

Critical Care Training for Non-Critical Care-Trained Providers

Now What Do I Do? Just-In-Time Learning Documents

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My Patient Is Hypotensive... Now What Do I Do?

1) Think about cause (and treat immediately reversible ones)

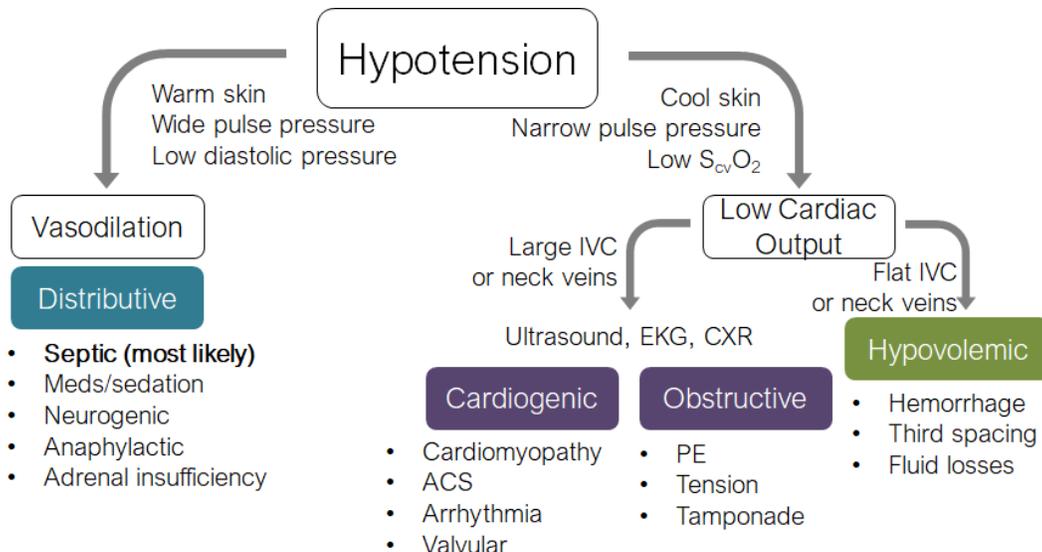


Figure adapted from Jenelle Badulak

2) Assess for volume tolerance (additional volume will not hurt) and responsiveness (additional volume will improve the situation)

- Consider: comorbidities (e.g., heart failure, chronic kidney disease), severity of hypoxemia, cardiac function, volume status
- If volume tolerance and volume responsiveness are likely, move to Step 3 below
- If tolerance and responsiveness unlikely, skip to #4
- If uncertain:
 - **Call for Help!**
 - Use advanced tools if available (e.g., ultrasound, pulse pressure variation)

3) Give fluid bolus and reassess

- Give 500 mL Lactated Ringer's IV and reassess
- Consider additional 500 mL boluses if blood pressure rises or urine output increases
- For patients with ARDS, limit initial resuscitation to 1-2L and be very judicious with additional fluids

4) Start norepinephrine

- Start at 0.02 mcg/kg/min; titrate to mean arterial pressure (MAP) \geq 65 mmHg or the patient's typical baseline blood pressure

“My Patient is Hypotensive Despite Fluids and Norepinephrine...Now What Do I Do?”

1) Call for Help!

2) Reassess the situation:

- Are the blood pressure measurements accurate?
- Do I have the correct diagnosis?
- Do I have source control? (e.g., is there a surgical infection that requires drainage or debridement, does the patient have a parapneumonic effusion?)
- Is my antimicrobial coverage appropriate?

3) Start vasopressin at 0.03 units/minutes if norepinephrine > 0.2 mcg/kg/min

4) Consider placing arterial line and central venous catheter, if not already present

5) Next steps:

- Evaluate for sepsis-induced or primary cardiomyopathy with bedside ultrasound or formal echocardiogram, electrocardiogram, and $S_{cv}O_2$ (provided the patient has a central line)

If echocardiogram consistent with cardiomyopathy or $S_{cv}O_2 < 60\%$:

