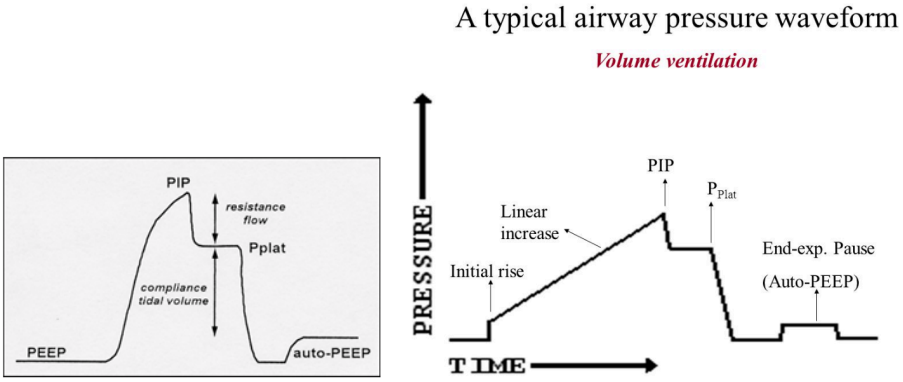
Basic Mechanical Ventilation
Respiratory failure is caused by failure to oxygenate (Type I respiratory failure), with resultant decrease in P_{O2} or failure to ventilate (Type II respiratory failure), with a resultant increase in P_{C02}.

Breathing Pattern consists of a Control variable, Breath sequence and a targeting scheme.
Ventilator can control only one variable at a time.

Equation of Motion: $P_{vent} = \dot{V} \times R + (V \times E) + PEEP$,
where P_{vent} is peak pressure, E is Elastance, V is volume, **R** is **resistance** and $\dot{V}$ is flow. **Elastance** is also the inverse of compliance.
Memorize this equation. This is the basis of all ventilator modes & the mechanism of action. If you understand this equation well, managing ventilator & understanding the variables & data on the ventilator becomes much easier.



Airway pressure= flow x resistance + alveolar pressure.
Alveolar pressure= (volume/ compliance)+ *PEEP*

Key points

- At end inspiration (inspiratory pause), there is ZERO flow & thus:
  - Airway Pressure (**plateau pressure**) = 0 x R + alveolar pressure = **alveolar pressure**
- **Flow ($\dot{V}$)** = Tidal Volume/Inspiratory time. Hence, in PC-CMV, when Inspiratory time is fixed and, if tidal volumes are increased, that means flow rate is increased.
- **Compliance** = ΔVolume/ΔPressure
- **Respiratory System Resistance** = ($P_{aw} - P_{plat}$) /Peak Inspiratory Flow Rate
- **Time Constant** = Compliance x Resistance
- **Work of breath** = Volume x Pressure. It is the work required to overcome the mechanical impedance to respiration. In other words, it is the work needed to overcome both elastic & airflow resistance.
- The **time constant** is a measure of the time needed for alveolar pressure to reach 63% of the change in airway pressure.

Understanding Equation of Motion in Pressure control mode (PC-CMV): A set pressure is applied (please note that driving pressure is the difference between set pressure and PEEP) to overcome the resistance and elastic recoil. Part of this driving pressure is used to overcome the resistance and thus air will flow into alveoli. The remaining pressure is used to overcome the elastic recoil force, resulting in alveolar distension. Now add the driving pressure to the extrinsic PEEP, you get the peak pressure.

Understanding Equation of Motion in volume control mode (VC-CMV): Just reverse the equation of motion to understand it easily. A set tidal volume/flow is delivered into the lungs. When air flows through the airways against the inherent resistance, it generates some pressure which is equal to flow x resistance. After air flows though the airways, it tries to open the alveoli against the elastic recoil thereby generating some pressure which is equal to the volume/ compliance. Then, finally you add the PEEP to these two pressures, it would give you the peak pressure.

The clinical application of the concept of **time constant** is that *very short inspiratory times* may lead to incomplete tidal volume delivery resulting in a decrease in PIP & MAP = hypercapnia & hypoxemia. Insufficient expiratory time may cause an increase FRC & inadvertent PEEP i.e. gas trapping.

During inspiration, airway pressure reflects the resistance to flow as well as the compliance of the system. Thus, an increase in inspiratory flow, bronchospasm, or airway secretions **increases PIP**, but does not directly affect P_{plat}.

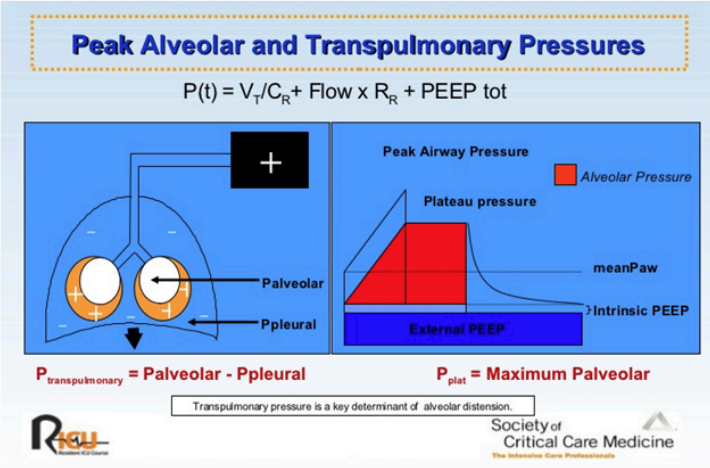
Pulmonary compliance is also important because it determines the ease of breathing. A lung that has decreased compliance (decreased stretchability) is difficult to inflate while one that has high compliance is easy to inflate but can be difficult to deflate.

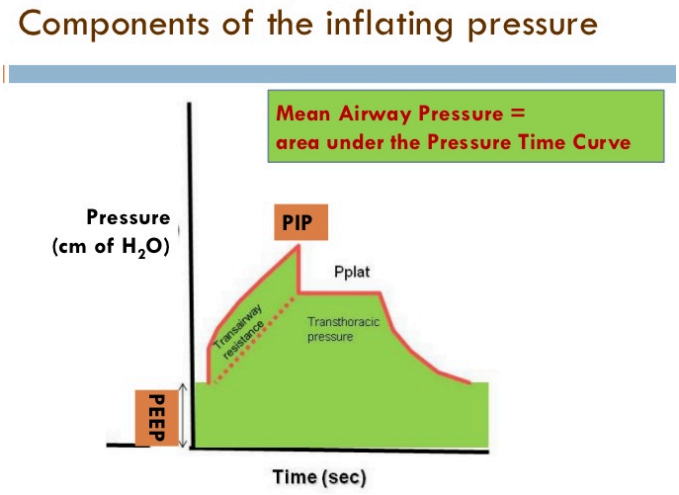
Compliance

Measurement of delivered tidal volume, peak airway pressure, plateau pressure (during an end-inspiratory occlusion lasting up to 2 sec) and PEEP permits the calculation of static & dynamic respiratory compliance.

Static compliance (Cst)	= TV/(Plateau pressure - PEEP) = 60-100 ml/cm H20	
Decreased by pneumonia, edema, atelectasis, pneumothorax, or restrictive lung disease.		
Dynamic compliance(Cdyn)	= TV/(Peak pressure - PEEP) = 50-80 ml/cm H20	
Airflow resistance becomes a factor. Decreased by bronchospasm, mucus plugging, kinked tube, or decreased static compliance.		

Normal airway resistance is less than 15 cm H20/L/s.





