it finishes.

- TRIGGER:
The variable controlling the initiation phase; controls how and by whom the breath is initiated

- LIMIT:
The variable controlling the inspiration phase

- CYCLING:
The variable controlling when the breath changes from an inspiration to an expiration.

- PEEP:
The variable controlling what pressure is applied at the end of expiration: Positive End-Expiratory Pressure.
Alveolar Pressure ("Plateau Pressure") and the Inspiratory Hold Manoeuvre

Most ventilators have a "inspiratory breath hold" button where you can stop the cycling of the ventilator and observe what happens to the pressure as the breath is held.

As soon as the flow stops, airway resistance falls and the pressure – formerly required to push air through the airway- drops to a plateau. Difference between this plateau and the peak pressure therefore must be the AIRWAY RESISTANCE, or "Ohmic resistance", or "flow-dependent resistance". It varies depending on airway diameter and the degree of bronchospasm.

What the hell are we measuring?
This is very similar to measuring intrinsic PEEP with an expiratory breath hold.
Once again, airway pressure has 2 components: (the resistance of the airways and the pressure in the alveoli)

**It is the alveolar pressure you are interested in.** This is what determines your oxygenation.
However, you're never measuring that directly, because the pressure gauge is deep inside the ventilator. You're measuring the pressure in the circuit, that is to say, the airway.

**Airway pressure = (resistance of airways) + (alveolar pressure)**

Resistance of airways = flow x resistance
Alveolar pressure = (volume over compliance) + PEEP

If airway pressure = flow x resistance + (volume over compliance) + PEEP,
.... and you take away flow (by stopping the inspiration), and you ignore (or subtract) PEEP

Airway pressure = (0 x resistance) + (volume over compliance)

Thus, in absence of flow,

**Airway pressure = alveolar pressure**

The alveolar pressure should not get above 30 cmH₂O.
PEEP: Positive End Expiratory Pressure and its Consequences

PEEP is airway pressure artificially kept above atmospheric pressure. In the stupidest form of saying it, **PEEP IMPROVES OXYGENATION** and **REDUCES WORK OF BREATHING**.

**HOW PEEP IMPROVES OXYGENATION:**
- Increasing lung volume by recruiting collapsed alveoli (thereby reducing the intrapulmonary shunt)
- Pushes alveolar oedema fluid out of the alveoli and into the interstitium

**HOW PEEP REDUCES THE WORK OF BREATHING:**
- Supplies the pressure required to overcome airway obstruction
- Supplies the pressure required to overcome Intrinsic PEEP
- Either way, you get an ALI-ARDS sort of inflammatory lung injury. This is counterproductive.

**EFFECTS OF PEEP ON PRELOAD:**

**CVP: 5cmH₂O**

**PEEP: 10cmH₂O**

**CVP: 15cmH₂O**

**PEEP: 10cmH₂O**

- Increased intrathoracic pressure, thus
  - Decreased venous return,
  - Thus reduced left ventricular stroke volume
  - Thus reduced left ventricular contractility
  - Thus reduced left ventricular oxygen demand
  - If the left ventricle is decompensating because it is overfilled and overstretched ("congestive" heart failure) the decreased preload will push it back into the more efficient area of the Frank-Starling curve.

**On intrapulmonary shunt**

In stupid terms:
Shunt is the percentage of blood passing through the lungs which *doesn't* get oxygenated.

Normally its no more than 4%.

**On Pressure**

For some reason, we measure gas partial pressure in mmHg, and ventilator gas pressures in cmH₂O.

1mmHg = 1.36 cm of H₂O

Measured in cmH₂O, sea level pressure is 1033.

Thus, we ventilate our patients with pressure differences which are minute in comparison to the total pressures in play in the environment around them, or even to the pressures in their own cardiovascular system.

**EFFECTS OF PEEP ON PRELOAD:**

A WELL-FILLED PATIENT WILL NOT HAVE A DECREASE IN STROKE VOLUME. Venous return pressure will overcome intrathoracic pressure, and the heart will fill normally.
- If the PEEP is causing hemodynamic instability, the patient needs more fluid.