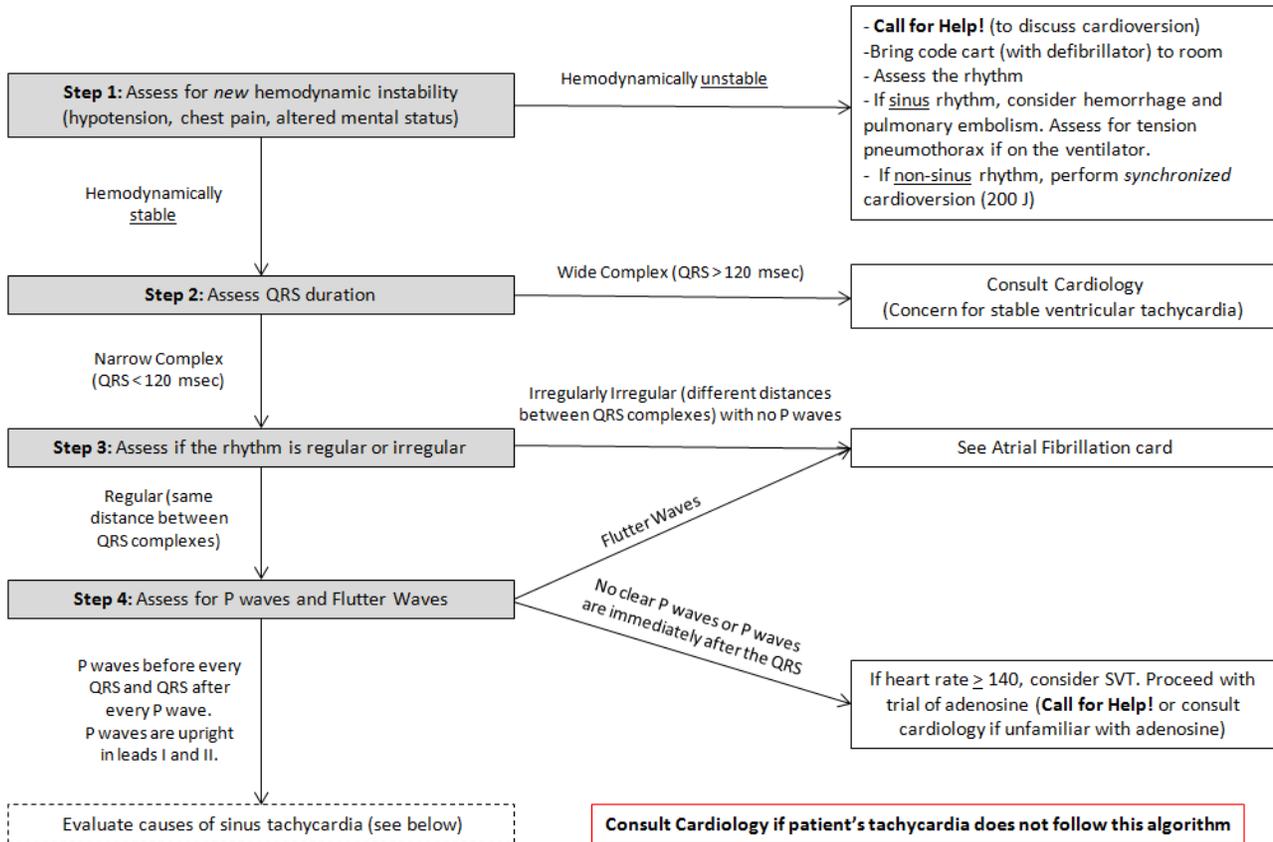
- Consider inotrope (dobutamine 2.5 mcg/kg/min)
- Consider repleting calcium to normal (ionized calcium > 1.0. mmol/L)
- Consider stress-dose steroids (hydrocortisone 50 mg q8 hours IV). Spot cortisol or ACTH stimulation testing are usually not helpful
- Re-evaluate fluid tolerance and responsiveness. Consider judicious 500 mL boluses
- Evaluate acid-base status: Consider continuous renal replacement therapy or bicarbonate infusion if the patient has a severe metabolic acidosis and pH <7.1

6) If vasopressor needs continue to increase, consider Palliative Care Consultation for goals of care discussion

My Patient is Tachycardic... Now What Do I Do?

1) Obtain an electrocardiogram

2) Follow the steps below



2) Evaluate for causes of sinus tachycardia

Do not just start a beta-blocker to improve the heart rate. Identify and treat the specific cause of the sinus tachycardia.