Basic Mechanical Ventilation

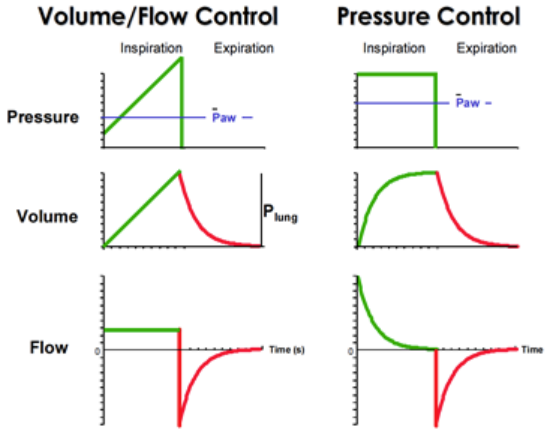
Transpulmonary pressure represents true lung pressure, & physiologically is > 0 cmH2O at end-exhalation.

Transpulmonary pressure < 0 cmH2O results in a lower FRC, lower compliance, & airways are prone to collapse on exhalation.

Plateau pressure is a surrogate of maximal alveolar distending pressure i.e. alveolar pressure.

Ventilator mechanism:

In volume control, flow & volume are fixed whereas pressure changes. In pressure control, pressure is fixed whereas flow & volume changes.



In volume control with descending ramp pattern, the pressure-time graph looks similar to pressure control mode.

In flow-time loop, look for expiratory limb as well. If the expiratory peak flow is high and expiratory time is less, it indicates less resistance.

In Spontaneous breaths, patient controls both start & size of breath. If machine controls any of them, it is a mandatory breath. However, spontaneous breath can be assisted or unassisted. Mandatory breaths are assisted by definition. Assisted spontaneous breath patterns include pressure support, automatic tube compensation, volume support, and proportional assist ventilation.

If machine controls any part of breath sequence, it is by definition a **mandatory breath**.

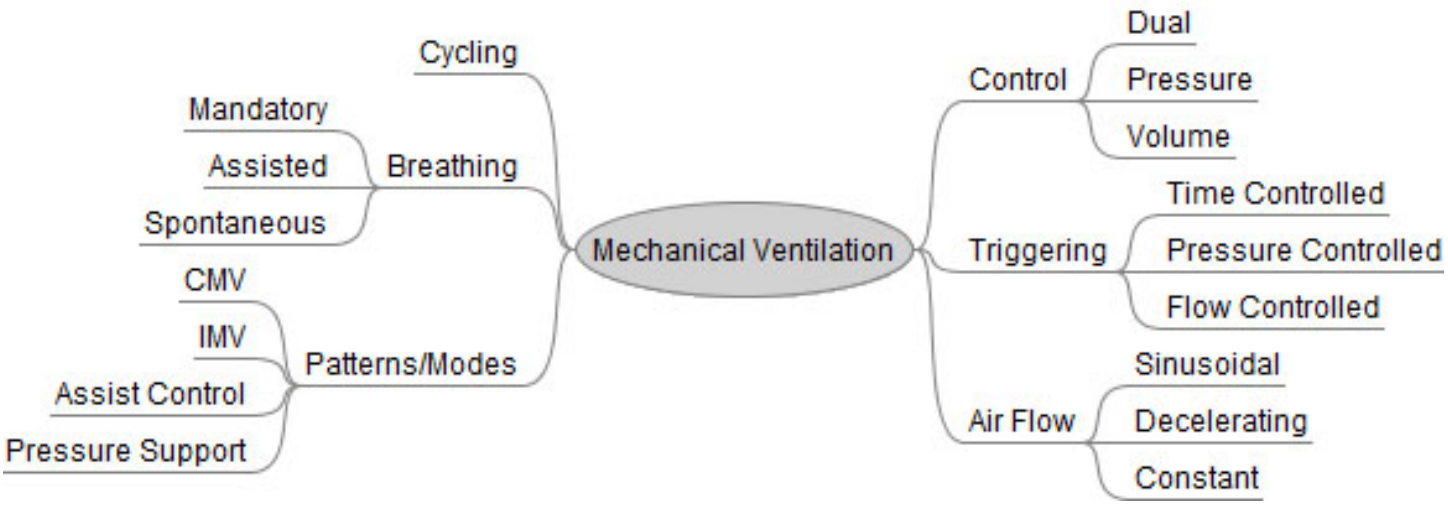
The classification of ventilators refers to the following elements:

1) **Triggering**: what causes the ventilator to start inspiration. Ventilators may be time triggered, pressure triggered or flow triggered.
   a. Time: the ventilator cycles at a set frequency as determined by the controlled rate.
   b. Pressure: the ventilator senses the patient's inspiratory effort by way of a decrease in the baseline pressure.
   c. Flow: modern ventilators deliver a constant flow around the circuit throughout the respiratory cycle (flow-by). A deflection in this flow by patient inspiration is monitored by the ventilator & it delivers a breath. This mechanism requires less work by the patient than pressure triggering.
2) **Control**: How the ventilator knows *how much flow to deliver*
   a. *Volume* Controlled (volume limited, volume targeted) & Pressure is variable
   b. *Pressure* Controlled (pressure limited, pressure targeted) & Volume is variable
   c. Dual Controlled (volume targeted (guaranteed) pressure limited)
3) **Cycling**: how the ventilator *switches from inspiration to expiration*: the flow has been delivered to the volume or pressure target - how long does it stay there?

Basic Mechanical Ventilation

a. Time cycled - such as in pressure controlled ventilation
b. Flow cycled - such as in pressure support
c. Volume cycled - the ventilator cycles to expiration once a set tidal volume has been delivered: this occurs in volume controlled ventilation. If an inspiratory pause is added, then the breath is both volume & time cycled

Normal minute ventilation is 5-8 L/min



	Triggered	Control	
VENTILATOR MODE	INITIATED	LIMITED	CYCLED
VC	Time	Volume	Volume/time
PC	Time	Pressure	Time
PRVC	Time	Volume	Volume
PS	Pressure/flow	Pressure	Flow
VS	Pressure/flow	Volume	Flow
CPAP	Pressure/flow	Pressure	Flow
SIMV	Time/pressure/flow	Volume	Volume/time

5 Basic Breath Types	Trigger	Target	Cycle
Volume Control (VC)	Elapsed time (rate per minute)	Flow (60 LPM or 1L/Sec)	Volume 500ml
Volume Assist (VA)	Patient Effort	Flow	Volume
Pressure Control (PC)	Elapsed Time	Pressure (20 cmH2O)	I Time 0.8 Sec
Pressure Assist (PA)	Patient Effort	Pressure	I Time
Pressure Support Ventilation (PSV)	Patient Effort	Pressure (PS 10 cmH2O)	Reduction of flow in circuit

For Pressure Support breaths: flow is delivered to elevate the circuit pressure to the Pressure Support setting (PS 10 cm H2O) and maintain it at that pressure until the flow drops below a variable percentage of the peak flow.

TAXONOMY:

8 Basic Breathing Patterns

Control Variable	Breath Sequence	Symbol
Volume	Continuous Mandatory Ventilation	VC-CMV
	Intermittent Mandatory Ventilation	VC-IMV
Pressure	Continuous Mandatory Ventilation	PC-CMV
	Intermittent Mandatory Ventilation	PC-IMV
	Continuous Spontaneous Ventilation	PC-CSV
Dual	Continuous Mandatory Ventilation	DC-CMV
	Intermittent Mandatory Ventilation	DC-IMV
	Continuous Spontaneous Ventilation	DC-CSV

VC-CMV (Assist control)

PC-CSV (No assist = CPAP mode, Assistance= pressure support or proportional assist or tube compensation)

Pressure Support is pressure or flow triggered, pressure limited, inspiratory flow cycled

Pressure-regulated volume control (PRVC or VC+), in which all breaths are mandatory, the rate is fixed, & the inspiratory pressure is varied to maintain a preset tidal volume. Most people think that PRVC is a volume control mode because we are setting up a target tidal volume. However, it is a pressure controlled & time cycled mode , which by definition is a pressure control mode. For each breath, the ventilator adjusts the driving pressure based on lung compliance & resistance, to deliver target tidal volume.

