EHC MICU Handbook

Your guide to sedatives, pressors and more

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Vasopressors & Inotropes

1. Overview

Vasopressors are agents that increase **mean arterial pressure (MAP)** primarily via **vasoconstriction ± increased cardiac contractility**. They are indicated in certain **shock states** when fluid resuscitation alone is inadequate. The goal is to increase blood pressure and thus perfusion.

Goal MAP: ≥65 mmHg (individualize for chronic hypertension (perhaps higher MAP goal), bedbound/limited mobility/cirrhosis (perhaps lower MAP goal ~60), or certain neurologic conditions (such as post-cardiac arrest with MAP goal 80-100 per AHA guidelines).

Inotropes are agents that increase **cardiac output (CO)** primarily via increased cardiac contractility. Vasodilation also occurs, resulting in decreased afterload, which may lead to decreased blood pressure. The primary goal is to increase cardiac output and thus perfusion.

CO (Perfusion or Cardiac Output) = HR x SV (stroke volume)
SV is influenced by: Pre-Load, Cardiac Contractility (Inotropy), and After-Load
MAP (BP) = CO x SVR (systemic vascular resistance)

2. Functional Categories

- **Inopressors** *(↑ contractility + ↑ vascular tone)*
  Norepinephrine (Levophed), Epinephrine, Dopamine
  Ideal for distributive shock (e.g., septic) and some cardiogenic shock.
- **Inotropes/Inodilators** *(↑ contractility + ↑ cardiac output + ↓ systemic vascular resistance)*
  Dobutamine, Milrinone
  Used in low-output heart failure or cardiogenic shock with high afterload (2nd-line after inopressor); risk of hypotension if used alone, so usually paired with Levophed
- **Pure Vasopressors** *(↑ SVR without significant cardiac stimulation)*
  Phenylephrine, Vasopressin
  Useful when tachyarrhythmias limit inopressor use or as adjuncts in septic shock
- **Other/Adjuncts**
  - **Methylene Blue:** Rescue agent for refractory vasodilatory shock; inhibits nitric oxide–mediated vasodilation. *Dose: 1–2 mg/kg IV over 20–60 min (avoid in G6PD deficiency), can also be used as continuous infusion*
  - **Midodrine:** Oral α1 agonist for chronic orthostatic hypotension or as an IV vasopressor weaning agent in the ICU. *Dose: 5-30mg q8*

3. Common Indications

- **Septic shock** – 1st-line: norepinephrine
- **Cardiogenic shock** – norepinephrine or epinephrine; add inotropes (dobutamine, milrinone) if perfusion (cardiac output) remains low; in bradycardic shock, Epi preferred
- **Obstructive shock** – treat cause + temporary pressor support
- **Distributive shock** (non-septic: anaphylaxis, neurogenic) – choice depends on etiology

4. Vasopressor Summary Table

Agent (Type)	Receptor Profile	Primary Effects	Starting Dose*	Common Clinical Range**	Key Pearls	Main Adverse Effects
Norepinephrine *(Inopressor – catecholamine)*	α1 > β1	↑ SVR ↑ MAP mild ↑ CO	1 mcg/min (0.05 mcg/kg/min)	1-30 mcg/min (0.05–1 mcg/kg/min)	1st-line in septic shock; less tachycardia than Epi	Arrhythmias, ischemia
Epinephrine *(Inopressor – catecholamine)*	β1 > α1	↑ SVR ↑ MAP ↑ HR ↑ CO	1 mcg/min	1-40 mcg/min	Adjunct in septic shock; Bradycardia; Anaphylaxis	Arrhythmias, ↑ lactate
Dopamine *(Inopressor – dose-dependent)*	DA > β1 > α1	Greater SVR/HR at higher doses	1 mcg/kg/min	1–20 mcg/kg/min	Not favored; ↑ mortality	Arrhythmias
Dobutamine *(Inodilator – catecholamine)*	β1 > β2 > α1	mild ↓ SVR mild ↓ PVR (-) or ↓ MAP ↑ HR ↑ CO	2 mcg/kg/min	2–20 mcg/kg/min	Cardiogenic shock with low CO; often with NE	Arrhythmias, hypotension
Milrinone *(Inodilator – PDE-3 inhibitor)*	PDE-3 inhibitor	↓ SVR ↓ PVR (-) or ↓ MAP ↑ HR ↑ CO	0.125 mcg/kg/min	0.125–0.75 mcg/kg/min	Cardiogenic shock with low CO; RV failure/pHTN	Hypotension, arrhythmias
Phenylephrine *(Pure vasopressor – α-agonist)*	Pure α1	↑ SVR ↑ PVR ↑ MAP ↓ HR Variable CO	40 mcg/min	20-400 mcg/min	Good if very tachycardic (AFib w/ RVR); avoid in low CO	Reflex bradycardia, ischemia
Vasopressin *(Pure vasopressor – non-catecholamine)*	V1	↑ SVR ↓ PVR	0.04 units/min (Fixed)	0.04 units/min (Fixed)	Add-on to reduce NE dose; RV failure/pHTN	Ischemia (digits, gut), ↓ Na
Methylene Blue *(Rescue adjunct)*	NO synthase & guanylate cyclase inhibitor	↓ Vasodilation	1–2 mg/kg IV bolus over 15 min	0.25-2 mg/kg/hr infusion	Refractory vasoplegia, BB or Ca-CB overdose	Serotonin syndrome, hemolysis in G6PD deficiency
Midodrine *(Oral vasopressor adjunct)*	α1	↑ SVR	5-10 mg PO q8h	2.5–40 mg PO q8h	Used for pressor weaning or chronic OH	Supine hypertension, piloerection

* **Starting Dose** = typical initial infusion; may start lower in frail patients or borderline MAP.
** **Common Clinical Range** = frequently used ICU range; higher doses in refractory cases.

SVR: Systemic Vascular Resistance **PVR:** Pulmonary Vascular Resistance
pHTN: Pulmonary Hypertension **OH:** Orthostatic Hypotension **NE:** Norepinephrine

- **mcg/kg/min = mcg/min ÷ weight (kg)**
- **mcg/min = mcg/kg/min × weight (kg)**
- Vasopressin: fixed-dose for shock; add after NE ≥ 0.25 mcg/kg/min (15 mcg/min)
- All can be given peripherally (except Vasopressin, as no antidote if tissue extravasation); transition to central access if prolonged and/or high-dosing; IO access is central (with plan to remove within 24-hrs)
    - If peripheral, give through largest and most proximal IV (avoid in hand/wrist/foot)
    - Antidote for extravasation: Phentolamine subcutaneously

5. Choosing a Vasopressor

1. **Hypotensive after adequate volume resuscitation?**
2. **Determine shock type**:
    - **Septic/distributive:** Norepinephrine → ± Vasopressin → ± Epinephrine *(Phenylephrine if tachyarrhythmic and high NE requirement)*
    - **Cardiogenic**: Norepinephrine ± Dobutamine/Milrinone
    - **Cardiogenic (RV Failure)**: Norepinephrine/Vasopressin ± Dobutamine/Milrinone *(Phenylephrine avoided given increase in PVR)*
    - **Obstructive:** Treat cause + Norepinephrine/Epi
    - **Anaphylaxis:** Epinephrine bolus (IM) ± IV infusion
3. **Reassess perfusion frequently** – MAP, urine output, lactate, cap refill, mentation

6. Practical Tips for Residents

- Correct **hypovolemia** first before vasopressors
- Titrate q5–15 min to avoid overshooting MAP and taper slowly to avoid rebound shock
- Dobutamine and Milrinone are <u>not</u> titrated to MAP, but to <u>Perfusion</u>
    - Should be <u>Continuous (not titratable)</u> on Epic with dosing changes made by you, not the nurse
    - One of best ways to assess perfusion is to assess for increase in Cardiac Output with POCUS (VTI estimate which correlates with Stroke Volume)

7. Key Adverse Effects

- **Arrhythmias** – especially Dopamine, Epinephrine, Dobutamine
- **Ischemia** – digits, mesenteric, skin
- **Lactate rise** – epinephrine (not always due to hypoperfusion)

8. Quick-Reference Receptor Table

Receptor	Location	Effect When Stimulated	Example Drugs
α1	Vascular smooth muscle	Vasoconstriction (↑ SVR)	NE, Phenylephrine, Epi
β1	Heart	↑ HR, ↑ contractility	Epi, Dobutamine, Dopamine
β2	Vascular & bronchial smooth muscle	Vasodilation, bronchodilation	Epi, Dobutamine
V1	Vascular smooth muscle	Vasoconstriction	Vasopressin

Improving ICU Outcomes

The **ICU Liberation Bundle (ABCDEF bundle)** was created by the Society of Critical Care Medicine (SCCM) to help implement the key elements of the **2018 Pain, Agitation/Sedation, Delirium, Immobility, and Sleep Disruption (PADIS)** guidelines. Compliance has been shown to decrease rates of hospital death, reduce delirium and coma rates, and reduce ICU re-admissions.

Assess, Prevent and Manage Pain

- Routine pain assessment using validated tools, prioritize pain before sedation

Both SATs and SBTs

- Daily interruption of sedation and assessment for readiness to wean and extubate

Choice of Sedation

- Preference for analgesia-first or analgesia-based sedation, minimize benzo's, and target light sedation

Delirium: Assess, Prevent and Manage

- Routine delirium monitoring with validated tools and implementing both non-pharmacological and pharmacological prevention strategies

Early Mobility and Exercise

- Prevent ICU-acquired weakness

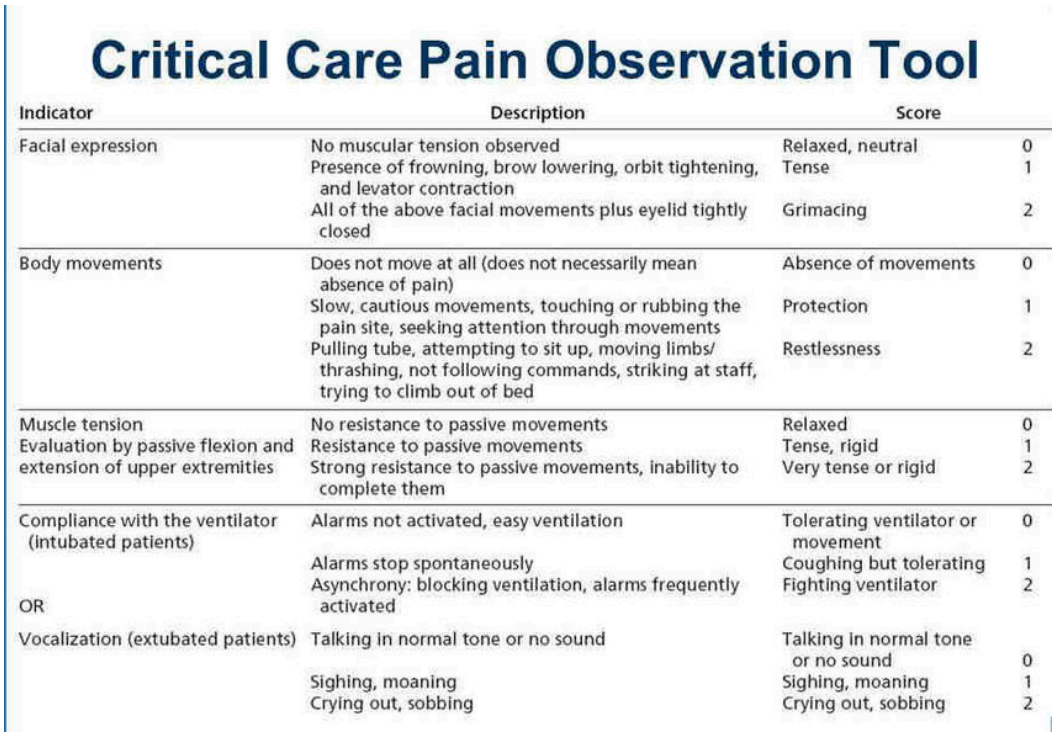
Family Engagement and Empowerment

- Actively involving family in care and decision-making

Analgo-Sedation and Delirium

1. **Analgesia** is the management or relief of pain/discomfort, without necessarily affecting consciousness/awareness. **Sedation** is a drug-induced depression of consciousness, ranging from minimal sedation (anxiolysis) to deep sedation and general anesthesia, with the goal of reducing anxiety, agitation, and/or awareness. Excessive sedation leds to higher ICU length of stay, days on mechanical ventilation, and higher delirium rates. The ideal level of sedation is one that achieves a calmed patient and able to follow commands; however, this will vary tremendously depending on the illness severity of the patient (severe ARDS patients will often require deep sedation to achieve ventilator synchrony).

2. The **Critical-Care Pain Observation Tool (CPOT)** and the **Behavioral Pain Scale (BPS)** are the two most valid and reliable pain scores for ICU patients who cannot communicate. They are assessed routinely by nursing. The CPOT is more commonly used in our MICU.
    - Ranges from 0 (no pain) to 8 (extreme pain). The goal is ≤ 2.