With "Basic Assessment and Support in Intensive Care" by Gomersall et al as a foundation. I built using the humongous and canonical "Principles and Practice of Mechanical Ventilation" by Tobins et al – the 1442 page 2nd edition
EFFECTS OF PEEP ON RIGHT VENTRICULAR AFTERLOAD

- Increased intrathoracic pressure = increased pulmonary artery pressure, thus
  - Increased right ventricular afterload
  - Thus, increased right ventricular work and thus oxygen demand
  - With a crappy right ventricle, this could really impair the left ventricular function- the left ventricle depends on the right for filling.

For normal RV systolic function, RV pressure must be greater than the sum of PA pressure and PEEP

<table>
<thead>
<tr>
<th>pressures</th>
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<tbody>
<tr>
<td>RA: 10cmH2O</td>
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<tr>
<td>RV: 20cmH2O</td>
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<tr>
<td>PA: 10cmH2O</td>
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<tr>
<td>PEEP: 5cmH2O</td>
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EFFECTS OF PEEP ON LEFT VENTRICULAR AFTERLOAD

- Left ventricular afterload = sum of systemic arterial resistance and left ventricular transmural pressure.
- Increased intrathoracic pressure = increased transmural pressure

i.e. the pressure generated by the left ventricle =

\[ P_{LV} = P_{transmural} = (P_{LV, systolic} + P_{PEEP}) \]

- In normal people, that contribution is tiny.
  - Remember, PEEP of, say, 10cmH2O is equal to 7.35 mmHg
  - In normal people, the left ventricle generates a systolic ejection pressure of about 80mmHg – and that's just to open the aortic valve.

- If your left ventricle is diseased, it can't pump effectively against the afterload, and pulmonary oedema ensues.
  - If pulmonary oedema ensues, the lung volume and lung compliance drop.
  - If the lung volume and compliance drop, the respiratory effort must generate lower pressures to suck more air in
  - The lower pressures decrease the transmural pressure, and thus increase the relative afterload.
  - The increased afterload causes more pulmonary oedema.

- Thus, by reducing the afterload-increasing effects of increased respiratory effort, PEEP it can't pump effectively against the afterload, and pulmonary oedema ensues.

PEEP-induced lung injury

- If you over-PEEP, you end up distending the lungs and cause barotrauma
  - This results in broken capillaries and increased alveolar-capillary permeability
  - The alveoli flood with fluid, and inflammatory mediators are released by the dying cells, resulting in more oedema, and, stupidly, increased PEEP requirements.

- If you under-PEEP, the atelectatic alveoli will open and close every breath cycle
  - The constant opening and closing of intermittently atelectatic alveoli will cause shear stress, and this also cause the release of inflammatory mediators.

PEEP-related organ effects

- reduced urine output due to decreased cardiac output, if your patient is already volume depleted
- decreased splanchnic blood flow due to decreased cardiac output, etc etc
- increased hepatic venous congestion due to decreased venous return to the heart
- increased INTRACRANIAL PRESSURE due to decreased venous return to the heart
- Decreased peribronchial lymphatic flow could actually decrease the rate of oedema removal, not to mention clearance of necrotic debris in pneumonia.
Intrinsic PEEP (PEEP$_i$), Gas Trapping and the expiratory hold manoeuvre

The Physics of Intrinsic PEEP:

- Airway opening pressure
- Alveolar pressure

When you exhale, airflow is driven by the difference between these two pressures.

The difference is created by the elastic recoil of the lungs and the chest wall.

The flow of air out of the lungs is also resisted by the expiratory airway resistance.

Typically, it takes 1.5 seconds to exhale a tidal breath.

Things which increase intrinsic PEEP are things which:
- Impair elastic recoil
  - Emphysema
- Increase expiratory resistance
  - Bronchospasm
  - Airway collapse at the equal-pressure point (where intrathoracic pressure equals intrabronchial pressure)

This is called “gas trapping”, or Dynamic Hyperinflation.

The key issue is that THERE IS NOT ENOUGH TIME FOR EXPIRATION.

The solution to this problem is to increase the I:E ratio. The patient needs more time to exhale the volume.