VENTILATOR MANAGEMENT

Four factors that are most important are:
- Time (RR)
- Volume (TV)
- Pressure (PIP, P_{plat})
- Flow

Volume Control Mechanical Ventilation

- Time - Set respiratory rate
- Volume - Set TV
- Flow - Set to deliver the TV. If the flow rate is too high, the volume is rapidly delivered to only the most compliant lung tissues (& not to the inelastic diseased tissues), at very high peak pressures. If the peak flow is too low, the patient will demand more gas than the ventilator is set up to supply & dyssynchrony with the machine occurs. A modem adjustment to this is the use of pressure augmentation; the ventilator senses that the patient's demands exceed the peak flow, & automatically increases the flow.
- Airway Pressure - Dependent on the interaction of the above and on the respiratory system compliance & airflow resistance

Pressure Control Ventilation

- Time - Usually set at 1 sec. Set the respiratory rate.
- Pressure - Set driving pressure
- Flow - dependent variable and changes to maintain constant pressure. The flow waveform is always decelerating in pressure control: this relates to the mechanics of targeting airway pressure; flow slows as it reaches the pressure limit.
- Volume - Dependent on the interaction of the above and on the respiratory system compliance and airflow resistance
- In pressure control, a pressure limited breath is delivered at a set rate. The tidal volume is determined by the preset pressure limit. This is a peak pressure rather than a plateau pressure limit.
- The key advantage of pressure targeted ventilation is unlimited flow during inspiration to satisfy the patient's demands. The harder the patient draws in, the greater the pressure gradient, and the higher the flow.

Pressure Support Ventilation

In pressure support, the patient triggers the ventilator and a pressure-limited breath is delivered: the patient determines the rate, the duration of inspiration & the tidal volume. The physician can determine how much work the ventilator can take from the patient, by altering the pressure limit.
Pressure is maintained at preset level until patient's inspiratory flow falls to a certain level (e.g., 25% of peak flow). The purpose of using CPAP (PEEP) is to restore functional Residual capacity to what is normal for the patient, when lung volumes are low: this reduces the workload of early inspiration.

Dual Control Modes - PRVC

- Time - Set RR
- Volume - Set TV
- Flow - dynamic based on patient variables
- Pressure - increases or decreases to maintain the set Tv (Dependent variable), but this is limited (i.e. controlled)

Airway Pressure Release Ventilation (APRV-BILEVEL)

CPAP is transiently decreased or "released" to a lower level during expiration.

Advantages

- Lower P_{aw} for a given TV but higher MAP
- Lower $\dot{V}E$, i.e. less dead space. $\dot{V}E$ = Minute Ventilation (L/min); $\dot{V}E$ is the total flow exhaled per minute., ie, $\dot{V}E = \dot{V}_A + \dot{V}_D$ (all flows). $\dot{V}_A$ = alveolar ventilation, $\dot{V}_D$ = dead space ventilation
- Spontaneous breathing
- Decreased sedation

Potential Disadvantages

- Volumes change with changes in compliance and resistance
- New technology
- Limited research and clinical experience. Limited studies did not show any benefit with outcomes, except for oxygenation.

Inverse Ratio Ventilation

Rationale

1. Sustained elevations in airway pressure may more effectively recruit collapsed alveoli.
2. I:E ratio of > 1: 1 may achieve higher mean airway pressure with lower peak alveolar pressure & lower PEEP than conventional mechanical ventilation (provided that excessive gas trapping does not occur).

Methods

1. Pressure controlled ventilation (PCV): prolong inspiratory time
2. Volume controlled ventilation: prolong inspiratory time indirectly by
    - Slow, constant inspiratory flow rate
    - Constant flow, with end-inspiratory pause
    - Decelerating flow
    - Problems
    - Marked increase in gas trapping (PEEPi)
    - Decreased TV with increased P_{CO2} , if there is insufficient time for expiration.
    - Discomfort (need deep sedation + paralysis)

PEEP

PEEP is *airway pressure artificially kept above atmospheric pressure at end expiration*.
Changes in cardiac output & lung compliance are likely to occur rapidly following an increase in PEEP, especially if PEEP>IO, & should be sought within the first 3-5 minutes at each level. As PEEP is

increased, PaO2 is measured sequentially as the primary index of a favorable response. If a substantial increase in PaO2 occurs with no evidence of either cardiac impairment or alveolar over distention (as assessed using static compliance), that PEEP level can be maintained & the FiO_2 titrated downward to maintain the target PaO2. Improvement in PaO_2 tends to occur more slowly than changes in cardiac function or compliance as PEEP is increased, & arterial blood gas specimens should be drawn 30-60 minutes after each change.
If PEEP is reduced prematurely, some alveoli may be unstable & collapse, which worsens oxygenation. If this happens, PEEP higher than the previous baseline level may be required to reopen the collapsed alveoli &, conceivably, the patient's requirement for mechanical ventilation may be unnecessarily prolonged. It is thus important to be able to predict when patients are 'ready' for PEEP weaning.
In obstructive lung disease, PEEP serves a different function from that in acute lung injury. Its purpose here is not to increase lung volume (which is already excessive), but to decrease the muscular effort required to trigger the ventilator or breathe spontaneously in the presence of dynamic hyperinflation & auto-PEEP.

Advantages of PEEP:
• increased mean airway pressure
• increased FRC (prevention of airway collapse)
• increase Pa02: increased capillary-alveoli interface for gas exchange, extra-vascular lung water displaced from alveolar interstitium to peribronchial interstitium
• maximizes recruitment of alveoli by preventing cyclic de-recruitment on expiration
• decreased airway resistance, especially with auto-PEEP
• reduced V /Q mismatch
• improved distribution of inspired gas
• reduced work of breathing by counteracting both intrinsic PEEP, overcoming airway resistance & improving compliance
• prevention of surfactant aggregation reducing alveolar collapse
• reduction in LV afterload (due to increased LV transmural pressure) - decreased preload & WOB

Disadvantages of PEEP:
- impaired CO_2 elimination due to increased dead space
- decreased RV preload, increased RV afterload, decreased LV compliance (due to intra-ventricular septum displacement)
- reduced urine output through increased ANP, decreased GFR & increased ADH
- increased pulmonary vascular resistance (PVR) (in West's zone I & II where increased alveolar pressure exceeds venous pressure)
- decreased flow in West's zone I causing increased dead space (PA > Pa > Pv)
- may worsen right to left intracardiac shunt by increased pulmonary vascular resistance
- decreased hepatic artery & portal venous flow (liver congestion & LFT changes)
- increased ICP
- decreased peribronchial lymphatic flow(? decrease pneumonia)
- Decreased splanchnic blood flow (at >20 cmH_2O PEEP) due to decreased cardiac output.

PEEP Titration:
PEEP titration the most neglected aspect in the care of mechanical ventilation. Many people set the patients on one PEEP and forget about adjusting it. Ideal PEEP is a dynamic process and keeps changing based on lung mechanics. Ideally, one should check the hemodynamics frequently (at the minimum once a day) & adjust the PEEP accordingly.

Ideal PEEP

The ideal level of PEEP is that which prevents derecruitment of the majority of alveoli, while causing minimal over distension of healthy alveoli. The goal of PEEP titration is always to recruit more lung and thereby improve lung compliance. Sometime, increasing PEEP will have no effect on diseased lung & will all affect healthy lung leading to overdistension of healthy alveoli. In those circumstances, compliance may actually decrease with drop in PO_2. So, don't assume that only increasing PEEP will improve compliance or P_{02}. (Most frequently, people always increase PEEP in hypoxic patient without paying attention to lung mechanics).