Critical Care Pain Observation Tool

Indicator	Description	Score	
Facial expression	No muscular tension observed	Relaxed, neutral	0
	Presence of frowning, brow lowering, orbit tightening, and levator contraction	Tense	1
	All of the above facial movements plus eyelid tightly closed	Grimacing	2
Body movements	Does not move at all (does not necessarily mean absence of pain)	Absence of movements	0
	Slow, cautious movements, touching or rubbing the pain site, seeking attention through movements	Protection	1
	Pulling tube, attempting to sit up, moving limbs/ thrashing, not following commands, striking at staff, trying to climb out of bed	Restlessness	2
Muscle tension Evaluation by passive flexion and extension of upper extremities	No resistance to passive movements	Relaxed	0
	Resistance to passive movements	Tense, rigid	1
	Strong resistance to passive movements, inability to complete them	Very tense or rigid	2
Compliance with the ventilator (intubated patients)	Alarms not activated, easy ventilation	Tolerating ventilator or movement	0
	Alarms stop spontaneously	Coughing but tolerating	1
	Asynchrony: blocking ventilation, alarms frequently activated	Fighting ventilator	2
OR			
Vocalization (extubated patients)	Talking in normal tone or no sound	Talking in normal tone or no sound	0
	Sighing, moaning	Sighing, moaning	1
	Crying out, sobbing	Crying out, sobbing	2

3. The **Richmond Agitation-Sedation Scale (RASS)** and the **Riker Sedation-Agitation Scale (SAS)** are the two most widely validated sedation scoring systems for ICU patients. The RASS is more commonly used.
    - RASS scores range from +4 (combative) to -5 (unarousable)
        - The ‘negative’ scores are the ones to commit to memory
        - Typical RASS goals for patients are 0 to -2
    - SAS scores range from 7 (dangerously combative) to 1 (unarousable)

Richmond Agitation and Sedation Scale (RASS)		
+4	Combative	violent, immediate danger to staff
+3	Very Agitated	Pulls or removes tube(s) or catheter(s); aggressive
+2	Agitated	Frequent non-purposeful movement, fights ventilator
+1	Restless	Anxious, apprehensive but movements not aggressive or vigorous
0	Alert & calm	
-1	Drowsy	Not fully alert, but has sustained awakening to *voice* (eye opening & contact ≥ 10 sec)
-2	Light sedation	Briefly awakens to *voice* (eye opening & contact < 10 sec)
-3	Moderate sedation	Movement or eye-opening to *voice* (but no eye contact)
-4	Deep sedation	No response to voice, but movement or eye opening to *physical* stimulation
-5	Unarousable	No response to *voice or physical* stimulation

4. **Delirium** is an acute change in attention and awareness that develops over hours to days, fluctuates in severity, and is not explained by a pre-existing neurocognitive disorder. It can be hyperactive, hypoactive (most frequent), or mixed, and affects up to 50% of all ICU patients (higher in intubated patients). This is associated with increased morbidity, mortality, and longer length of stays.

5. Daily screening for delirium is recommended in all ICU patients using validated tools, most commonly the **Confusion Assessment Method for the ICU (CAM-ICU)**, and can be used on intubated patients. Patients should not be sedated and have a RASS goal of 0 for highest reliability.

The patient is CAM-ICU positive (patient has delirium) if:

- RASS ≥ -3, AND
- Acute onset change in mental status or fluctuating course in mental status, AND
- >2 errors in letters attention test, AND
- Either RASS is not 0, OR combined number of errors to questions and commands >1

6. Delirium is best identified early with pro-active measures taken to prevent its incidence with a focus on reducing modifiable risk factors, improving cognition, and optimizing sleep, mobility, hearing, and vision. Specific interventions include reorientation, cognitive stimulation, use of clocks, minimizing light and noise to improve sleep, reducing sedation, early mobilization, and enabling use of hearing aids/glasses. **The use of antipsychotics do not reduce the duration of delirium or improve outcomes and should be reserved for severe agitation**. The use of Dexmedetomidine (Precedex) has shown some promise in reducing the incidence of delirium. There is even less evidence for the pharmacological treatment of hypoactive delirium.

7. Common Analgesics in the ICU

Drug	Mechanism	Dosing, PK/PD	Key Pearls	Adverse Effects
Fentanyl	Mu opioid receptor agonist	*Bolus:* 25mcg-100mcg q30-q60min *Infusion:* 25 - 200 mcg/hr Onset: 1-2 min Half-Life: Depends on duration of infusion (<1hr to up to 20hrs) Metabolism: Liver	Most common, Rapid onset with no histamine release, Good in ESRD patients, Hypotension should not occur with infusions (only boluses), Highly lipophilic	Respiratory depression, Constipation, Delirium, Serotonin Syndrome, Hyperalgesia, Tolerance, Withdrawal if prolonged, Chest wall rigidity (rare)
Hydromorphone (Dilaudid)	Mu opioid receptor agonist	*Bolus:* 0.4mg - 1mg q1-2 hrs *Infusion:* 0.4mg/hr - 3mg/hr Onset: ~5 min Half-Life: 2-3 hrs Metabolism: Liver	Rarely used as infusions (usually comfort care), Minimal histamine release, Minimal drug interactions	Respiratory depression, Constipation, Delirium, Serotonin Syndrome, Hyperalgesia, Tolerance, Withdrawal if prolonged, Caution in ESRD pts
Morphine	Mu opioid receptor agonist	*Bolus:* 2-10mg load, then 4-8mg q4 Onset: ~5 min Half-Life: 2-5 hrs Metabolism: Liver	Rarely used as infusions (usually comfort care), Higher histamine release	Caution in ESRD pts, Hypotension (may be attributed to histamine release)
Methadone	Mu opioid receptor agonist	PO: 10mg q8-12 Onset: 30-60min (PO) Duration: 4-8hrs Metabolism: Liver	Usually used to mitigate opioid withdrawal, Long & variable half-life	Respiratory depression, Constipation, Delirium, QTc prolongation
Ketamine	NMDA Antagonist	*Bolus:* 0.5mg/kg - 2mg/kg *Infusion:* 0.1 mg/kg/hr - 5 mg/kg/hr Onset: ~1min Half-life: 2-5 hrs Metabolism: Liver	Analgesia: 0.1 - 0.5mg/kg/hr Sedation (dissociative): 1 - 5mg/kg/hr Sedative, analgesic, anti-epileptic, preserves airway, bronchodilator, Opioid-sparing	Hypertension, tachycardia, Bronchorrhea, *Avoid doses between 0.5-1mg/kg/hr as ↑ risk unpleasant side effects* *Emergence reactions:* delirium, hallucinations (treat w/ Benzo's or Precedex)

50mcg Fentanyl ~ 5mg IV Morphine ~ 15mg PO Morphine ~ 0.75mg IV Dilaudid ~ 3.75mg PO Dilaudid ~ 10mg Oxycodone ~ 150mg Tramadol

8. Common Sedatives in the ICU

Drug	Mechanism	Dosing, PK/PD	Key Pearls	Adverse Effects
Propofol	GABA receptor agonist, with other effects on Glutamate and Cannabinoid receptors	Infusion: 5-50 mcg/kg/min Half-Life: 30-60 min after infusion Metabolism: Liver	Most common, Quick-on & quick-off Anti-epileptic at higher doses (60-80 mcg/kg/min)	Hypotension, Bradycardia, Hypertriglyceridemia, Propofol Related Infusion Syndrome (PRIS)
Dexmedetomidine (Precedex)	Central *alpha*-2 agonist	Infusion: 0.2-1.5 ug/kg/hr Half-life: ~2 hrs Metabolism: Liver	Infusion only, we do not bolus this Mostly used as weaning-aid for extubation or adjunct in EtoH withdrawal Minimal respiratory depression, may reduce delirium	Hypotension, Bradycardia, Dry mouth Avoid prolonged infusions (> 2 days) as risk of tolerance and withdrawal
Midazolam (Versed)	GABA receptor agonist	Infusion: 1-10 mg/hr (sedation), or 0.05-2 mg/kg/hr (seizures) Half-Life: 3-11 hrs (builds up) Metabolism: Liver	Rarely used, ↑ delirium Minimal hemodynamic changes Anti-epileptic (doses at different units)	Delirium, Respiratory depression, Risk of tolerance and withdrawal
Ketamine	NMDA Antagonist	*Bolus:* 0.5mg/kg - 2mg/kg *Infusion:* 0.1 mg/kg/hr - 5 mg/kg/hr Onset: ~1min Half-life: 2-5 hrs Metabolism: Liver	Analgesia: 0.1 - 0.5mg/kg/hr Sedation (dissociative): 1 - 5mg/kg/hr Sedative, analgesic, anti-epileptic, preserves airway, bronchodilator, Opioid-sparing	Hypertension, tachycardia, Bronchorrhea, *Avoid doses between 0.5-1mg/kg/hr as ↑ risk unpleasant side effects* *Emergence reactions:* delirium, hallucinations (treat w/ Benzo's or Precedex)

9. In most intubated patients, the go-to cocktail will usually be Propofol + Fentanyl (each titrated to different goals). Sedation is not mandatory in the ICU, pain control is more important. Different patients will require different strategies.

SATs/SBTs and Extubation

Spontaneous Awakening Trial (SAT)

A strategy used in the ICU where, for brief periods during the day (usually in the early morning), analgesia and sedation are decreased or stopped in order to assess breathing ability, neurological status and overall stability of the patient. This gives the medical team a better picture when a patient is ready to be extubated. This time also allows us to titrate the dose of sedative medications to make sure that we are using the lowest dose possible to achieve a target level of sedation.

Benefits of Spontaneous Awaking Trial (SAT):

- Allows us to perform a baseline neurological exam and to assess sedation needs
- Reduces ICU and hospital length of stay
- Decreases time of mechanical ventilation
- Decreases risk of ventilator-associated pneumonia (VAP)
- Reduces length of sedation exposure (linked to lower delirium/coma)
- Improves mortality (when combined with Spontaneous Breathing Trials)

Safe markers to attempt SAT:

- No severe agitation or active seizures
- Not on paralytics
- No severe hemodynamic or respiratory instability
- No active myocardial ischemia

Spontaneous Breathing Trial (SBT)

Similarly, an SBT is a ventilator weaning strategy whereby following appropriate sedation reduction/cessation (as above), the ventilator mode is switched to Pressure Support/CPAP (usually) where patients initiate their own breaths and the ventilator provides some level of support with each breath (typically PS 5, PEEP 5 // which corresponds to BiPAP 10/5). This support is merely meant to off-set the resistance of the endotracheal tube. This is performed in the early morning (either completed by RT or Fellow) and lasts ~30 minutes.

Safe markers to perform SBT (in addition to SAT criteria as above):

- Oxygen saturation > 88%
- FiO2 of < 50%
- PEEP $\leq$ 8 cm H2O
- Minimal pressors requirements

The patient is monitored during the SBT for agitation, tachypnea/bradypnea, hypoxemia, and/or hemodynamic instability. If a patient 'fails' an SBT, you must know the reason why. In most cases, the patient will be placed back on full ventilator support with restarting of sedatives (at 50% the prior dose) and the SBT re-trialed the next day. There is a lot of 'grey' zone in determining whether a patient can be attempted for extubation, despite not passing the SBT. For example, just being intubated while off sedation can cause a lot of agitation/anxiety, yet those patients may do well once extubated and is not a strict contraindication to not extubate. A 'pass' of SBT also does not mean the patient will do well after extubation (as it does not account for factors such as laryngeal edema or ability to manage secretions).

Cuff-Leak Test

This test helps predict the risk of post-extubation stridor due to laryngeal edema. Performing this test is recommended after the patient passes an SBT but prior to planned extubation in certain high-risk patients, including traumatic/prolonged intubations (>6 days), large endotracheal tube size (> 8), females, and/or prior recent extubations. The test has a high false-positive rate and should only be performed in those with high-risk features.

The test is performed by placing the patient on full vent support (volume-controlled) and the endotracheal tube is fully deflated. The difference between the delivered inspiratory tidal volume and the measured expiratory tidal volume is evaluated over several breaths. A cuff-leak is present if there is a significant difference between the two (typically >110 mL and/or > 10-15% of inspiratory tidal volume), indicating air passage around the tube and out the mouth (expiratory volume not being sensed by the ventilator), suggesting the absence of significant laryngeal edema. An audible gurgling sound is also indicative of a leak. In this case, having a leak is a good thing. A 'positive' cuff-leak test means that there is minimal or no leak, which is a bad thing, and places them at higher-risk of developing post-extubation airway obstruction (stridor).

A positive cuff-leak test should prompt the use of steroids prior to the extubation attempt. There are several proposed regimens, but a single dose of Solumedrol 40-60mg IVP may be all that is needed. The extubation can then be attempted as soon as 4-6 hours following this. A repeat cuff-leak test should not be done prior to the planned extubation in the event of an earlier positive test with steroids having been given 4-6 hours beforehand.

Extubation

Removal of the endotracheal tube can be considered if a patient passes the SAT/SBT and cuff-leak test (if indicated). A minimum time of 30-minutes during an SBT is all that is needed, and prolonged SBTs are not recommended. Be sure to assess for secretion burden with in-line suction, as excessive secretions are not accounted for during an SBT. An ideal scenario is a strong cough in response to suctioning, with the ability to follow commands, move all extremities, and lift up the head off of the bed (best physical measure of extubation readiness). If a patient is deemed ready for extubation by the entire team, respiratory therapy and nursing is informed and an Extubate order is placed on Epic. During extubation, a member of the medical team should be present in the room along with nursing and respiratory therapy. Following extubation, you should listen at the neck for stridor and the chest for good air entry.

The patient may be extubated to simple face-mask or nasal cannula, or to high-flow nasal cannula (HFNC), or to BiPAP. Patients at higher-risk of extubation failure include those with hypercapnea, COPD, CHF, obesity, advanced age, and/or prolonged intubation. These patients should be extubated directly to BiPAP (preferred if tolerated) and/or HFNC, and should be maintained for 24-48 hours to best reduce the reintubation risk. If HFNC is used, the flow rate should be kept high (50 or 60 L/min). A combination of BiPAP and HFNC can be used if patients are having issues tolerating prolonged BiPAP.

Extubation failure is most commonly defined as the need for re-intubation within 48-72 hours following extubation. There is never a 100% guarantee of successful extubation despite 'passing' all of the above criteria, and a re-intubation rate of ~15% is generally considered acceptable.