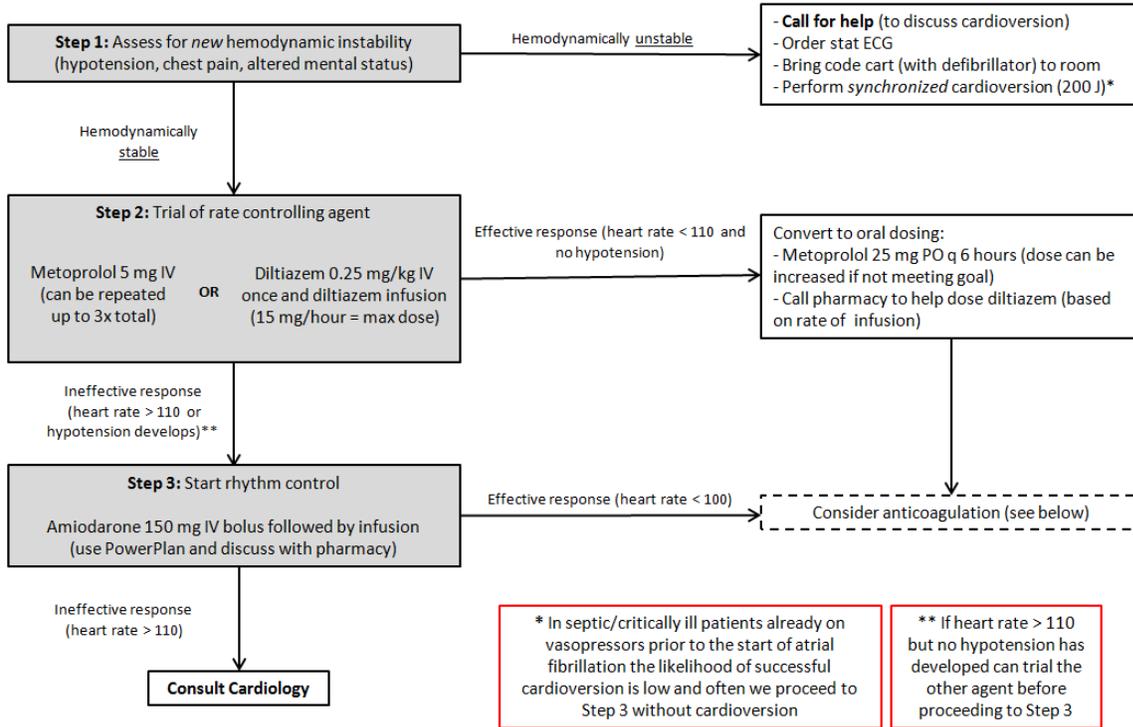
- Pain and anxiety
- Fever / infection
- Hypoxemia
- Intravascular volume depletion (e.g, over-diuresis, vomiting/diarrhea, NPO)
- Blood loss (including non-obvious sources like the retroperitoneum)
- Alcohol withdrawal
- Pulmonary embolism (uncommon cause of sinus tachycardia in absence of hypoxemia, hemodynamic changes, shortness of breath)

Note: thyroid studies are typically unhelpful in critically ill patients and should not be ordered unless other clinical signs point toward hyperthyroidism as likely diagnosis.

My Patient Is In Atrial Fibrillation (or Flutter)... Now What Do I Do?

1) Obtain an electrocardiogram if not done already

2) Follow the steps below



3) Evaluate for common causes of new onset atrial fibrillation

- Sepsis/critical illness (most common cause in the ICU)
- Ischemic heart disease or valvular disease
- Anemia
- Medications (e.g. vasopressors)
- Alcohol withdrawal
- Thyroid disease (typically TSH should not be tested in critically ill patients)
- Pulmonary embolism (uncommon cause, do not automatically order CTPA)
- Sleep disordered breathing

4) Consider anticoagulation

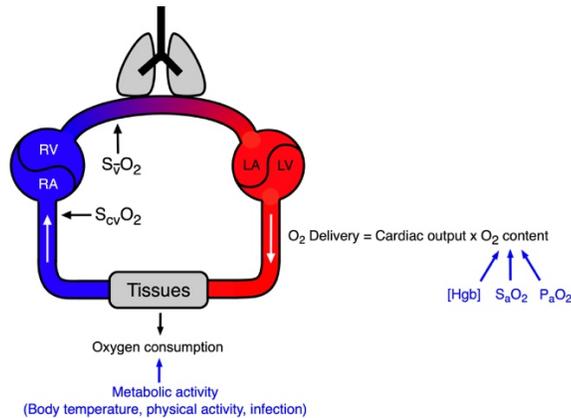
Call for Help! to make decision about anticoagulation in the next 12 hours.

For many critically ill patients, including those whose atrial fibrillation is driven by sepsis or respiratory failure, the risks of systemic anticoagulation outweigh the benefits. In these patients re-evaluate the need for systemic anticoagulation once they are stable enough to transfer to the acute care service (depending if they are still having issues with atrial fibrillation).