In patients who are on mechanical ventilators, one can adjust the PEEP in many different ways -

- Adjust PEEP & if it improves compliance, it will result in higher tidal volume (in pressure control mode) & lower peak/Plateau pressures (in volume control mode).
- Adjust PEEP & if it improves compliance, it will result in higher P_{02} and SaO2. If we always keep sats around 90%, a higher P_{02} will reflect in higher Sa02, which can be noticed without a need for ABG.
- Adjust PEEP & if it improves compliance, plateau pressure will come down.
- Adjust PEEP & if it opens up new alveoli, EtCO2 should come down.
- If increased PEEP results in overdistention of healthy alveoli, it leads to compression of pulmonary vasculature leading to drop in cardiac output.
- PEEP can be adjusted based on pressure-volume curves, ideal PEEP being just above lower inflection point. Please note that if the flow rates are higher than 60 L/min, it increases resistive load & overestimates the lower inflection point leading to unnecessary PEEP.
- If one doesn't want to spend time on the ventilator, use at least ARDS.net table or LOV's strategy.
- In general, a P/F ratio < 150 will benefit from low Fi02 & higher PEEP strategy & P/F ratio > 150 will benefit from higher FiO2 & low PEEP strategy.
- PEEP setting in **obese**: **Transpulmonary pressure** (or transalveolar pressure) is the pressure difference between the alveolar space & the pleural space. It is the pressure required to maintain alveolar inflation & is therefore sometimes referred to as alveolar distending pressure. In morbidly obese patients, the massive chest wall adds positive pleural pressure. The plateau pressure that we are most worried about is actually a reflection of alveolar pressure but not the alveolar distending pressure, which is the one that's actually harmful for the lungs. So, in obese patients, even though the plateau pressure is > 30, their alveolar distending pressure may be much less, depending on their positive pleural pressure. In those patients, don't hesitate to increase PEEP, even as high as 40. A PEEP of 20 in a obese patient with a pleural pressure of +20 is equivalent to end expiratory transalveolar pressure of 0. It may lead to atelectasis.

PEEP in Unilateral or Markedly Asymmetric Lung Disease

Patients who have lobar pneumonia, lobar or whole-lung atelectasis, and other markedly asymmetric pulmonary involvement present a special problem in mechanical ventilation, particularly in the presence of severe hypoxemia. Such patients illustrate why PEEP should not automatically be applied as treatment for hypoxemic respiratory failure. Respiratory system compliance is much higher in relatively normal areas of lung than in areas of consolidation or collapse. As a result, application of PEEP may preferentially expand these more normal areas and not produce the desired effect in the involved lung. Distention of normal lung tissue stretches and narrows pulmonary vessels, which can raise pulmonary vascular resistance sufficiently to divert blood to the abnormal areas. Accordingly, applying PEEP can worsen rather than improve arterial oxygenation in such instances.

PEEP in Acute brain injury:

Patients who have closed head injury or other acute brain insult may lose the normal auto regulation of cerebral perfusion pressure (CPP). In such patients anything that decreases mean arterial pressure or

raises central venous pressure must be avoided. Thus, PEEP should be used cautiously in patients who have acute brain injury, as the raised intrathoracic pressure is transmitted via the vertebral veins to the central nervous system. Maneuvers that induce coughing and may raise intracranial pressure, such as tracheal suctioning, are avoided whenever possible in these patients.

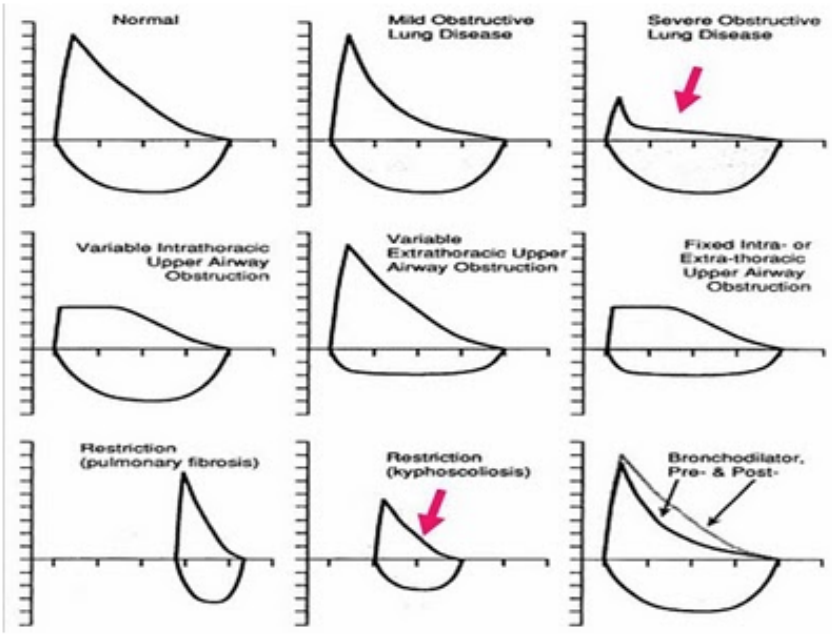
Physiologic effects of positive-pressure ventilation

1. Impaired cardiac function due to reduced right ventricular preload as a result of raised mean intrathoracic pressure and increased right ventricular afterload because of increased lung volume (which may not happen if gas trapping does not occur and if tidal residual volumes are physiologic).
2. Marked increases in lung volume cause an increase in pulmonary vascular resistance. This increases right ventricular afterload, which may compromise the forward output of that chamber. The increase in right ventricular volume associated with raised pulmonary vascular resistance may in turn impair left ventricular function, since both ventricles share the interventricular septum, pericardial sac, and certain circumferential muscle fibers.
3. Increase in juxtacardiac pressure that occurs as a result of the increase in intrathoracic pressure induced by positive pressure causes a functional reduction in left ventricular afterload.
4. Positive-pressure ventilation does not always impair cardiac function, & in certain circumstances it may even improve it. Although the work of spontaneous breathing normally accounts for a small proportion of overall oxygen consumption, this may increase drastically during acute respiratory failure. When cardiac function is severely impaired, as in cardiogenic shock, the heart may not be able to meet the demands imposed by this excessive work of breathing.
5. Increased intracranial pressure. Positive pressure ventilation can increase jugular venous pressure, which in turn can impede venous return from the brain & cause increased intracranial pressure (ICP).

Flow-Volume Loops

Flow limitation can arise from 3 factors:

1. Decreased maximal static expiratory pressure (neuromuscular disease)
2. Increased airway resistance (asthma)
3. Decreased lung elastic recoil pressure (emphysema)