Examining the Sedated Patient

A physical exam on a sedated patient can present unique challenges as compared to our typical bedside evaluation of an awake patient on general medicine floors. Sedated patients are unable to verbalize symptoms, interact, or localize pain. This has required the creation of practical tools to help us assess each patient systematically. Although challenging, a proper physical exam is important for early detection of clinical deterioration, for guidance in sedation/analgesia, and for response to therapy.

Physical Exam (System-based approach)

General:

- Take note of general appearance, ventilator settings and waveforms (are they triggering the vent?), vitals monitor, all drips/infusions (know doses of sedatives/analgesics/vasopressors), verify arterial-line monitor is at mid-chest level
- Take note of all nasogastric tubes, IVs, central lines, arterial lines, foleys, drains, etc… Make sure they are clean and in place; any signs of phlebitis?
- If friends/family members in the room, can ask them to quickly step-out while you complete the exam. Draw the curtains closed every time you examine someone.
- Depending on the patient, may require POCUS. Do this during your pre-rounds and ask fellow for assistance if needed.

Neurological: Perhaps the most important

- Degree of sedation (RASS level), assess if they can follow commands and move all extremities, ask ‘yes’ and ‘no’ questions if awake (in pain?, etc…), speak to them and explain what is happening/orient them every day (if able)
- If not awake or responsive, check all extremities for withdrawal to pain (pinching at upper arms/legs, nailbed pressure, rib notching, supraorbital pressure, etc…), pupillary and corneal reflexes
- If on paralytics, know what the Train-of-Four (TOF) is (nursing checks this regularly)
- Can check reflexes and signs of ankle clonus (if applicable)
- If intubated or with tracheostomy, always use in-line suction to assess for gag reflex, strength of cough, and to assess quantity/quality of secretions
    - Use 2-hands; one hand always holds onto the ET Tube, while the other hand advances the in-line suction all the way in and applies suction on the way out

HEENT:

- Pupillary and corneal reflexes as above
- Inspection of nares and oropharynx for any bleeding, ulcerations, or lesions
- Facial asymmetry

Respiratory/Ventilation:

- Take note of ventilator settings, waveforms, and if they are triggering the vent
- Take note at the depth of the ET Tube (numbers on the tube, usually in reference to the lips or upper/lower teeth), to ensure tube migration didn't occur. Check that the ET Tube is securely attached to the tube holder and on their face and not loose/falling off.
- Check the ventilator tubing for lots of water build-up or condensation as this can cause 'fluttering' noise and a 'jittery' appearance on the vent wave-form. If present, try to suction it up with the in-line suction. May have to lift up and bend the tubing so that the water deposits at the ET Tube where your suction can reach it
- Look for signs of respiratory distress, use of accessory muscles, and pattern of breathing (regular/irregular)
- **Auscultation**:
    - Mechanical breath sounds (if intubated; from ventilator air flow)
    - Clear vs "harsh" sounds (reflects the tube or circuit turbulence)
    - Wheezing, Rhonchi, Crackles
    - Diminished breath sounds (Consolidation, Atelectasis, Effusions)
    - Absent breath sounds (Pneumothorax, Unilateral mainstem tube placement)
- Use in-line suction to assess secretions and cough/gag reflex
- Listen for an air leak and look at the ventilator for signs of air leak (more information under Ventilation chapter)

Cardiovascular:

- Look for any obvious signs of elevated jugular venous pressure
- **Auscultation:** loud heart sounds, new murmurs, gallops, rubs, etc…
- **Peripheral perfusion:** feel all extremities (temperature), check pulses (dorsalis pedis and radial are easy to assess), cyanosis (especially at digits), capillary refill (great marker of perfusion), mottling (begins at knees and progresses outwards as gets worse)
- Lower extremity and sacral edema (will present before leg edema)

Abdominal:

- Look for any ecchymoses (intra-abdominal hemorrhage) or distention
- **Auscultation:** For example, absence of bowel sounds could indicate severe intraabdominal conditions like obstruction, paralytic ileus, severe electrolyte abnormalities or medication-induced
- Tap for any tympany
- Palpate for rigidity or involuntary guarding, liver and spleen enlargement
- Feel suprapubic region for evidence of bladder distension/urinary retention

Posterior:

- Not done regularly but can be performed when the patient is turned
- Check for any ulcers, sacral edema, or ecchymoses (retroperitoneal hemorrhage)

Mechanical Ventilation: Part I

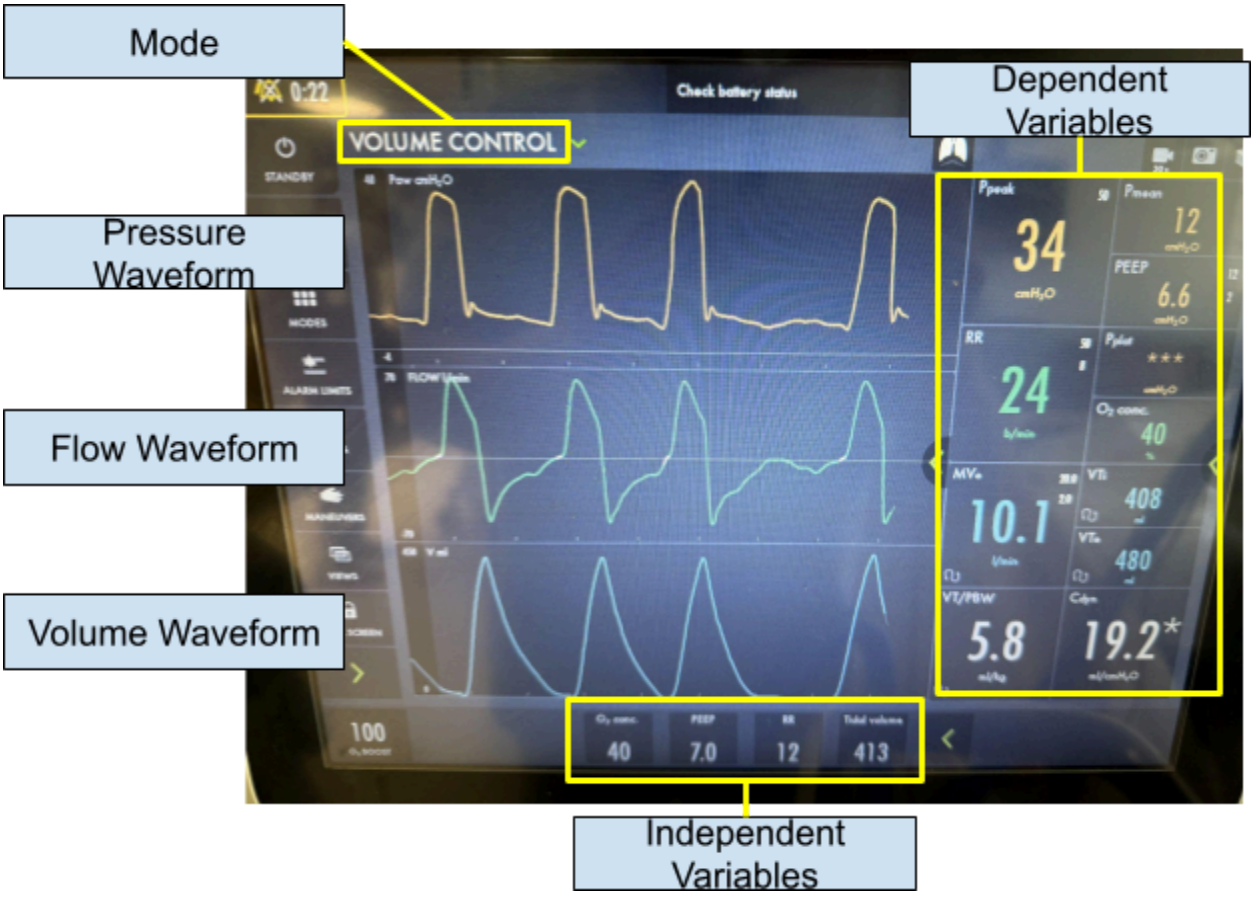
Mechanical ventilation is a cornerstone of ICU care, and you'll quickly realize that many of your patients will be intubated and ventilated. This section will walk you through the basics of ventilator modes, settings, and how to approach common scenarios you'll encounter.

In general, on the ventilator, **oxygenation** is controlled by FiO2 and PEEP. **Ventilation** (CO_2 release) is controlled by Tidal Volume (TV) and Respiratory Rate (RR). **Minute Ventilation** is the result of TV x RR.

In mechanical ventilation, you can choose to control either **volume** or **pressure**, but not both simultaneously. In **volume-controlled** modes, the ventilator guarantees a set tidal volume (*independent* variable) with each breath, while the pressure (*dependent* variable) required to deliver that volume will vary depending on the patient's lung compliance and resistance. In contrast, **pressure-controlled** modes guarantee a set inspiratory pressure (*independent* variable), but the tidal volume delivered (*dependent* variable) will fluctuate based on the patient's lung mechanics.

Additionally, ventilator modes can be broadly categorized as **control** or **support** modes. In **control** modes (Volume Control or Pressure Control), the ventilator is in charge—it delivers breaths at a set rate and pattern regardless of patient effort. In **support** modes (Pressure Support), all breaths are patient-triggered and the ventilator simply assists each breath with a set inspiratory pressure, allowing the patient to control their own respiratory rate and effort.

This is an example of a typical ventilator screen:



Definitions for some of the terms that you need to know:

- **Tidal Volume (VT):** Amount of air delivered with each breath (mL)
    - In the dependent variable box above, this is also split into VTi (inspiratory) and VTe (expiratory). This is where you can see if there is a big discrepancy in between the two (concern for an air-leak), as they should roughly be equal (within ~50mL or so)
- **Respiratory Rate (RR)**: Number of breaths per minute. On the vent, the set RR is the *minimum* rate (the vent will not allow any breaths lower than your set number), but the patient can breath at any rate faster (you cannot make them breath slower if they are taking their own breaths).
- **Fraction of Inspired Oxygen (FiO_2)**: Oxygen concentration delivered to the patient (%)
- **Positive End-Expiratory Pressure (PEEP)**: Pressure maintained in lungs at end of expiration to keep alveoli open
- **Peak Inspiratory Pressure (PIP)**: Highest pressure during inspiration. Goal < 30.
- **Plateau Pressure (Pplat)**: Pressure measured during an inspiratory pause; reflects alveolar pressure (eliminates resistance). Goal < 30.
- **Driving Pressure (DP):** Distending pressure applied to the alveoli during a breath; estimate of strain. The most important to note. DP = Pplat - PEEP. Goal < 15.
- **Compliance**: How easily the lungs expand (change in volume per unit pressure)
    - Essentially, Tidal Volume / Driving Pressure
    - Decreased (worsened) compliance can occur from a lung problem (edema, fibrosis, pneumonia, pleural effusion) or a non-lung problem (chest wall rigidity, obesity, ascites, spinal deformities)
- **Minute Ventilation (VE)**: Total volume of air entering lungs per minute (tidal volume × respiratory rate)
- **I:E Ratio (Inspiratory:Expiratory Ratio)**: Relative duration of inspiration vs expiration
- **Trigger Sensitivity**: How much effort a patient must exert to initiate a ventilator breath
    - Can also be selected between Flow or Pressure-triggered

Common ventilator settings that need to be adjusted:

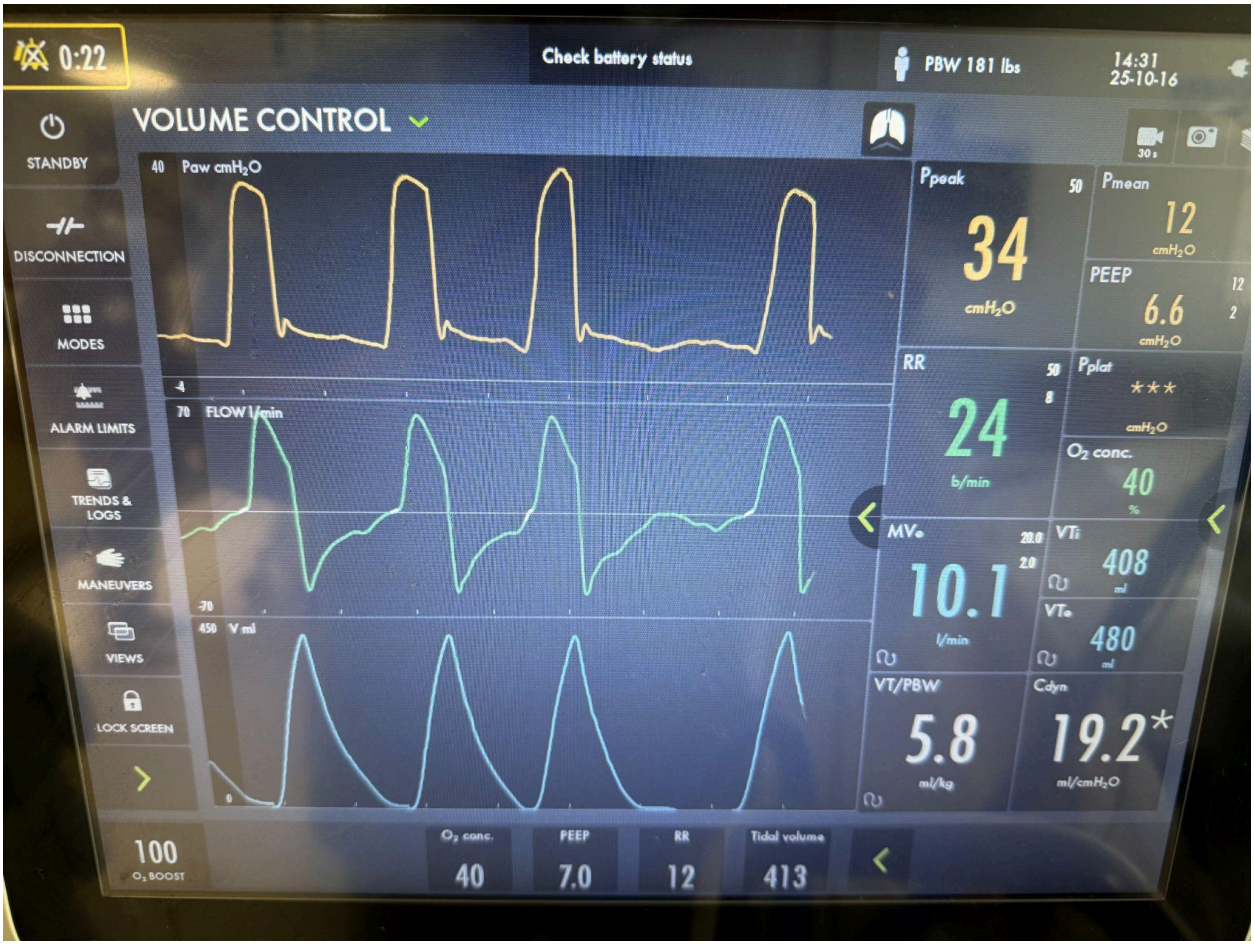
- **Tidal Volume (VT)**
    - Usually set between 6–8 mL/kg of ideal body weight (IBW, need height for calculation) to optimize ventilation and minimize lung injury
- **Respiratory Rate (RR)**
    - Start at 12-14 and adjust based on ventilation needs
- **Fraction of Inspired Oxygen (FiO_2)**
    - Adjust to target SpO_2 92–96%
- **Positive End-Expiratory Pressure (PEEP)**
    - Start at 5 and titrate based on oxygenation and lung mechanics

- Other settings such as inspiratory time, flow rate and trigger sensitivity can also be adjusted but less commonly

Next, we'll go over the most common ventilator modes. They are Volume-Control (VC), Pressure-Control (PC), Pressure-Support (PS), and Pressure-Regulated Volume Control (PRVC).