My Patient's Central Venous Oxygen Saturation Is Abnormal... Now What Do I Do?

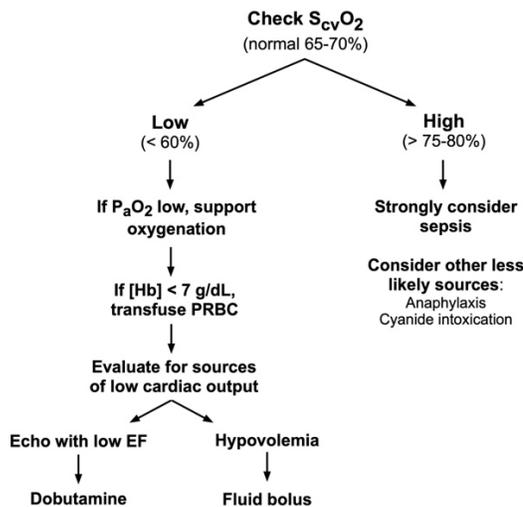
1) What information do we get from the central venous oxygen saturation?

The central venous oxygen saturation ($S_{cv}O_2$) is the oxygen saturation of venous blood drawn from a central line whose tip resides in the distal superior vena cava. It is a surrogate for the mixed venous oxygen saturation (drawn from the pulmonary circulation, $S_{\bar{v}}O_2$) and is determined the balance between oxygen delivery to the tissues (determined by hemoglobin concentration, arterial oxygen saturation, and cardiac output) and tissue oxygen consumption, which is determined by metabolic activity. The normal value for the $S_{cv}O_2$ is typically 65-70%.



2) Before you act on a measured value, confirm the location from which it was sampled. Blood gases drawn from peripheral IVs, veins in the arm or hand or the femoral vein cannot be used to assess a central venous saturation. Only if the sample was drawn from a central line should you proceed to the next steps.

3) Interpreting the $S_{cv}O_2$ and responding to deviations from normal



Call for Help! if you are starting dobutamine

My Non-Intubated Patient Is Hypoxemic... Now What Do I Do?"

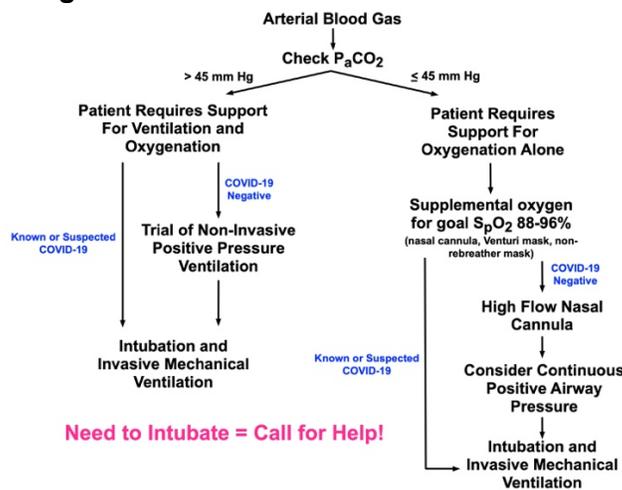
1) Ensure the pulse oximeter is providing accurate data

Bad pulse oximetry waveforms suggest erroneous data. If unable to rectify the problem, check an arterial blood gas to measure the P_aO_2 .

2) Evaluation upon initial presentation

- Plain chest radiograph
- Arterial blood gas
- Basic laboratory studies (WBC count, B-type natriuretic peptide)
- Electrocardiogram (if indicated)

3) Initial management algorithm



Note: The decision to intubate is never based on specific SpO_2 or PaO_2 threshold and, instead must take into account an assessment of the entire clinical picture including the patient's work of breathing, mental status and hemodynamic stability.

4) Initiate disease/problem specific interventions

Problem	Intervention
Heart failure and/or volume overload	Diuresis
Large pleural effusions	Diuresis, consider thoracentesis
Pneumonia	Antibiotics
Lobar or whole lung collapse	Chest physiotherapy
COPD / Asthma exacerbation	Corticosteroids, inhaled bronchodilators
Suspected pulmonary embolism	Consider CT pulmonary angiogram *, lower extremity duplex

* For known or suspected COVID-19 patients, discuss the risk/benefits of traveling for CT scan

5) What is the target S_pO_2 and P_aO_2 ?