Airway Pressure Release Ventilation (Inverse Ratio Ventilation)
Airway pressure release ventilation (APRV, also known as Bi-Level and Bi-phasic is a time-cycled, pressure-targeted form of ventilatory support. APRV is actually a variation of pressure-targeted SIMV that allows spontaneous breathing (with or without pressure support) to occur during both the inflation and the deflation phases. APRV differs from conventional pressure-targeted SIMV in the inspiratory: expiratory (I: E) timing.
Specifically, conventional pressure-targeted SIMV uses a "physiological" inspiratory time with I: E ratio < 1: 1. Spontaneous breaths thus occur during the expiratory phase. In contrast, APRV uses prolonged inspiratory time producing so-called inverse ratio ventilation (I:E ratios of up to 4 or 5: 1). Spontaneous breaths thus now occur during this prolonged inflation period.
APRV has also been described as CPAP with regular intermittent and brief releases in airway pressure. The release phase results in alveolar ventilation and removal of C02. APRV, unlike CPAP, facilitates both oxygenation & CO_2 clearance. The CPAP drives oxygenation while the timed releases aid in CO_2 clearance.
Several "rules" are involved in APRV:
1. The **expiratory time** is the key variable - it should be short enough to prevent derecruitment and long enough to obtain a suitable tidal volume. The expiratory time is set between 0.4 to 0.6 seconds - the tidal volume is your target (between 4 & 6ml/kg). If the tidal volume is inadequate, the expiratory time is lengthened; if it is too high (> 6ml/kg) the expiratory time is shortened.
2. The high CPAP (PEEP) level is set at plateau pressure or the mean airway pressure level from the previous mode (pressure control, volume control, etc). If you are starting off with APRV then start high (28cmH_2O or less) and work your way down. Higher transalveolar pressures recruit the lungs.
3. Low PEEP is set at 0 cmH_2O. The large pressure ramp allows for tidal ventilation in very short expiratory times.
4. The inspiratory time is set at 4-6 seconds (the respiratory rate should be 8 to 12 breaths per minute never more).
5. Neuromuscular blockade should be avoided: the patient should be allowed to breathe spontaneously (this is beneficial). The breaths can be supported with pressure support - but the plateau pressure should not exceed 30 cm H2O.
6. There are two different ways to wean patients from APRV. If lung mechanics rapidly return to normal, the patient should be weaned to pressure support. If ARDS is prolonged, then the high CPAP level is gradually weaned down to 10 cmH_2O, & then the patient is converted to a standard vent wean.

PRVC:
It is a PC-CMV dual mode. The volume target is set and there is also a set pressure limit. The machine tries to deliver the same tidal volume but at the same time trying to limit the pressure. If pt. can't get adequate tidal volume in one breath, the machine increases the driving pressure for next breath. If pt. gets more tidal volume, machine decreases the driving pressure. Thus, the driving pressure is changed between breaths.
Disadvantages: when the patient demand is increased (when work of breathing is increased), the patient will get more tidal volume for the set pressure, but at the cost of increased work of breathing, The ventilator will sense this as increased tidal volume from increased compliance and then it will decrease the support i.e. decrease the driving pressure resulting in worsened work of breathing.

High Frequency Oscillation Ventilation (HFOV)

- It is ventilation with small tidal volumes at high frequencies i.e. TV 1 - 4mL/kg at 60-100/min & is often used with high PEEP. It is essentially a vibrating CPAP.
- 1 Hz = 1 cycle/sec = 60 breaths/min

Basic Mechanical Ventilation

- The only indication is when conventional ventilation fails and if no ECMO is available.
- Contraindications are severe airflow obstruction and raised intracranial pressure.
- It maintains lung recruitment, avoids over distention, and does not rely on bulk flow for oxygenation & ventilation.
- Mean airway pressure is very high & pressures oscillate around MAP.
- TV is less than dead space -> normal bulk flow inadequate -> but gas delivery into the system still
- undergoes gas exchange by a number of mechanisms but not well understood. Both inspiration & expiration are active processes.
- Utilize highest possible frequency to minimize tidal volume (only decrease for CO2 control if amplitude of oscillations maximal).
- Factors that determine PaO2 are FiO2 & mean airway pressure.
- Factors that determine PCO2 are amplitude (power) of oscillations, frequency of oscillations, inspiratory time and cuff leak. Increase in amplitude or reduction in frequency leads to increased tidal volumes.
- Typical initial settings:
  1. Bias flow 40 L/min
  2. Inspiratory time 33%
  3. mP_{aw} 34 cm H2O (also called a CPAP or PEEP in this mode)
  4. FIO_2 1.0
  5. Amplitude (delta P) 90 cm H_2O. It is pressure swing around MAP.
- Initial frequency based on most recent arterial blood gas:
  - pH <7.10 =4Hz
  - pH 7.10- 7.19 =5Hz
  - pH 7.20- 7.35 =6Hz
  - pH >7.35 =7Hz
- Complications include decreased venous return, intracranial hemorrhage from high MAP, pneumothorax, increased need for sedation and paralytics.
- Oxygenation problems: Increase FiO2, increase MAP by 2-3 cmH_2O every 30 mins, increase inspiratory time, repeat recruitment maneuver, consider ECMO & consider flolan or NO.
- Inadequate ventilation: Increase delta P by 5-10 cm up to a max of 90cm H_2O, decrease frequency by 1hz every 2-3 hours & check for cuff leak
- OSCILLATE trial: Increased mortality with early use of HFOV in moderate to severe ARDS. Also, there is higher risk of hemodynamic instability due to higher mean airway pressures.

Recruitment Maneuvers:

Recruitment maneuvers are transient increases in transpulmonary pressure designed to open up collapsed alveoli. A sustained pressure above the tidal ventilation range is applied, and PEEP is used to prevent derecruitment. In the absence of PEEP many alveoli expand and collapse during the respiratory cycle. Others,"sticky" alveoli do not participate in gas exchange. Using PEEP, the alveoli that open in inspiration remain recruited. However some "sticky" alveoli remain collapsed.

Two factors influence whether or not recruitment maneuvers are successful: the pressure applied must be in excess of the current plateau pressure, and the pressure must be sustained, in order to inflate lung units with long time constants. The most effective method of doing this is to apply a CPAP of I 0 em higher than plateau pressure for 30 to 40 seconds to restore alveolar recruitment.

Recruitment may worsen oxygenation by shunting blood to poorly aerated regions and may also contribute to ventilator-induced lung injury (VILI) due to over distension and repeated opening of healthy alveoli.

The main causes are
1. Trigger insensitivity.
2. Missed trigger or ineffective or wasted efforts. Common causes arc inadequate respiratory efforts or increased flow to the ventilator circuit during nebulizer treatments or Auto PEEP in COPD
3. Double triggering due to coughing, low tidal volumes as in ARDS, low flow states or increased ventilatory demand
4. Trigger delay due to decreased respiratory drive, sleep, hypocapnia & sedation.
5. Auto trigger due to increased sensitivity or leaks in the system
6. Flow starvation
7. Inadequate volumes
8. Premature cycling due to short i-time or coughing
9. Too fast or too slow pressurization rate

PEARLS:
• When present, intrinsic PEEP can increase work of breathing. It can be offset by applying extrinsic PEEP (up to 80% of iPEEP).
• Ramp style flow pattern results in lower peak airway pressure and higher mean airway pressures compared to square wave pattern.
• In ramp style pattern, peak airway pressure is the plateau pressure as flow returned to zero at end inspiration. There is no need to put an inspiratory pause.
• The plateau pressure is the pressure applied to the small airways and alveoli. It is believed that control of the plateau pressure is important, as excessive stretch of alveoli has been implicated as the cause of ventilator induced lung injury. The peak pressure is the pressure measured by the ventilator in the major airways, & if plateau pressure is normal, it strongly reflects airways resistance.
• In pressure controlled ventilation, the pressure limit is (usually) the plateau pressure due to the dispersion of gas in inspiration. In volume control, the pressure measured by the ventilator is the peak airway pressure, which is really the pressure at the level of the major airways.
• Dyssynchrony is a term which describes a patient fighting the ventilator. If it persists after adequate sedation, in the majority of cases it is due to inadequate flow delivery from the ventilator. If the flow of gas is inadequate, the patient attempts to suck gas out of the ventilator - which is extremely unpleasant. This only occurs in volume control modes. In pressure control, flow is unlimited! - the reason is that flow is related to the pressure gradient between the upper and lower airway - a deeper attempted inspiration makes the pressure in the alveoli more negative in relation to the upper airway, and the pressure gradient is larger - and the flow greater.
• Removal of positive pressure ventilation may unmask LV failure & may precipitate pulmonary congestion by increasing venous return.
• PEEP < 10 is unlikely to affect CVP
• There is no evidence that a gradual reduction of ventilation support accelerates the ventilator discontinuation process. Patients either pass weaning trial or not.
• RSBI (Tobin Index) is a measure of readiness for weaning trial. It is applied before starting the weaning process. Score of< 105 signifies that patient is ready for weaning trial.
• Causes of failed extubation: Upper airway resistance (supraglottic edema), poor cough and excessive secretions, poor airway reflexes leading to aspiration, respiratory weakness masked by pressure support, increased cardiac load induced by removal of CPAP, and onset of new pathology.
• Minimal vent. Settings for weaning: Martin Tobin has argued that adding either 5 cm H_2O as ''physiologic" PEEP or pressure support of 7 cm H20 to overcome the resistance in an endotracheal tube (or both, as is usually done) may actually reduce the "spontaneously" breathing patient's workload by >40% and may result in failure once extubated. An alternative is to have the ventilator set on "flow-by,"