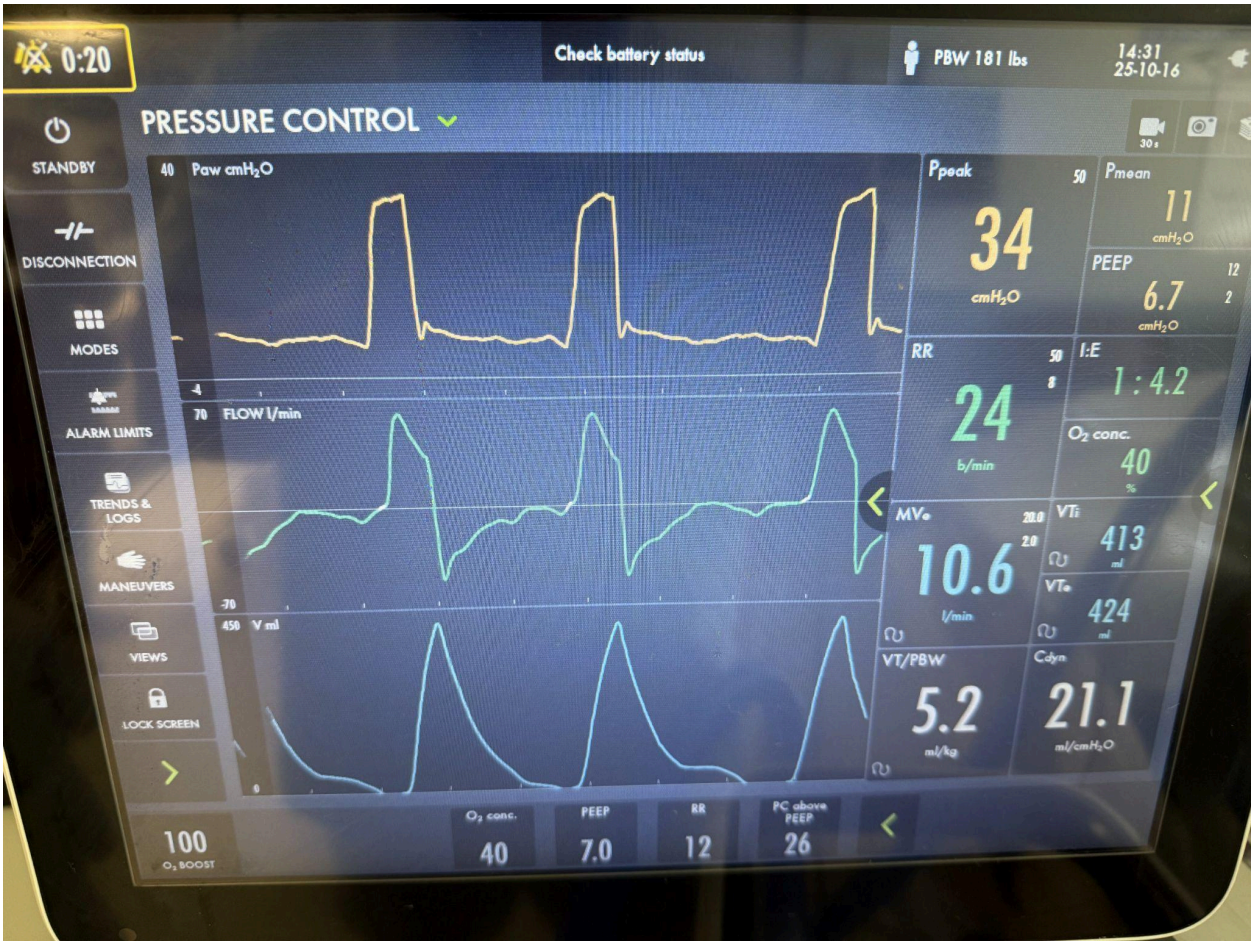
Volume Control (VC)

- **What is it:**
  - The most commonly used mode. The ventilator delivers a preset **tidal volume** with each breath, regardless of the pressure required. Note that the ventilator will have a pressure-limit alarm set as a safety mechanism and will not deliver any further volume if it requires a pressure above that set limit.
  - Pressure varies depending on lung compliance and resistance.
- **Pros:**
  - Precise control of ventilation and CO_2 removal
  - Easy to monitor lung compliance (via peak and plateau pressures)
  - Limits volutrauma (guarantees low tidal volume ventilation)
- **Cons:**
  - Risk of barotrauma if compliance worsens
- **Best suited for:**
  - Patients needing guaranteed minute ventilation (e.g., sedated or paralyzed)
  - ARDS with lung-protective ventilation



Pressure Control (PC)

- **What is it:**
  - The ventilator delivers breaths at a set **inspiratory pressure** for a set time.
  - Tidal volume varies depending on compliance and resistance.
- **Pros:**
  - Limits peak airway pressure (potentially reducing barotrauma)
  - Consistent inspiratory time and flow
- **Cons:**
  - Tidal volume is not guaranteed
  - May require more frequent adjustment and careful monitoring
- **Best suited for:**
  - May be more comfortable in certain patients
  - ARDS with lung-protective ventilation
  - Sedated or paralyzed patients at risk of high airway pressures



Pressure Support (PS)

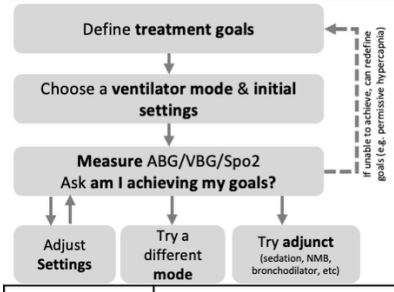
- **What is it:**
  - Patient-initiated mode, where each breath is assisted with a set **pressure** (PS above PEEP). Used during an SBT, also known as CPAP trial, for vent weaning and readiness to extubate assessment
  - No mandatory breaths. Note, there is an Apnea back-up where the ventilator will auto-switch to mandatory breaths when the ventilator doesn't sense a breath after a set number of seconds (can be manually increased up to 45 seconds)
- **Pros:**
  - Promotes comfort and synchrony
  - Encourages spontaneous effort and diaphragm activity
- **Cons:**
  - No guaranteed minute ventilation
  - Risk of fatigue or hypoventilation if support is insufficient
  - Risk for volutrauma if patient taking in very large tidal volumes
- **Best suited for:**
  - Patients breathing spontaneously
  - Weaning trials or lightly sedated, cooperative patients

Pressure-Regulated Volume Control (PRVC)

- **What is it:**
  - A hybrid mode combining features of volume control and pressure control. The ventilator delivers a **set target tidal volume**, but adjusts the **inspiratory pressure automatically** from breath to breath to achieve that volume with the **lowest possible pressure**.
  - A “better” version of VC
- **Pros:**
  - Guarantees a consistent tidal volume while limiting excessive airway pressures
  - Adapts to changes in lung compliance and resistance
  - Often more comfortable and synchronizes better than pure volume control
- **Cons:**
  - Rapid patient effort changes can confuse the ventilator’s pressure adjustment algorithm, causing variable tidal volumes
  - Potential for delayed response if lung mechanics change suddenly
  - May not be ideal for patients with high spontaneous drive (can lead to double triggering or breath stacking) as they want higher flow, which the vent is trying to limit
- **Best suited for:**
  - Any patient deemed for VC, with added benefit of adaptive pressure control
  - Stable respiratory drive patients (not those with air-hunger)

OVERVIEW OF VENTILATOR MODES by Nick Mark MD

ONE onepagericu.com @nickmmark Link to the most current version →



Goals for mechanical ventilation:

1. **Oxygenation** – support PaO2/SpO2
2. **Ventilation** – maintain pH
3. **Patient comfort** – vent synchrony, ↓ sedation
4. **Facilitate weaning** – minimize muscle loss, promote readiness to wean from support

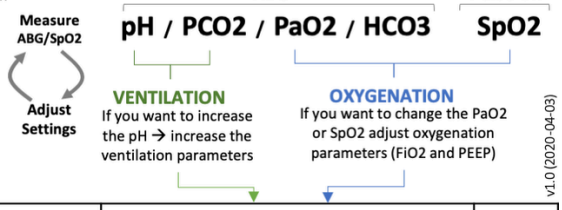
Ventilator Modes:

Fall into two broad categories: ***pressure*** and ***volume*** modes. *Each mode has three features*:

- Trigger (T) – what initiates a breath?
- Cycle (C) – what ends a breath?
- Limit (L) – what stops a breath early?

Each mode has **Pros** and **Cons** to consider.

Measurement and optimization:



v1.0 (2020-04-03)

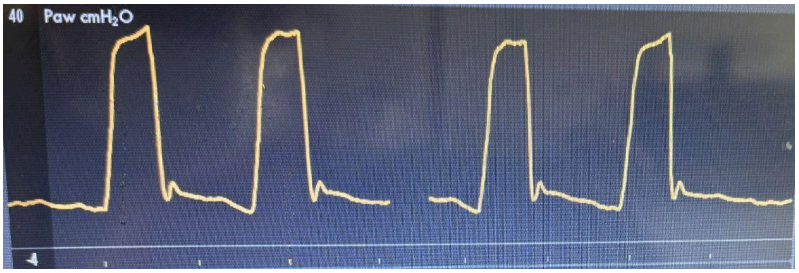
Mode	Description	Pros	Cons	Major settings / example	Monitor
VC Volume Control (a.k.a. assist control volume)	Every breath delivered (mandatory and patient triggered) is the same set **volume (TV)** T – time/pressure/flow, C – volume, L – volume	Good general-purpose mode; Ensures a minimum MV is achieved. Good mode for lung protective ventilation (LPV)	Requires you to monitor pressures to avoid barotrauma. (See my *OnePager* on ARDS for details.)	**RR, TV, PEEP, FIO2** **12 bpm, 450cc, +8, 60%** *(RR – respiratory rate, TV – tidal volume)*	Pressures (Ppeak, Pplat)
PC Pressure Control (a.k.a. assist control pressure)	Every breath delivered (mandatory & patient triggered) is a set **pressure (IP)** for a **set time (T_I)** T - time/pressure/flow, C – time, L - pressure	Good for limiting pressure; may be more comfortable for select patients. Also can be used for LPV (no difference in mortality)	Requires you to monitor volumes to avoid volutrauma or hypoventilation	**RR, IP, T_I, Risetime, PEEP, FIO2** **12 bpm, 25 cmH_2O, 0.9 sec, 0.15 sec, +8, 60%** *(IP – inspiratory pressure, T_I – inspiratory time)*	Volumes (TV, MV)
PRVC Pressure Regulated Volume Control (a.k.a. VC+, APV, Autoflow)	***Hybrid*** PC mode that dynamically changes inspiratory pressure to deliver a desired volume T - time/pressure/flow, C – volume, L - volume	Guarantees TV but delivers pressure-controlled breaths; (e.g. low risk of causing VILI), which potentially may be more comfortable for patients	In patients who are struggling (e.g. high WOB) this mode will provide ***less*** support	**RR, TV, T_I, Risetime, P_{max}, PEEP, FIO2** **12 bpm, 450cc, 0.9 sec, 0.15 sec, 30 cmH_2O, +8,60%** *(P_{max} – maximum pressure)*	Pressures & volumes
SIMV Synchronous Intermittent Mandatory Ventilation	Delivers mandatory breaths with a fixed volume but patient **can't** trigger (patient breaths are not the same as mandatory breaths); can use PS T – time , C – volume, L - volume	May be useful for patients with hiccups to avoid alkalemia	Seldom used; not effective for weaning; often found to be uncomfortable	**RR, TV, PEEP, FIO2** **12 bpm, 450 cc, +8, 60%**	Pressure (Ppeak Pplat)
PS Pressure Support	**All** breaths are patient initiated; ventilation determined solely by patient (no backup rate). T – pressure/flow, C – flow, L - pressure	Ideal weaning mode (used in SBTs and for prolonged periods); most comfortable because it allows patient to control ventilation	Does not guarantee a rate; need to monitor to ensure adequate ventilation	**PS, PEEP, FiO2** **+10, +5, 40%** *Note that **PS** is above **PEEP** so "Ten over Five" PIP = 15cmH2O*	Volumes (TV, MV)
APRV Airway Pressure Release Ventilation (a.k.a. Bi-Vent)	**Inverse ratio ventilation** (e.g. I time > E time) that allows patient to breath spontaneously; can combine w/ PS T – time, C – time, L - pressure	Great for ARDS patients who are spontaneously breathing (e.g. not on NMB); *may improve comfort & oxygenation (but no mortality benefit)*	Complex mode/settings; Risk of VILI if settings are done improperly; doesn't make sense if on NMB	**T_{High}, T_{Low}, P_{high}, P_{low}, FIO2** **5.5 sec, 0.5 sec, 25 cmH_2O, 0 cmH_2O, 60%** *($T_{High/low}$ – time high/low, $P_{High/low}$ – pressure high/low, also note that **Plow** is analogous to **PEEP**)*	Volumes & gas exchange PCO2 / EtCO2