GAS TRAPPING
VOLUME CONTROLLED ACV

With "Basic Assessment and Support in Intensive Care" by Somersall et al as a foundation, I built using the humongous and canonical "Principles and Practice of Mechanical Ventilation" by Tobins et al – the 4th edition page 2nd edition
PEEP and the work of breathing due to Intrinsic PEEP

Intrinsic PEEP has all the benefits and problems of actual machine-generated PEEP. It is a end-expiratory positive pressure. A spontaneously breathing patient will have to fight hard to get air into their lungs if there is too much intrinsic PEEP; the pressures their respiratory muscles generate must turn that positive pressure into a negative pressure- and not just slightly negative, but **Significantly Negative** (to create a large enough gradient between the airway opening pressure and the alveolar pressure, to suck air past the obstructed bronchospasming airway).

The obstruction to **flow-dependent expiratory resistance**.

The $P_i$ is the positive intrathoracic pressure generated by the trapped gas.

Intrapleural pressure must overcome it in order to change the pressure gradient and allow air to flow into the lung (inside pressure must be negative compared to the outside).

**PEEP makes it easier to generate this gradient.**

\[
\begin{align*}
0 \text{ cmH}_2O & \quad 0 \text{ cmH}_2O \\
+5 \text{ cmH}_2O & \quad +6 \text{ cmH}_2O \\
-1 \text{ cmH}_2O & \quad +4 \text{ cmH}_2O \\
\end{align*}
\]

**Greatly Increased Inspiratory effort**

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**PEEP and the work of breathing due to airway narrowing**

**Work of breathing**

In the spontaneously breathing patient, the lungs must generate a certain negative pressure to draw air into the lungs. Let's call it $X$.

If there is an airway obstruction, work of breathing increases (because greater pressure must be generated to overcome the obstruction).

This is **flow-dependent airway resistance**.

In order to overcome it, the diaphragm must generate additional pressure. Let's call it $Y$.

...so ideally, if the PEEP you apply exactly matches that pressure, the effort of breathing is reduced back to its pre-obstructed state.

In absence of flow limitation, PEEP pressure is transmitted directly to the alveoli.

Because airway pressure = flow $\times$ resistance $+$ (alveolar pressure) $+$ PEEP, when resistance approaches zero, airway pressure = alveolar pressure $+$ PEEP.

Thus in a normal person, the PEEP goes straight to the alveoli.

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**PEEP indications and contraindications**

### ARDS:

- There are people who have reduced aerated lung volume
- *Yes, it helps to set a high PEEP to increase the aerated lung volume*
- Yes, very high PEEP can cause VILI
- *No, nobody can agree on how high the PEEP should be*

The **ALVEOLI Trial**: by ARDS network, (2004) - randomized 549 patients to high PEEP vs low PEEP

- Same volumes and plateau pressures; *No survival benefit*.
- The investigators recommend you have a try of high PEEP ventilation, and if it improves the oxygenation of your ARDS patient, then you are a winner; and if it doesn’t work, don’t feel too bad, because there is no evidence it makes any difference in the long run anyway.

### ASTHMA:

- These people have an increased intrinsic PEEP and higher airway resistance.
  The main problem is high expiratory resistance, so it takes a while to empty the lungs.
- Less responsive to PEEP than COPD
- This is probably because there is too much intrinsic PEEP; plus mucus plugs might be blocking some of the airways, making them immune to the benefits of PEEP.
- The result is that PEEP might actually cause worsening hyperinflation and increased gas trapping.
- *Some data suggests that low level PEEP is beneficial.*

### COPD:

- These people have an increased intrinsic PEEP and higher airway resistance.
- PEEP reduces the workload of respiratory muscles by counteracting both intrinsic PEEP and airway resistance
- Also, it may actually splint the airways, resulting in improved emptying of the trapped gas.
- *No trials to test whether PEEP in COPD is of any use whatsoever.*

### PULMONARY OEDEMA:

- The main problem is a negative intrathoracic pressure generated due to decreased lung volume and decreased lung compliance
- PEEP works directly on this problem.
- Most people would agree that about 10cmH2O of PEEP is the standard of care for cardiogenic pulmonary oedema.

### Contraindications to PEEP

- **Tension Pneumothorax** - it will get worse
- **Hypovolemic shock** – cardiac output will decrease
- **Bronchopleural fistula** - it won’t heal
- **High intracranial pressure** - it will get higher

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