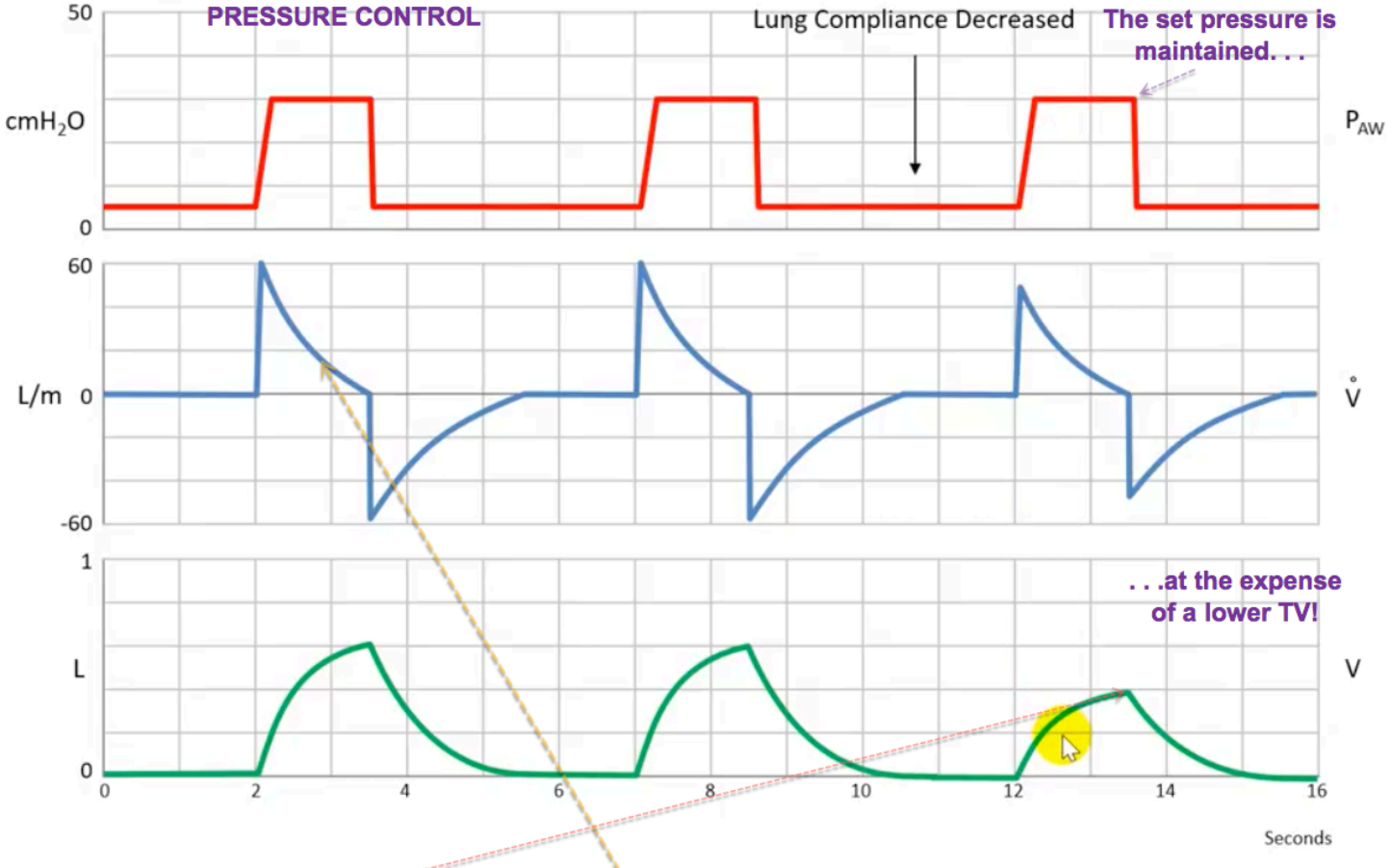
Basic Mechanical Ventilation
with pressure support & PEEP set at zero
• Failure of exhaled tidal volume to match inhaled tidal volume may be due to auto-PEEP or leak around ET cuff or chest tubes or broncho pleural fistula.
• Extubate high risk patients to NIPPV, which would reduce reintubation rates, especially in COPD.
o Single Lung ventilation: Eg. Single lung transplant, bleeding in unilateral lung. Set initial tidal volume at 4 ml/kg & adjust RR to maintain normocapnia. PEEP in normal lung may help in preventing atelectasis but at the same time, the shunt on non ventilated lung increases due to increased diversion of blood flow from normal to abnormal lung induced by PEEP.
o Low tidal volume strategy is good in even non ARDS but those at risk for ARDS like multiple transfusions, sepsis, pancreatitis, trauma or high risk surgery.
o Cardiac oscillations as trigger: base line of pressure time wave forms moves up and down with the heart beat thereby triggering synchronized breaths.
o We measure gas partial pressure in mmHg, and ventilator gas pressures in cm H_2O. 1mmHg = 1.36 cm of H_2O. Measured in cm H_2O, 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.
o Weaning from ventilator is a wrong concept. Liberation from ventilator is the right concept. Weaning with decreasing pressure support day by day is a poor strategy. When the primary lung pathology is reversed, we should aim for liberation from ventilator.
o Changing from square wave to ramp pattern increases the I-time, if the flow rate is constant. Hence, E-time may be lessened & pt may go into dyssynchrony. Hence, if pt is tachypneic & you want to keep long enough E-time, *increase the flow rate when changing from square wave to ramp pattern*.
o In normal patients, expiratory time constant is around 0.3 seconds & hence would need about 1.5 secs for expiration, i.e. 5 time constants.