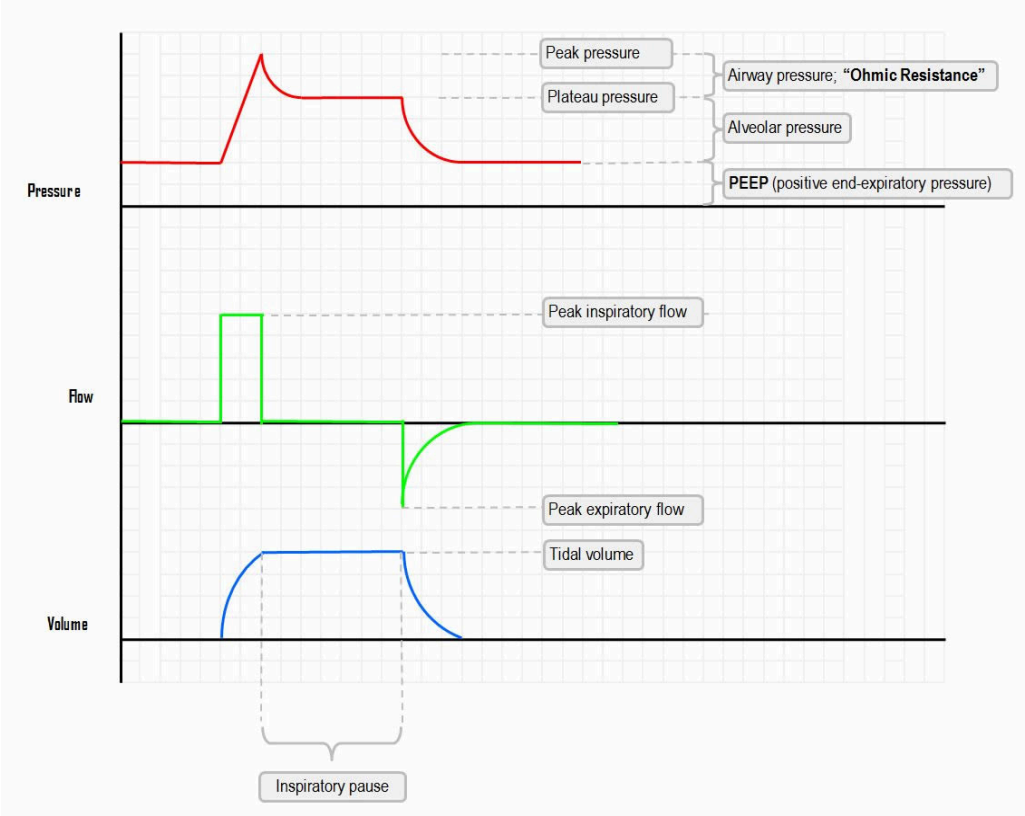
Mechanical Ventilation: Part II

Ventilator Waveforms: The Basics

Waveforms are one of the best tools to understand what's happening in the lungs and how the patient is interacting with the vent. The default view on the ventilator will show 3 waveforms (Pressure-Time, Flow-Time, and Volume-Time), from top to bottom in that order.

Pressure-Time Waveform



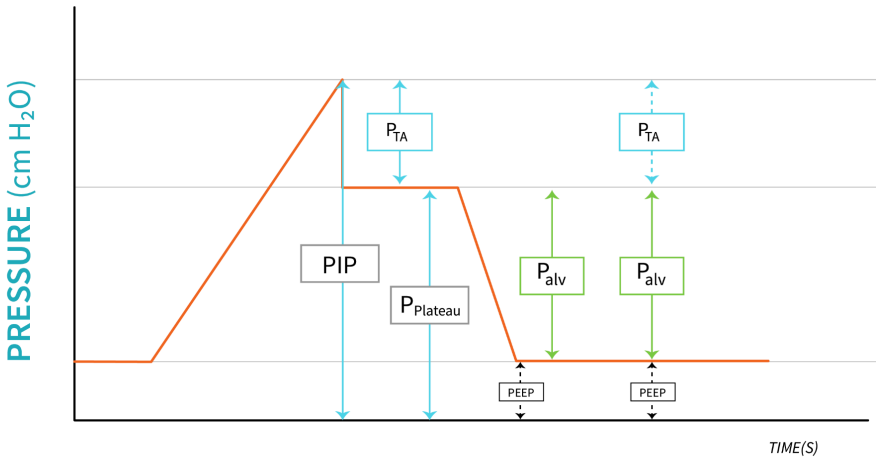


- Total (peak) pressure is a combination of Compliance (lung + chest wall) and Resistance.
- If Flow (Q) is 0, there can be no resistance ($R = \Delta P / Q$)
- An Inspiratory Hold/Pause will stop all Flow following inspiration (as graph shows above) and check a Pressure. This eliminates Resistance from the equation and indirectly reflects Compliance. This new pressure is now called the Plateau Pressure.
- An Inspiratory Hold/Pause must be manually pressed on the ventilator for a few seconds in order to obtain the waveform as above. As such, the Plateau pressure will only appear as you are pressing the button.

- The Trans-Airway Pressure (Pta) is the difference between the Peak and Plateau pressures and is usually 3-7.
    - A High Peak Pressure (>30) with a High Plateau Pressure (thus a small Pta) indicates that the alveolar pressure is also high, reflecting a Compliance problem and not a Resistance one (↑ pLateau = L for Lung = compLiance problem)
    - A High Peak Pressure (>30) with a Low Plateau Pressure (thus a high Pta) indicates a Resistance problem.
- See Troubleshooting section later on for more detailed information about Peak and Plateau pressures

Reminders…

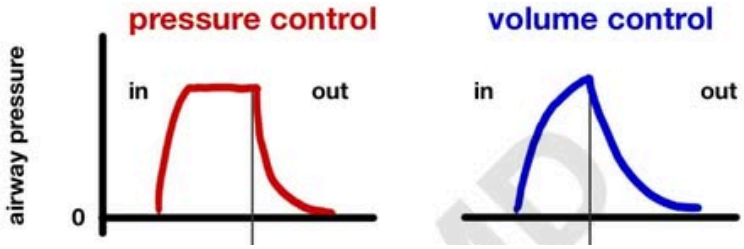
- **Positive End-Expiratory Pressure (PEEP)**: Pressure maintained in lungs at end of expiration to keep alveoli open
- **Peak Inspiratory Pressure (PIP)**: Highest pressure during inspiration. Goal < 30.
- **Plateau Pressure (Pplat)**: Pressure measured during an inspiratory pause; reflects alveolar pressure (eliminates resistance). Goal < 30.
- **Trans-Airway Pressure (Pta):** Reflects resistance. Typically between 3-7.
    - Pta = PIP - Pplat
- **Driving Pressure (DP) or Alveolar Pressure:** Distending pressure applied to the alveoli during a breath; estimate of alveolar strain. The most important to note.
    - DP = Pplat - PEEP. Goal < 15.



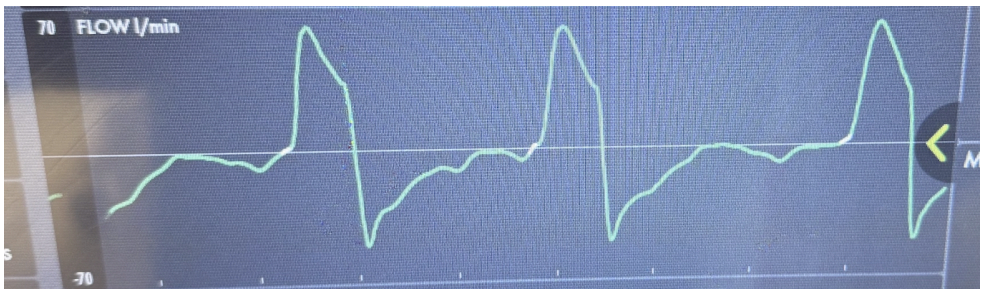
PIP: Peak Inspiratory Pressure **Pplateau:** Plateau Pressure **Pta**: Trans-airway Pressure

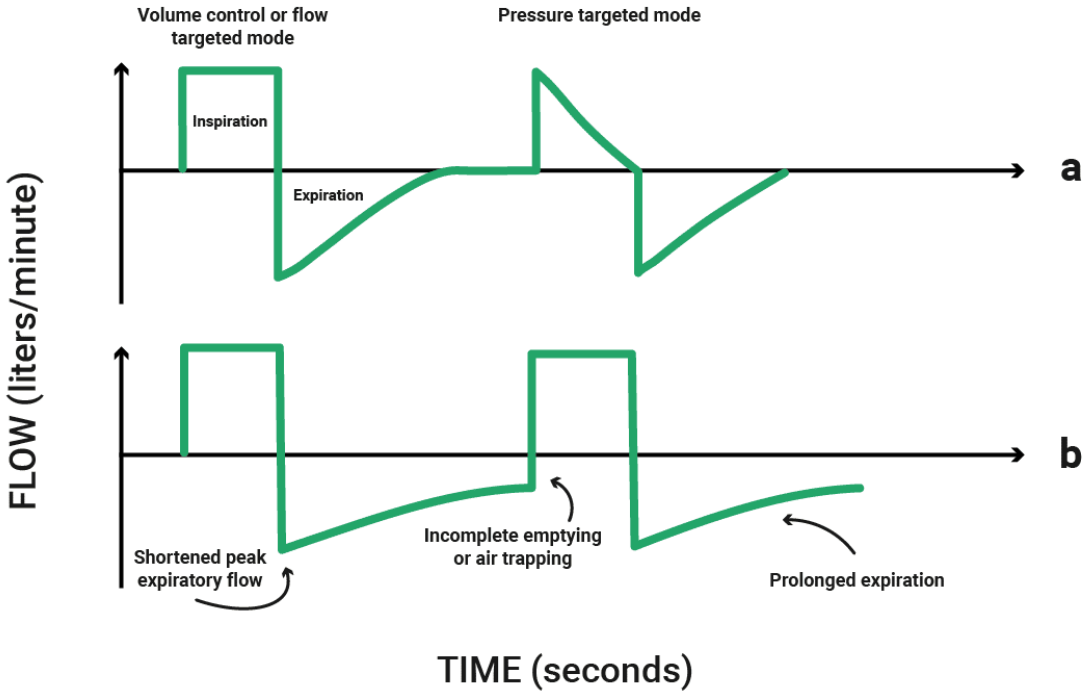
PEEP: Positive End-Expiratory Pressure **Palv:** Alveolar Pressure

- In **Volume-Controlled modes**, the pressure curve will change depending on the patient's lung mechanics.
- In **Pressure-Controlled modes**, the pressure curve will be flat at a set level, as the pressure is constant.



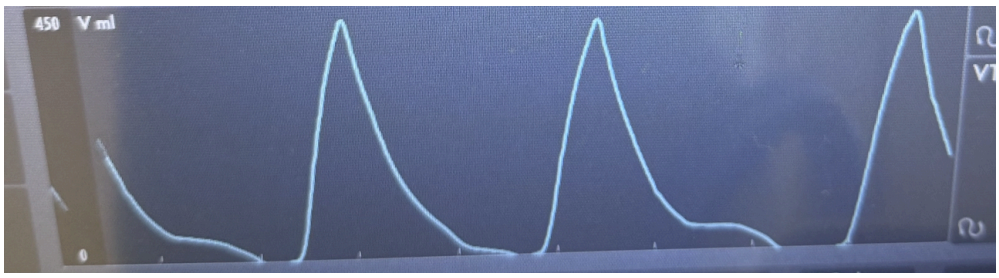
Flow-Time Waveform

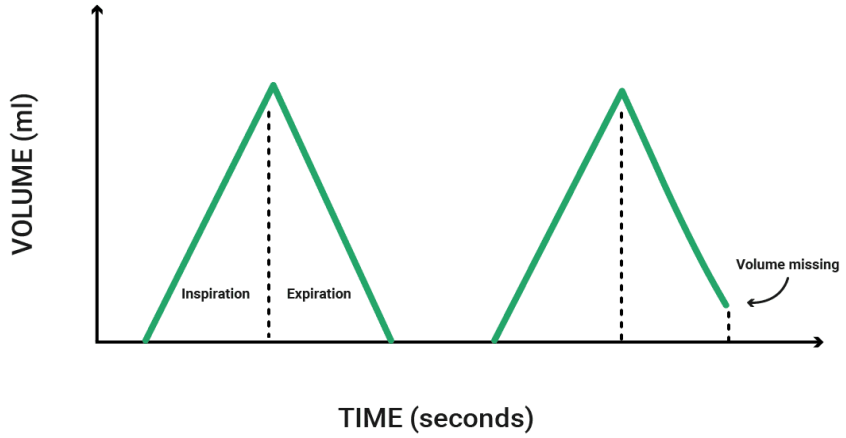




- The Flow-Time waveform is the only one which can have both positive (inspiration) and negative values (expiration).
- **Picture A**: Example of Flow-Time curve in Volume or Flow-Controlled Modes (1st wave)
    - The inspiratory flow is constant (square wave) and can be manually selected on the ventilator (in Volume-Control / VC mode, for example)
- **Picture A**: Example of Flow-Time curve in Pressure-Controlled Modes (2nd wave)
    - The inspiratory flow is in a decelerating ramp pattern (most common) and most mimics natural inspiration
- Ideally, during expiration, the flow should gradually return to 0 (baseline) before the initiation of the next breath (inspiration).
- **Picture B:** Example of Auto-PEEPing or Air-Trapping; when the expiratory flow does not return to baseline before the next inspiration, which represents prolonged expiration (seen in asthma/COPD). If this happens for a prolonged time, the lungs will gradually retain more and more volume, which can result in hemodynamic instability.
    - See Troubleshooting section later for more details

Volume-Time Waveform

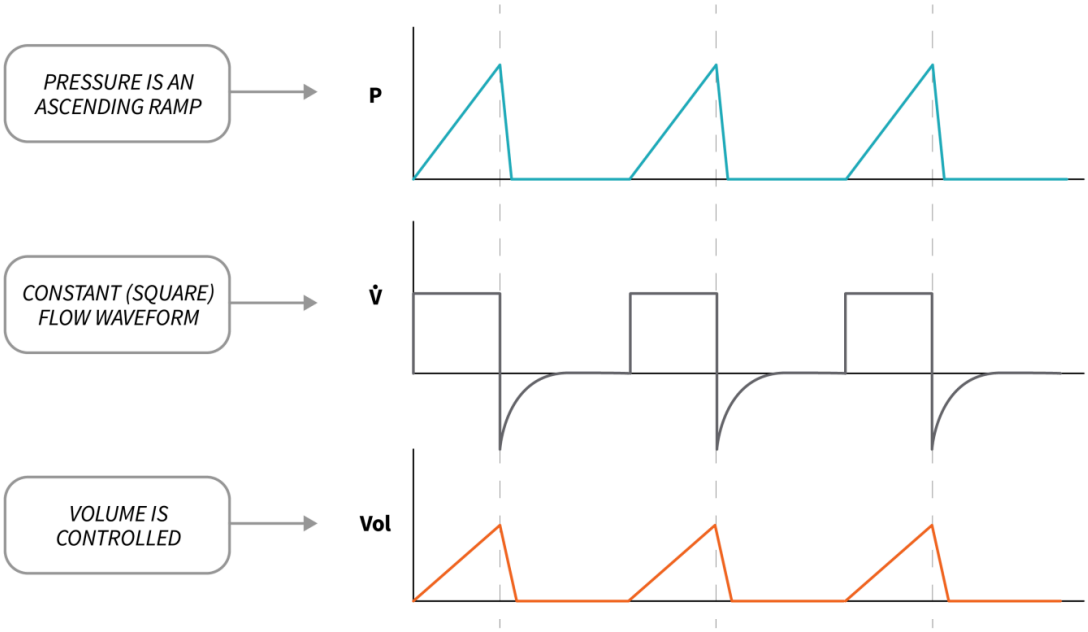




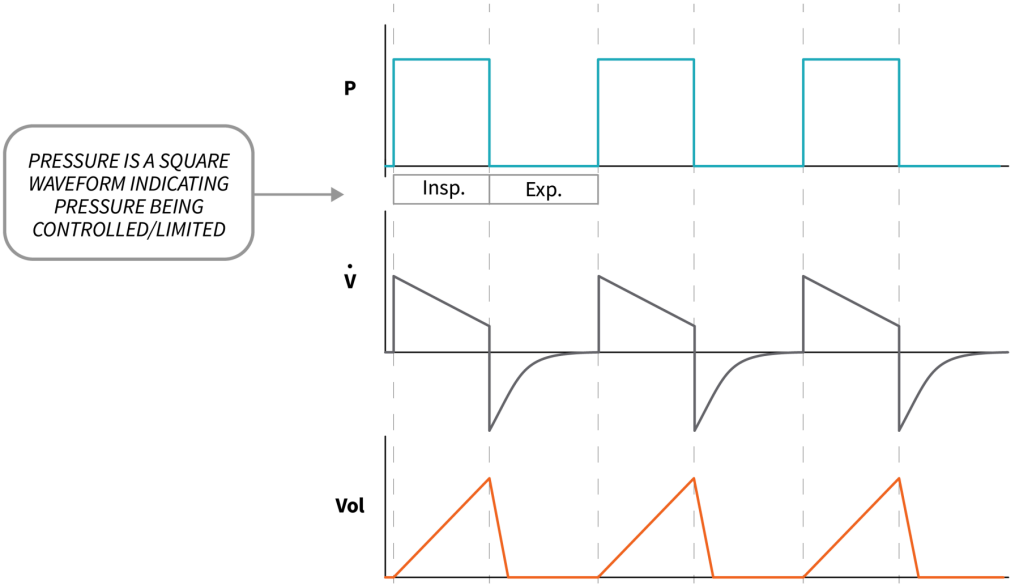
- The inspiratory limb shows the volume increasing, while the expiratory limb shows the volume decreasing.
- In a healthy patient-ventilator interaction, the inspiratory volume equals the expiratory volume, and the curve returns to the 0 baseline.
- If the expiratory limb doesn't return to the zero baseline, it may indicate Auto-PEEPing or Air Trapping, because the next inspiration is occurring before the prior expiration had time to return to 0 baseline.
    - If there is no Air Trapping, then it can also indicate an Air-Leak, meaning the ventilator is not sensing some of the expired breath and it is being ‘leaked’ out where the ventilator can’t sense it
        - Usually not-fully-inflated ET Tube cuff balloon (because some of the expired air will leak around the deflated balloon and exit through the patient’s mouth), instead of back through the ET Tube
        - Can also be ET Tube migration (usually pulled up near the vocal cords), or indicative of a broncho-pleural fistula (where alveolar air is being let out through a chest tube, for example)

    - Concern for an Air-Leak can also be confirmed by looking at the VTi (inspiratory) and VTe (expiratory) tidal volumes. They should be close to each other (within ~50mL or so). A larger discrepancy, consistently, is indicative of an Air-Leak and warrants further investigation.

Volume Control (VC)



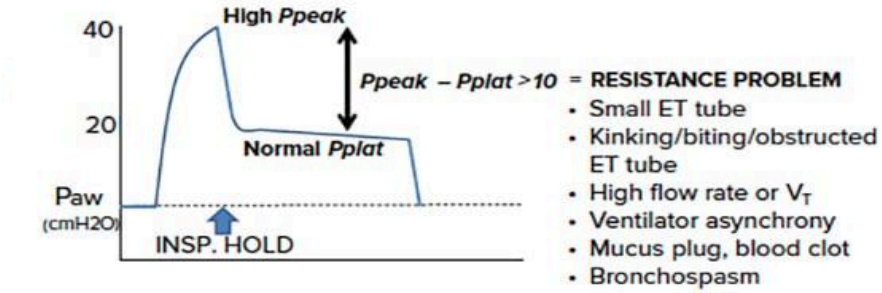
Pressure Control (PC)

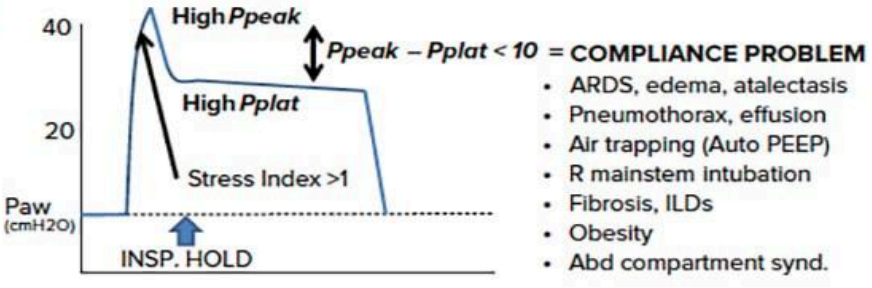


Common Problems & Troubleshooting

High Peak Pressure

- **Definition**: Peak inspiratory pressure > 30 cmH_2O
- **Causes**:
    - Remember that Peak Pressure is a combination of Compliance (lung + chest wall) and Resistance.
        - Increased Airway Resistance
            - Secretions, Bronchospasm, Kinked ETT, Biting Down
        - Decreased Compliance
            - Lungs: ARDS, pulmonary edema, pneumothorax, atelectasis, pulmonary fibrosis
            - Outside Lungs: Large pleural effusion, abdominal distension, kyphoscoliosis, obesity, Fentanyl rigid chest syndrome
        - Other
            - Inappropriate vent settings, vent malfunction, patient-ventilator dyssynchrony (coughing, discomfort, agitation, etc…)
- **What to do**:
    - You must determine which component is the problem (Resistance or Compliance)
    - Check Plateau Pressure (press “inspiratory hold” on vent for a few seconds):
        - A High Peak Pressure (>30) with a High Plateau Pressure (thus a small Pta) indicates that the alveolar pressure is also high, reflecting a Compliance problem and not a Resistance one (↑ p<u>L</u>ateau = <u>L</u> for Lung = comp<u>L</u>iance problem)
        - A High Peak Pressure (>30) with a Low Plateau Pressure (thus a high Pta) indicates a Resistance problem
    - If Resistance problem:
        - Ensure proper ventilator settings and patient synchrony
        - Listen to breath sounds
        - Trace the vent tubing from the vent to the ET Tube and assess for any kinks, water condensation, or obstruction
        - Ensure patient not biting down on ET Tube, Use in-line suction and see if any secretions come up (and ensure you can pass the entire length of the suction through the tube, or whether it is being stopped prematurely)
        - Listen to patient (?wheezing, ?decreased breath sounds)
    - If Compliance problem:
        - Ensure proper ventilator settings and patient synchrony
        - Listen to breath sounds
        - Check POCUS (eval for bilateral lung sliding, B-lines, presence of large pleural effusions or atelectasis, abdominal ascites)
        - Check CXR if unsure
        - Treat underlying cause; ensure lung protective ventilation strategies and minimize barotrauma

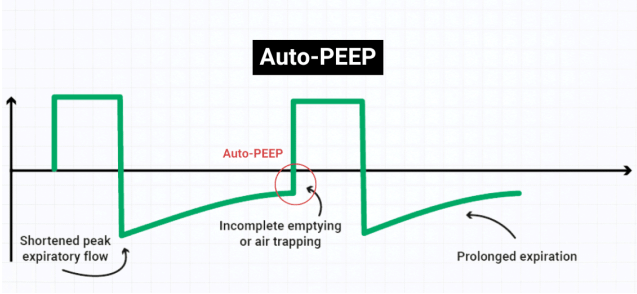




Hypoxemia / Desaturation

- Ensure proper ventilator settings and ensure true hypoxemia (look at SpO2 waveform)
- Check airway (ET Tube in place?, cuff fully inflated?, patient biting tube?, secretions?)
- Check ventilator circuit (disconnected?, air-leak?)
- Think about the patient: pneumothorax, secretions, worsening ARDS, pulmonary edema