Reference

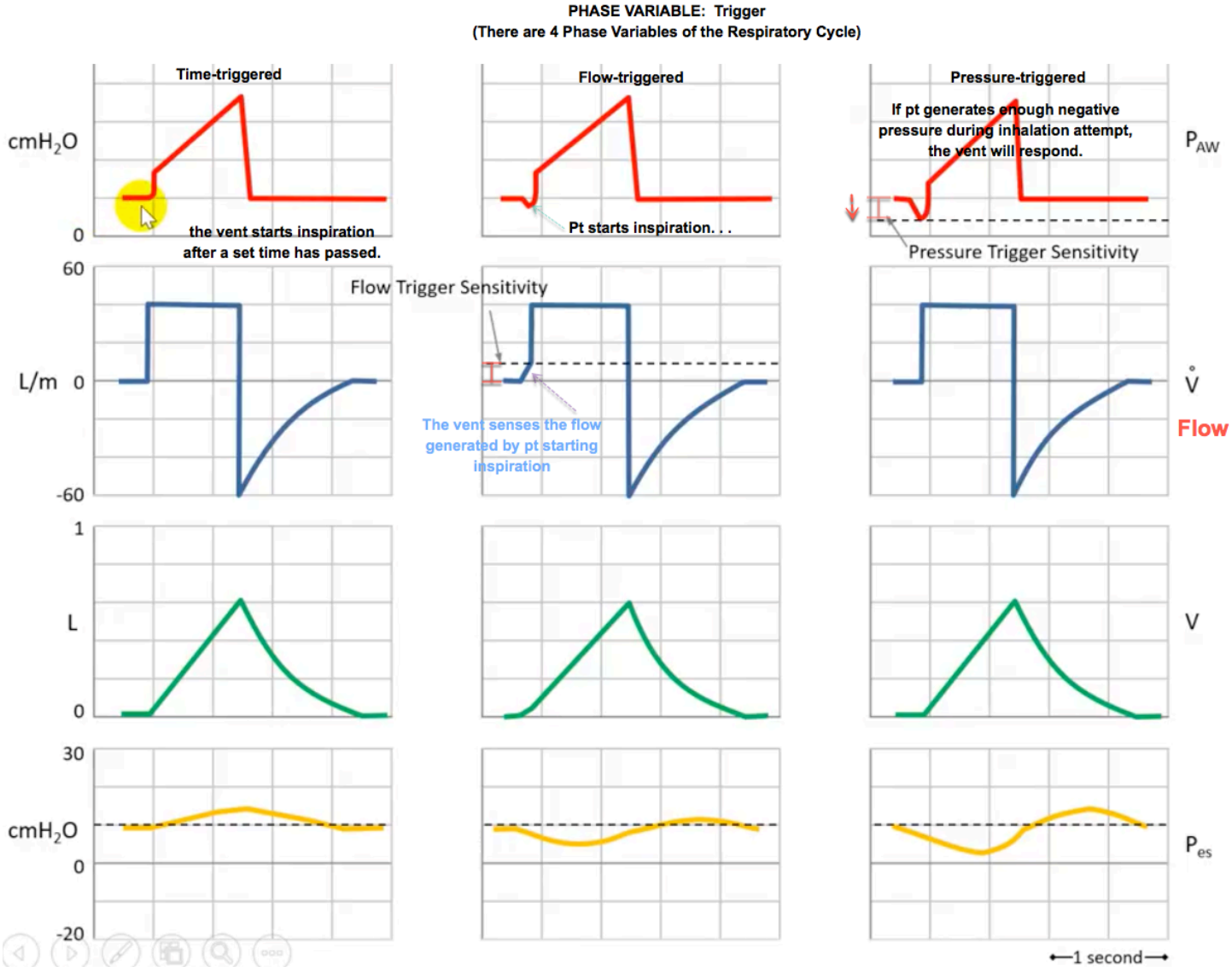
http://pocketicu.com/index.php/2017/02/24/mechanical-ventilation/

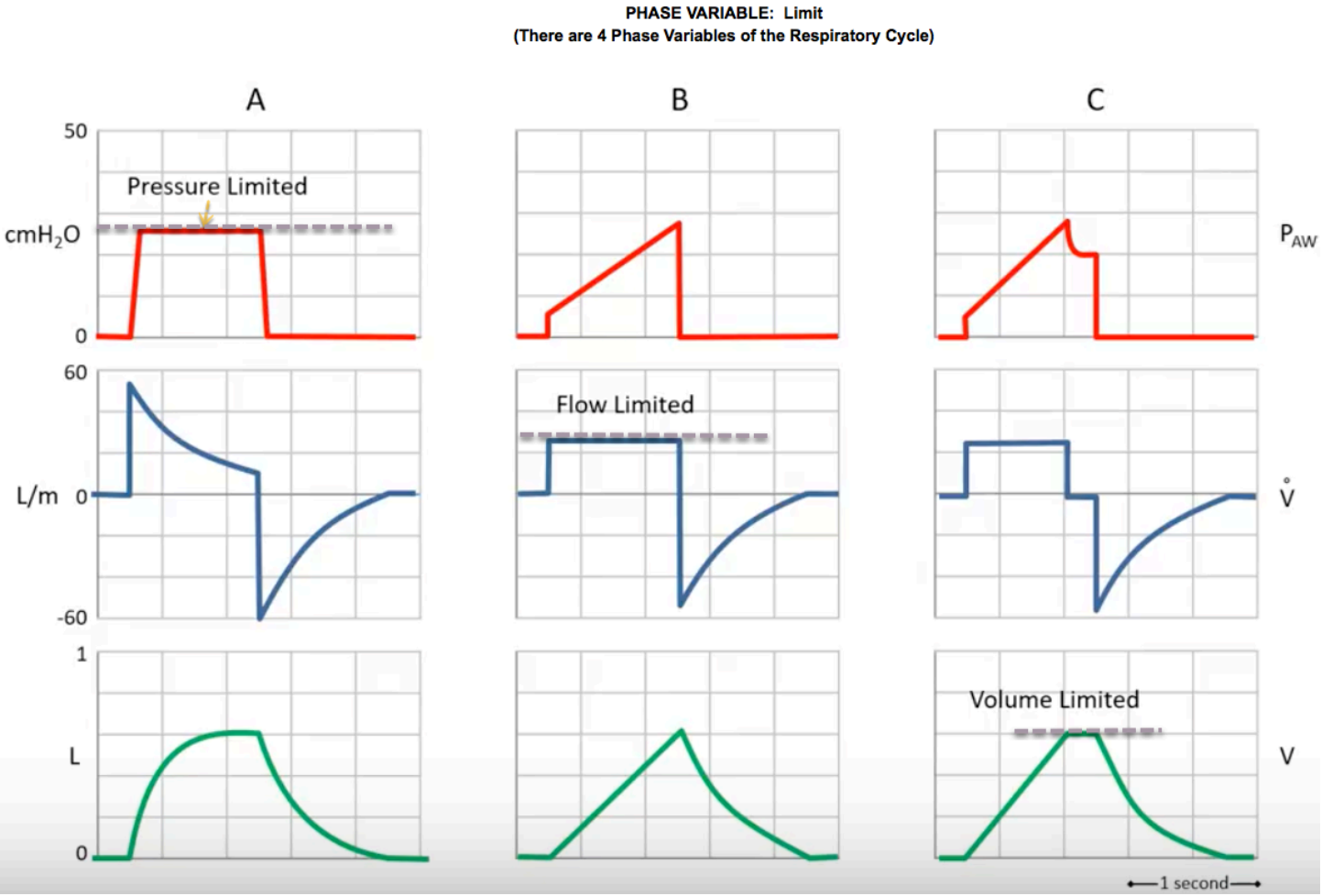
https://lifeinthefastlane.com/ccc/modes-of-ventilation/