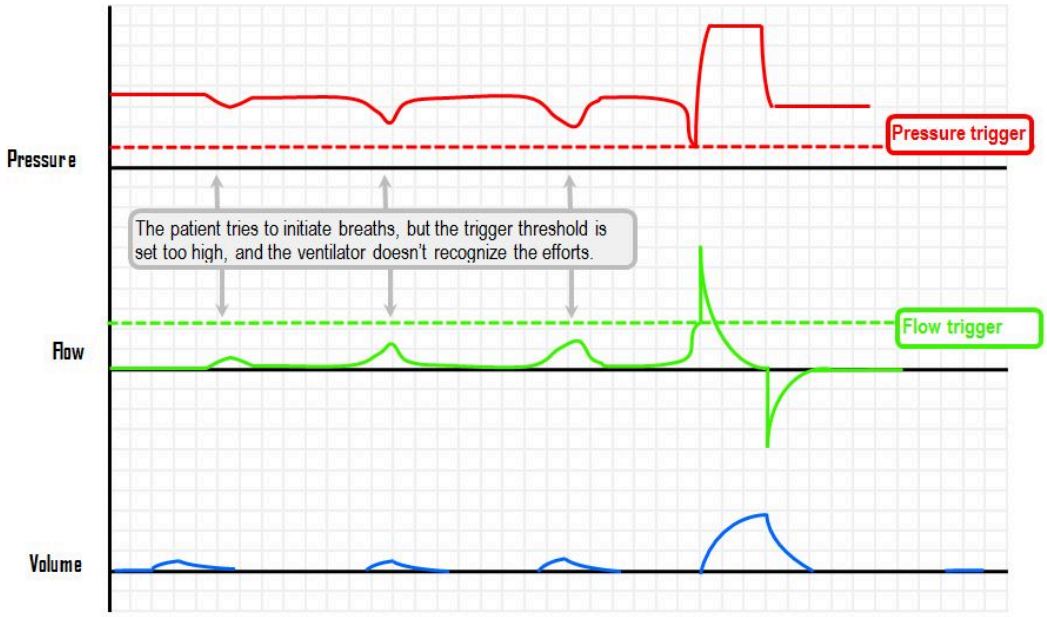
Auto-PEEP / Air-Trapping

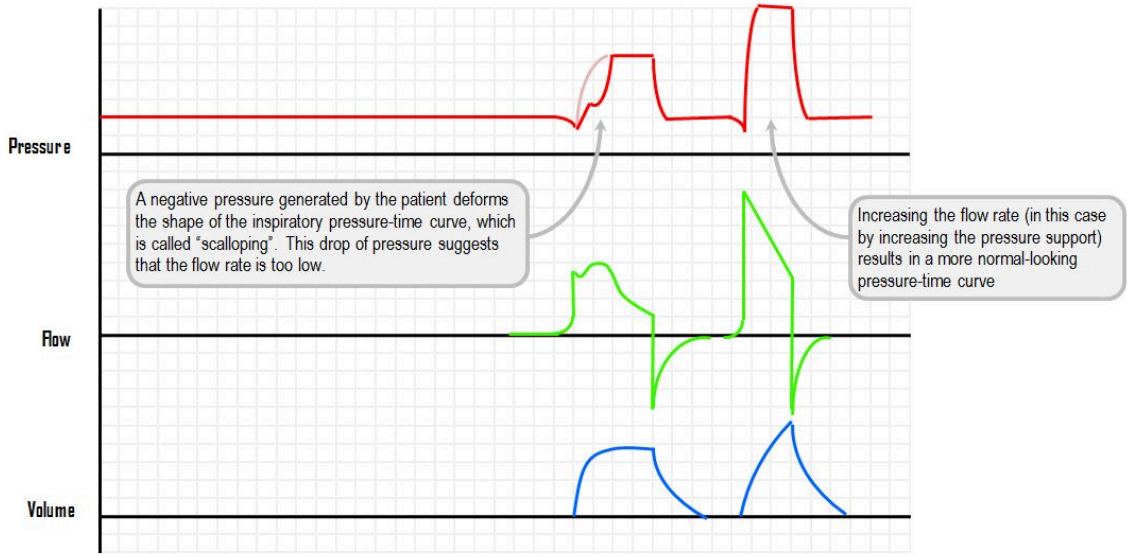


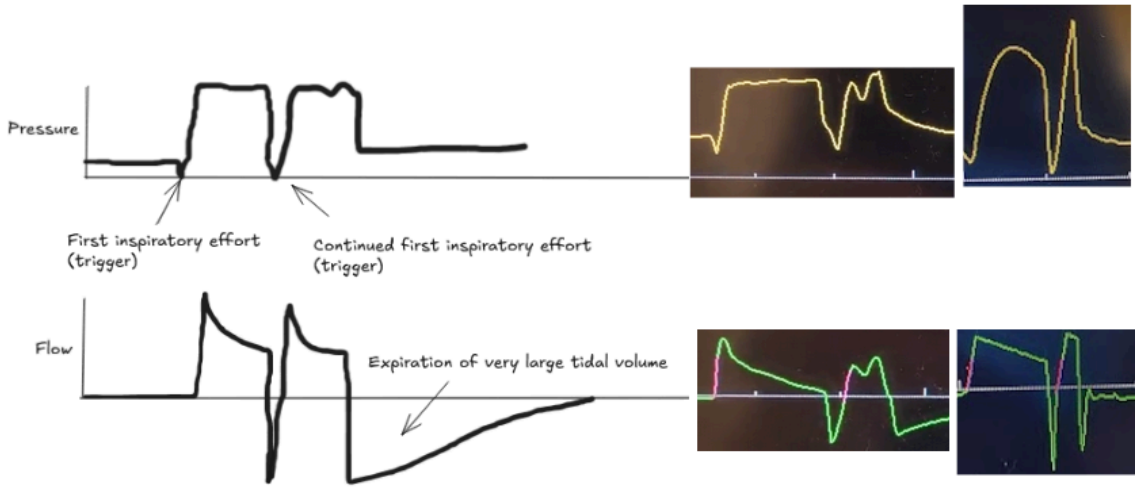
- When the expiratory flow does not return to baseline before the next inspiration, which represents prolonged expiration (seen in asthma/COPD). If this happens for a prolonged time, the lungs will gradually retain more and more volume, which can result in hemodynamic instability.
- Prolong expiration: Decrease RR or TV (if able to), Increase Inspiratory Flow (which would decrease inspiratory time, thus increasing expiratory time)
- Add intrinsic PEEP, sedate more, give more bronchodilators, and/or disconnect from vent temporarily and allow air to leave lungs then place back on vent w/ better settings

Ventilator Dyssynchrony

- Signs: Patient appears uncomfortable, tachypneic, “fighting the vent,” irregular waveforms
- Common causes:
    - Inadequate analgesia / sedation, coughing
    - Mismatch between patient effort and vent settings
- What to do:
    - Look at the waveforms and determine what dyssynchrony is present and address the underlying etiology







Double-Triggering ("Breath Stacking"): Pt wants more Tidal Volume or longer inspiration

POCUS Basics

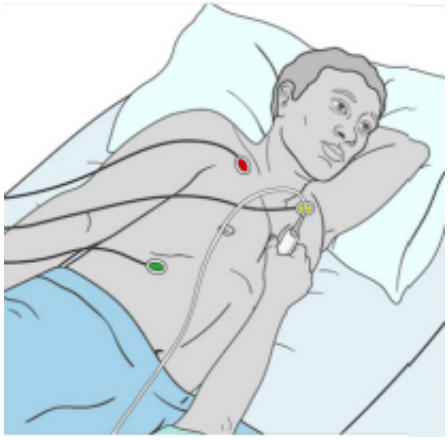
Point-of-Care Ultrasound (POCUS) can be a valuable tool in guiding the diagnosis and management of critically ill patients. The objective of this chapter is to serve as a basic guide for performing ultrasound in a systematic manner and obtaining good quality images. Interpretation of images and clinical application is beyond the scope of this guide.

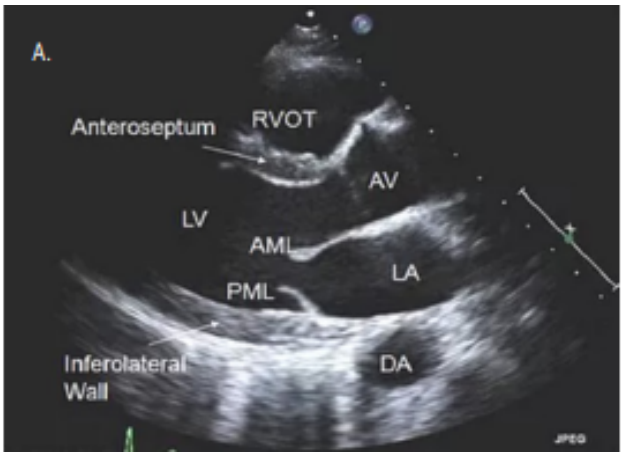
- If you need assistance, ask a senior resident, fellow, and/or attending.
- If you find pathology, get extra images and show your fellow/attending.
- Please take good care of the machines. They benefit you, your colleagues, and your patients. After each use, clean the probe with disinfectant wipes. A new sterile gel packet must be used for each patient (throw away the old one in between patients). During procedures, be sure to use a sterile barrier probe cover and sterile gel. Afterwards, be sure to plug in each US to ensure full charge. Place machines on Standby (not Off) when done.

Cardiac POCUS

View 1: Parasternal Long Axis (PLAX)

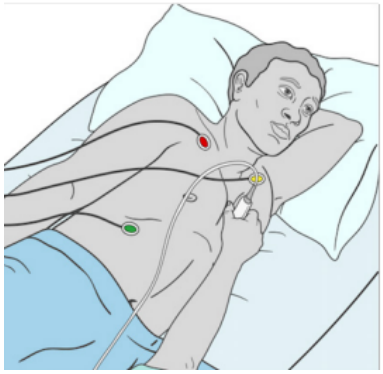
- Place the Cardiac Probe in the Left 2-4th intercostal space, along the sternal border, with the probe marker pointing towards the patient's R shoulder
- Demonstrate LA, MV, LV, interventricular septum, AV, aortic outflow, RV outflow tract, and descending aorta (DA)
- Maneuvres to help open the intercostal spaces and optimize your view: HOB 15 degrees, place the patient in slight left lateral decubitus position with their arm above the head if possible

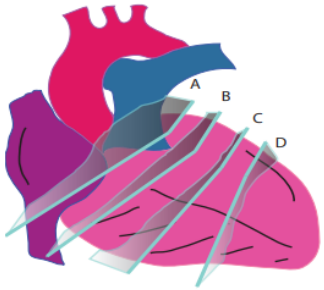


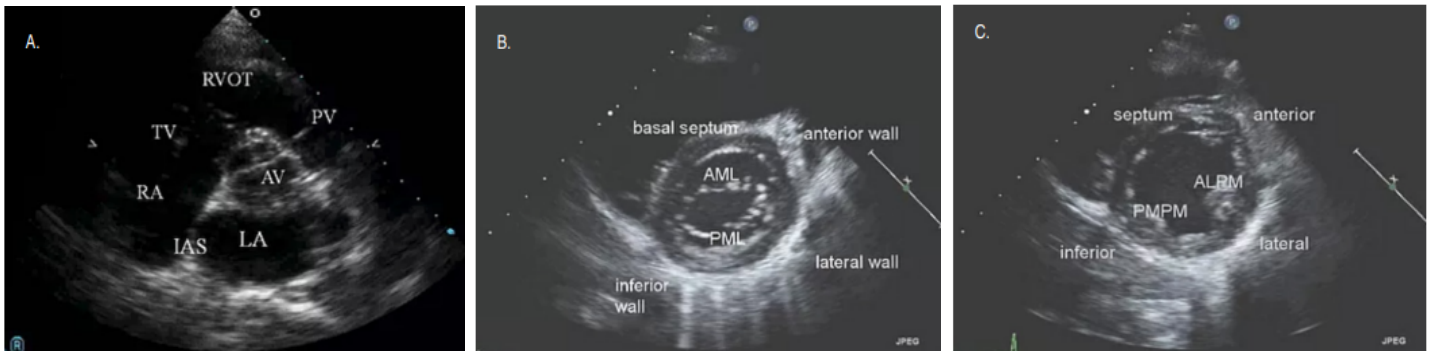


View 2: Parasternal Short Axis (PSAX)

- Once a PLAX view is obtained, rotate the probe 90 degrees clockwise towards the patient's left shoulder
- Tilt probe from base to apex to obtain short axis views of (A) aortic/tricuspid/pulmonic valve level, (B) mitral valve level ('fish mouth'), (C) LV mid-papillary muscle level (should see completely around muscle) (MOST IMPORTANT)
- Best for global function & wall motion abnormalities; interventricular septal flattening
- Maneuvers to help optimize view: End expiratory hold to reduce overlying lung volume, moving probe towards patient's right shoulder

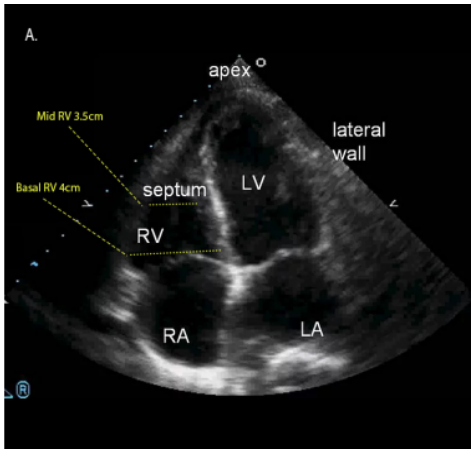


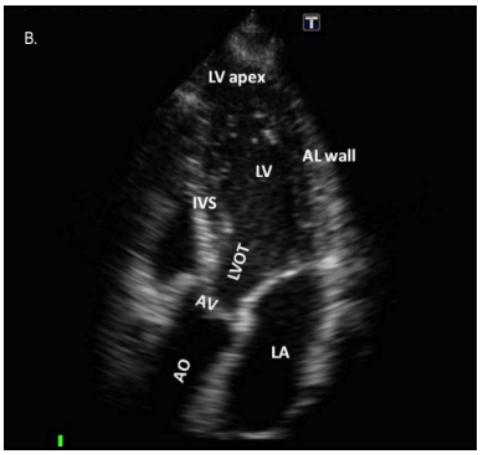




View 3: Apical 4-Chamber (A4C) / 5-Chamber (A5C)

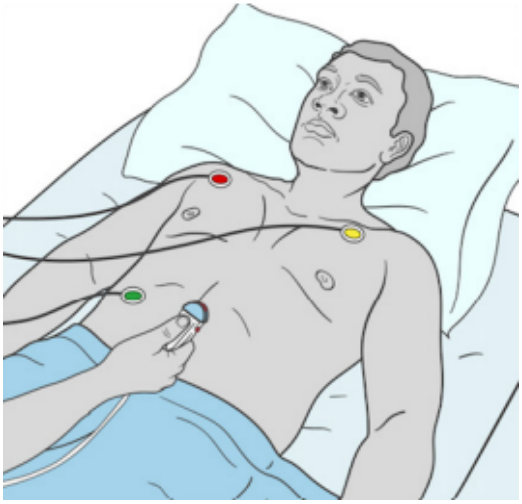
- Place the probe at the cardiac apex from PMI palpation (inferior to nipple, mid-clavicular line), with the probe marker pointing towards the patient's left. Angle the probe upwards/flatter and towards the R shoulder, so that the probe would be 'looking' at the heart.
- 2 Views can be obtained:
    - A) Apical 4 chamber: center apex in the middle of the screen, with septum parallel to beam. Should see LV, RV, LA, RA
    - B) Apical 5 chamber: Angle the probe upwards/flatter to the chest a tiny bit more towards the R shoulder until LVOT and AV comes into view
- Good for comparing chamber sizes, septal bowing, and pericardial effusions