https://www.youtube.com/watch?v=0jhaPL_Oveo

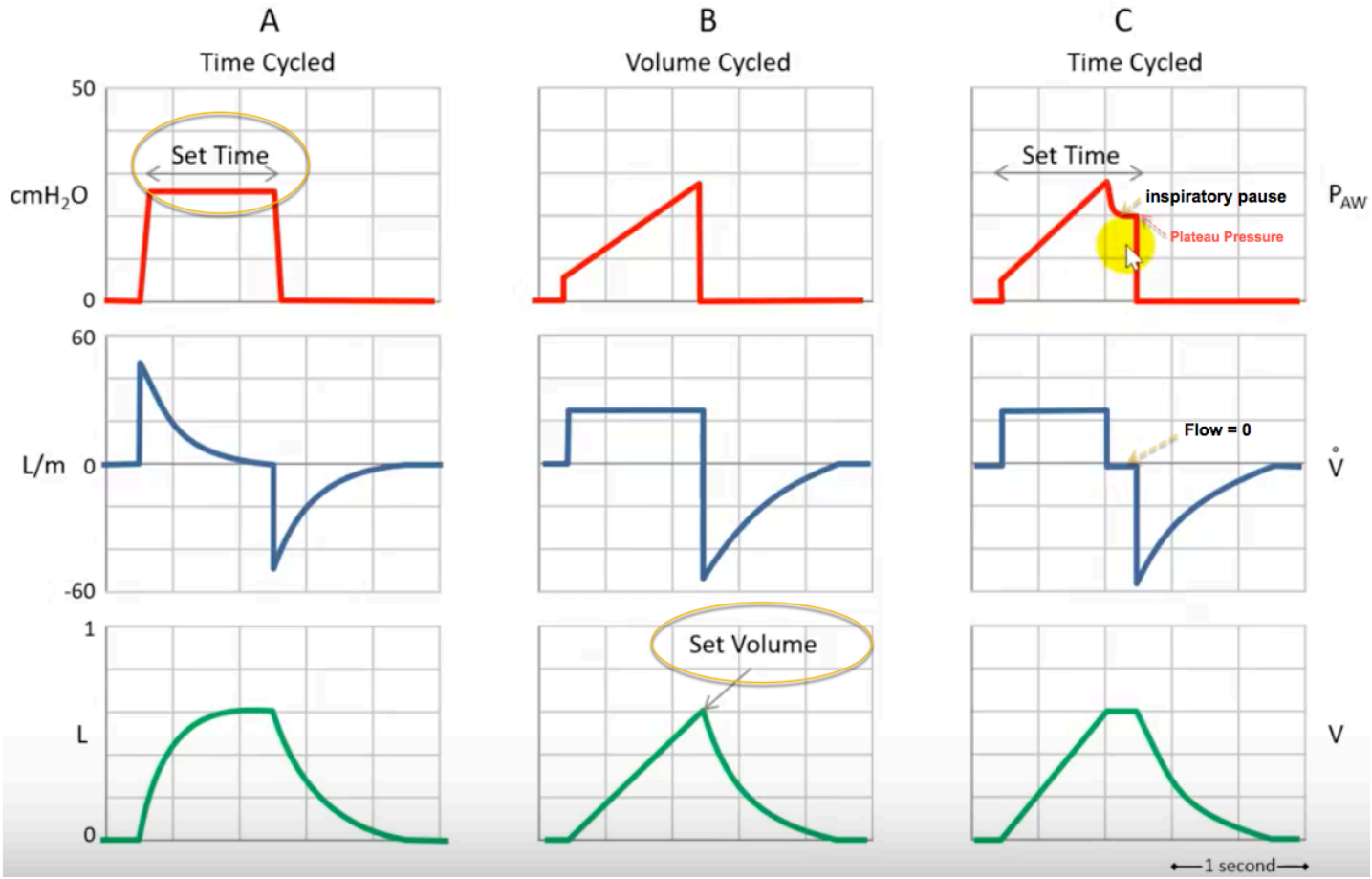


Note: if there's <u>decreased lung compliance</u>, the same pressure will be delivered by the vent, but the TV will be lower. Note also the decelerating ramp flow in Pressure Control ventilation.

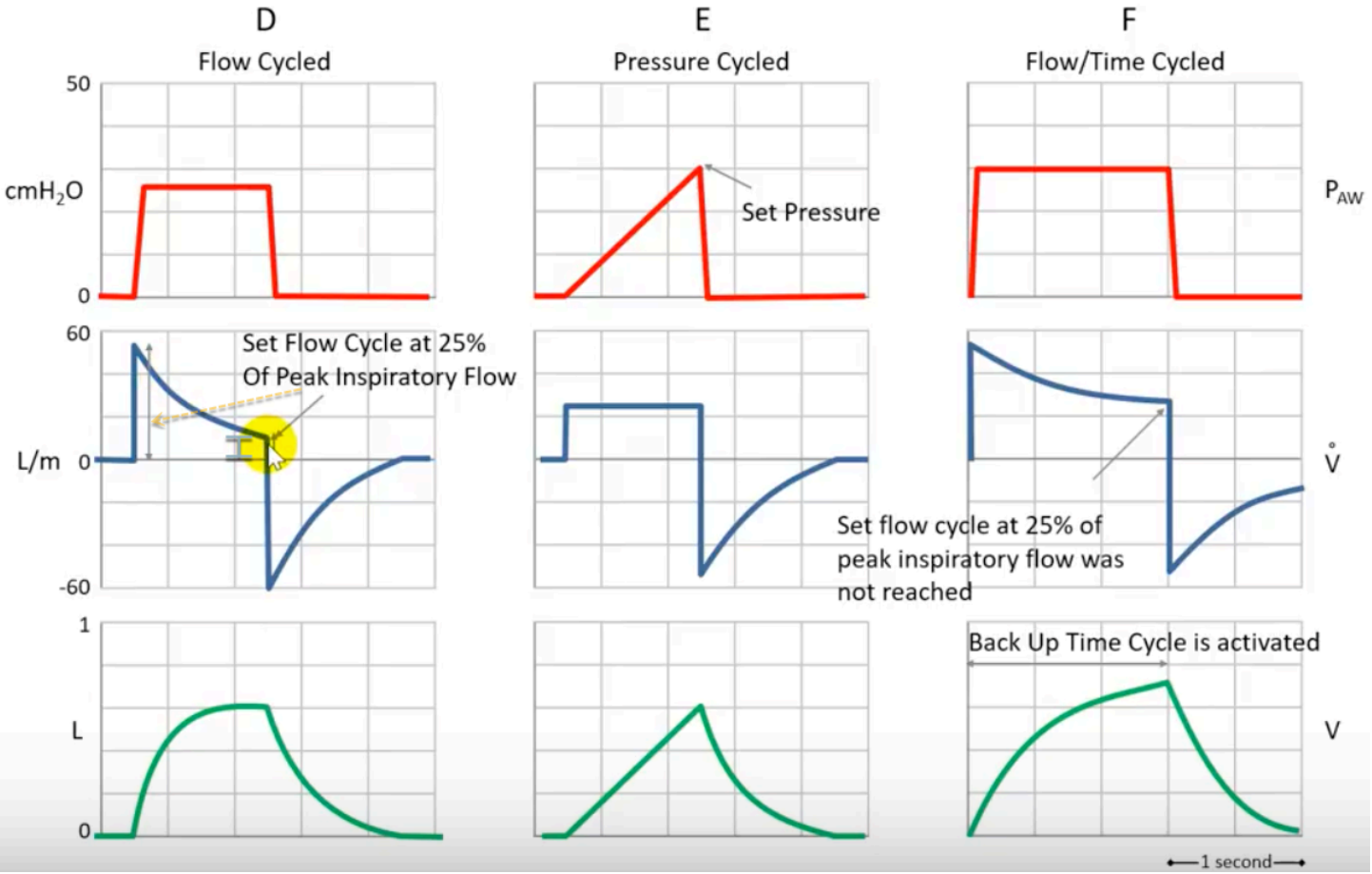

PHASE VARIABLE: Trigger
(There are 4 Phase Variables of the Respiratory Cycle)
Time-triggered
Flow-triggered
Pressure-triggered
If pt generates enough negative pressure during inhalation attempt, the vent will respond.
cmH2O
0
the vent starts inspiration after a set time has passed.
Pt starts inspiration. . .
Pressure Trigger Sensitivity
PAW
60
Flow Trigger Sensitivity
L/m
0
-60
The vent senses the flow generated by pt starting inspiration
V̊
Flow
1
L
0
V
30
cmH2O
0
-20
Pes
1 second


PHASE VARIABLE: Limit
(There are 4 Phase Variables of the Respiratory Cycle)
A
B
C
50
Pressure Limited
cmH2O
0
PAW
60
Flow Limited
L/m
0
-60
V̊
1
Volume Limited
L
V
0
1 second

PHASE VARIABLE: Cycle (how to transition from inspiration to expiration)
(There are 4 Phase Variables of the Respiratory Cycle)



PHASE VARIABLE: Cycle (how to transition from inspiration to expiration)
(There are 4 Phase Variables of the Respiratory Cycle)



Basic Mechanical Ventilation