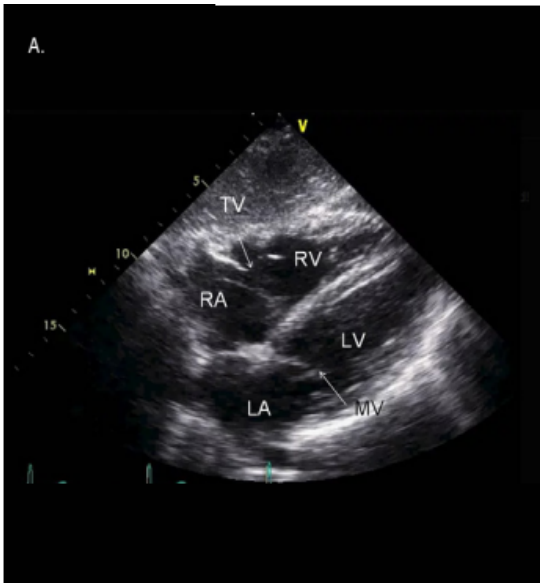




View 4: Subxiphoid/Subcostal

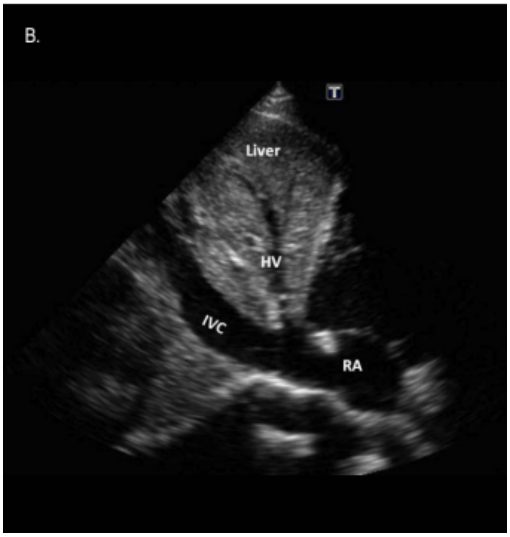
- Place the probe in the subxiphoid space, nearly flat on the patient's skin, angled slightly towards the patients L shoulder, with the probe marker pointing towards the patient's left
- Best for assessing global function, and presence of pericardial effusion
- Alternative view if A4C can't be visualized (essentially the A4C view, just a bit sideways); 1st view to get during Cardiac Arrest
- Maneuvers to help optimize view: supine, legs bent to relax abdomen, deep end-inspiratory hold





View 5: IVC

- Once you obtain your subxiphoid view, rotate the probe 90 degrees counter clockwise so that the probe marker is towards the patient's head, and have the probe facing straight down into the abdomen
- Gently fan the probe towards the patient's left and right to view the entirety of the IVC as a cylinder; fanning a bit more towards the patient's left would show you the aorta instead
- IVC draining into the RA, with the Hepatic Vein (HV) joining the IVC

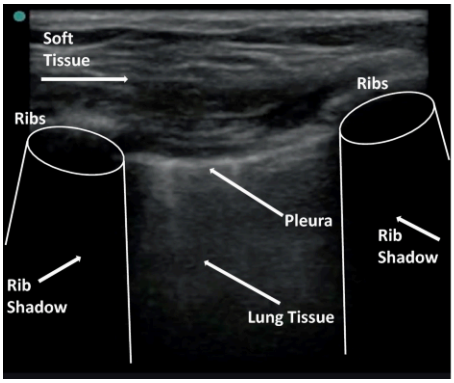


Lung POCUS

Lung POCUS is an extremely useful tool in evaluating respiratory complaints, and can provide valuable information while waiting for further imaging such as an Xray or Chest CT. It is important to understand that Lung POCUS findings, such as “A Lines” and “B lines” are not physical representations of lung anatomy, but rather are artifacts created by ultrasound waves travelling through air/fluid/interstitium within the lung.
Lung POCUS is always performed with the probe marker facing upwards (towards the patient's head) and the probe straight down into the chest (perpendicular with the skin). The Phased Array probe is mostly used, and the US machine preset setting is usually ‘Abdomen’ (not lung). Several points anteriorly are chosen, then mid-axillary line (diaphragm), then posterior to that line (PLAPS point; most sensitive for pleural effusions). This is repeated on the other side.

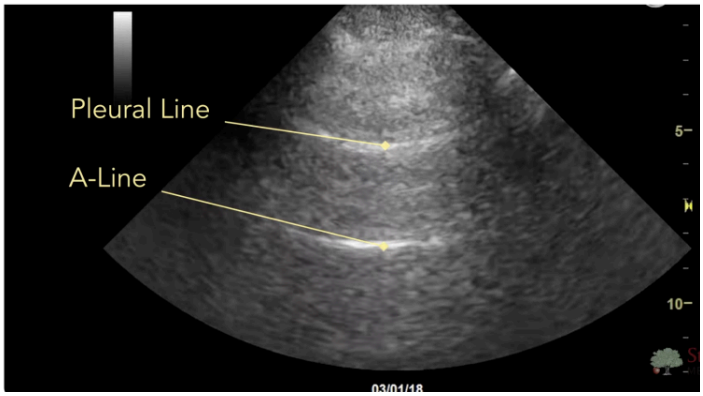
Batwing Sign

- When assessing apical/mid lung-zone areas on POCUS, it is important to confirm that you are in the correct position by ensuring that your ultrasound probe lies between two ribs, creating the “batwing” sign



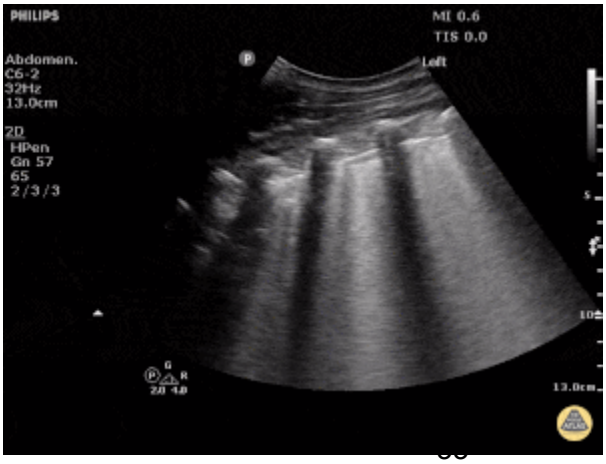
A-lines

- A-lines are horizontal lines that appear during lung ultrasound, signifying normal, aerated lung tissue directly beneath the probe.
- The presence of A-lines helps rule out significant pathology in the superficial lung areas, but deeper pathology could still be present.
- They are ‘echoes’ of the pleural line, and can be several A-Lines ‘deep’



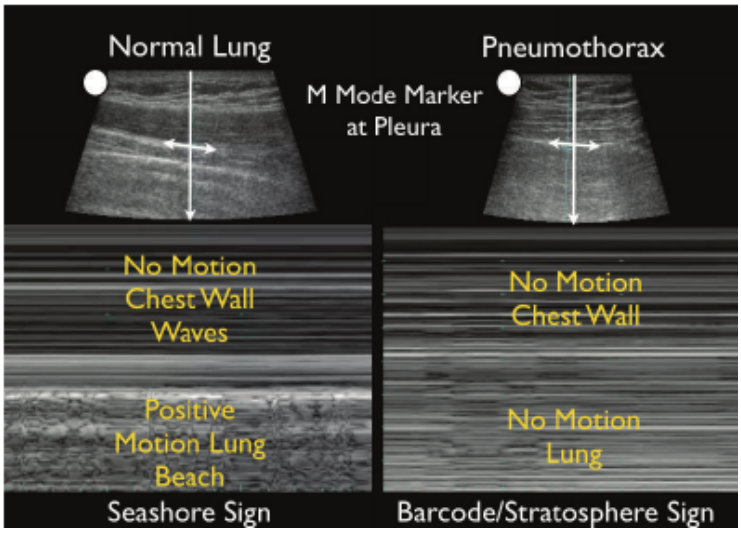
B-lines

- B-lines are vertical lines (“spotlights”) that start at the pleural interface and extend to the bottom of the ultrasound screen, moving with the patient’s breath.
- B-lines suggest the presence of interstitial fluid and are commonly associated with conditions like pulmonary edema, consolidation, fibrosis, or cancer (lymphangitic spread).



Lung Sliding

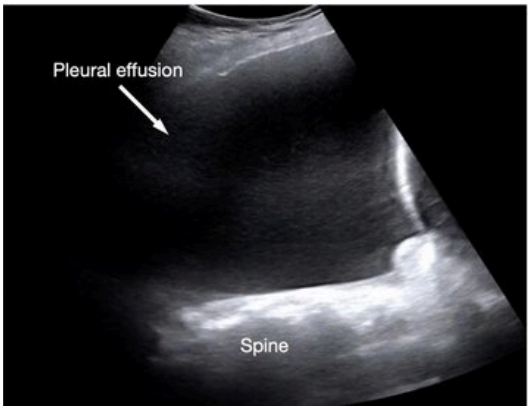
- Normally, the visceral and parietal pleura move together during respiration, a phenomenon seen on ultrasound as lung sliding. This is visualized as tiny movements or sliding of the bright white line (pleural line) between the ribs
- Absence of Lung Sliding: May be an indicator of pneumothorax. When air is present between the pleural layers, it prevents the visceral and parietal pleura from moving together, causing the lung sliding to disappear on the ultrasound image.
    - Other causes: unilateral intubation, lung fibrosis, reduced tidal volumes, post-pleurodesis

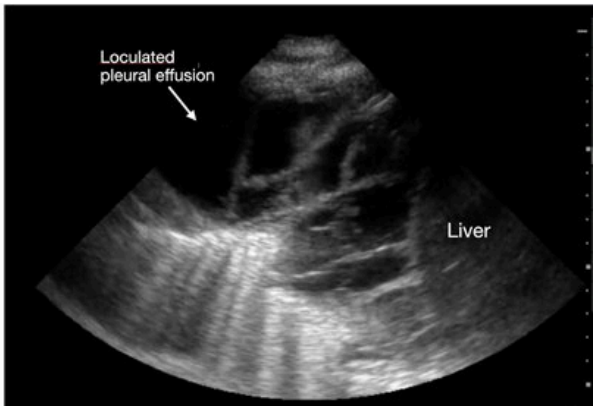


- If you are unsure of the presence/absence of lung sliding, “M-Mode” can be used
    - Seashore vs Barcode signs
- The Linear US Probe can also be used to especially focus on the pleura

Pleural Effusions

- To identify the presence/absence of an effusion, the interface between the liver/spleen (depending on the side being examined), the diaphragm and the lung must be identified. Place probe at mid-axillary line, and move up/down until diaphragm in center
- Pleural effusions will appear above the diaphragm, as an anechoic space
- Once an effusion is detected, assess its size and extent. The presence of septations (internal divisions within the fluid) or loculations (compartments) can suggest a more complex effusion, such as an empyema, which might require different management than a simple effusion. Fluid can also appear hazy/fuzzy and also suggest a complex effusion (hematocrit sign, for example; representing blood)





Dialysis in the ICU

Common terms to know

Access	
AV (Arterio-Venous)	Blood taken from vein, passed through machine and then back into artery (eg fistula)
VV (Veno-Venous)	Blood taken from vein, passed through machine and then back into vein (eg Shiley catheter)

Physiologic Mechanisms Used (EHC dialysis machines can be set up to do one or both)	
Diffusion (commonly referred to as Dialysis)	Movement of solute (eg urea, potassium) from area of high concentration to area of lower concentration across a semi-permeable membrane. **TLDR: Removes solute (eg toxins, K, etc…)**
Ultrafiltration	Removal of solvent (water) from solute by passing solvent through a semipermeable membrane driven by differences in hydrostatic pressure. NOTE: The process of filtration will also remove some solute via convection (friction force applied by moving solvent on surrounding solute which will "drag" solute across membrane in process called solvent drift). **TLDR: Removes solvent ("water")**

Methods of Dialysis	
Intermittent Haemodialysis (iHD)	Most efficient method; runs over 3-4 hours, therefore more solutes can be cleared more quickly, but increased risk of fluid shifts
Sustained Low Efficiency Dialysis (SLED)	Hybrid between iHD and CRRT; runs over 6-8 hours on same machine for iHD
Continuous Renal Replacement Therapy (CRRT)	Slowest; runs continuously over 24 hours with slower blood flow so less efficient solute removal but more hemodynamically gentle. As it's so slow, it sometimes requires adjustment of the filtration fraction or adding anticoagulation (if safe depending on pt's clinical status) to prevent filter clotting. Has own special machine.

Most forms of dialysis can be understood by applying the above terms. For example, CVVHDF will be continuous (24-hours), veno-venous (Shiley catheter), haemo (blood), dialo (diffusion), filtration (ultrafiltration), etc…

Intermittent Haemodialysis (3-4 hours)	
Advantages	**Disadvantages**
Fastest way to remove “toxins” and fluid	Rapid removal of fluid leads to increased risk of hypotension
Generally don’t need anticoagulation as blood flow through filter is quick	Rapid removal of solute leads to increased risk of disequilibrium syndrome

Sustained Low Efficiency Dialysis (SLED) (6-8 hours)	
Advantages	**Disadvantages**
Quicker removal of solute and fluid than CRRT	Slower removal of solute and fluid than iHD
Reduced risk of hypotension than iHD due to slower removal	May still remove fluid and solute too fast for hemodynamically unstable patients
Usually does not need anticoagulation/needs a shorter duration of anticoagulation than CRRT	
Patients can be “off circuit” for portions of day (decreased sedation, increased movement)	

Continuous Renal Replacement Therapy (CRRT) (24 hours)	
Advantages	**Disadvantages**
Slower therapy leads to less aggressive fluid shifts and lower risk of hypotension	Slower blood flow rate leads to slower and less solute removal
	Often interrupted during the day for imaging, transport, procedures, etc…
	Due to slow rate, has increased risk of clotting in the filter which can be mitigated by adjusting filtration fraction or adding anticoagulation if safe for patient
	Prolonged therapy means prolonged immobilization
	Increased nursing workload (only done in MICU) and cost