- S_pO_2 : 88-96%
- P_aO_2 : 60-90 mm Hg

My Patient Just Got Intubated... Now What Do I Do?

1) Choose Your Ventilator Settings

Mode: Volume assist control

Tidal Volume: 8 ml/kg of ideal body weight

Rate: Based on an assessment of the patient's minute ventilation needs. This can be done based on an assessment of the patient's bicarbonate:

Bicarbonate (mEq/L)	Target Minute Ventilation (L/min)
22-26	6-8
16-20	10-12
< 12	15-20

F_IO₂: 1.0

PEEP: 5 cm H₂O

2) Place Orogastric Tube

This is for enteral access for medications. Tube feeds can be held at this stage and should not be immediately started in patients on escalating doses of vasopressors.

3) Choose Your Sedation and Pain Management Plan

Sedative: Propofol infusion. Titrate for Richmond Agitation Sedation Score (RASS) score of 0-1.

Pain Control: Start with fentanyl boluses (25-100 µg q 30 min prn); Change to infusion if insufficient.

4) Check a Chest Radiograph to Confirm Position of Endotracheal and Orogastric Tube

5) Obtain a Blood Gas in 15-30 minutes

- Check the acid-base status: Adjust ventilator rate accordingly with the goal of achieving a pH relatively close to normal (7.35-7.45). This can be difficult to achieve with a severe primary metabolic acidosis. Further details on how to adjust the ventilator rate is provided in the information sheet "I Just Got The Blood Gas Results... Now What Do I Do?"
- Check the P_aO₂: If the P_aO₂ > 100 mm Hg, decrease the F_IO₂ to target S_pO₂ > 88%. Further changes in PEEP and F_IO₂ can be made by monitoring S_pO₂ rather than checking repeat blood gases. For patients with ARDS, follow the PEEP/ F_IO₂ ladder.

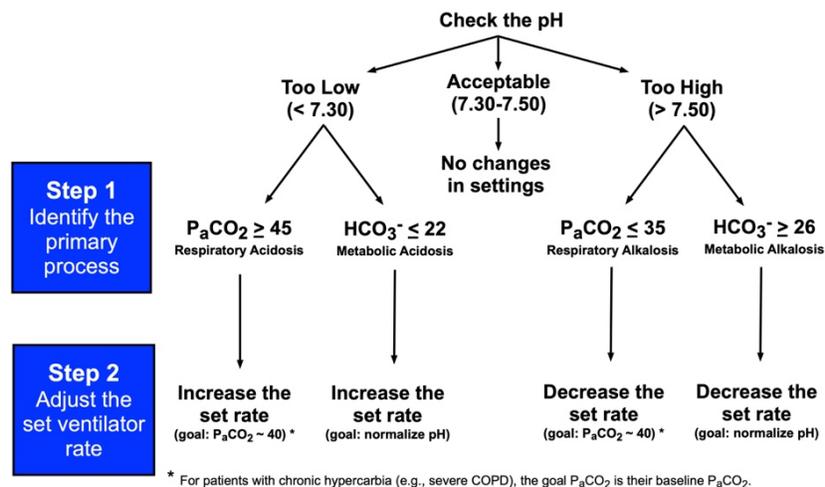
To avoid oxygen toxicity, do not allow the S_pO₂ to remain at 100%.

I Just Received Blood Gas Results On My Ventilated Patient... Now What Do I Do?

1) Assess oxygenation (this cannot be done with a venous blood gas)

- $P_aO_2 < 60$: Increase $F_I O_2$ and/or increase PEEP
- $P_aO_2 > 100$: Decrease $F_I O_2$ until S_pO_2 90-96%

2) Assess and address the acid-base status



3) Adjustments in the ventilator rate

A simple rule of thumb can be used to determine the proper change in the respiratory rate:

$$\text{New rate} = \text{Current rate} \times \frac{\text{Current } P_aCO_2}{\text{Target } P_aCO_2}$$

Call for Help! if desired rate is > 35

4) In What Situations Will I Have Trouble Achieving The Goal pH or P_aCO_2

There are several situations in which it can be difficult to achieve the goal P_aCO_2 or pH

- Severe ARDS: in patients with a high dead-space fraction, it can be hard to normalize P_aCO_2 and pH despite a high minute ventilation (Most patients can tolerate a pH down to 7.15 in these situations). **Call for Help!**
- Severe metabolic acidosis: despite significant decreases in P_aCO_2 , the pH may remain low. **Call for a Help!**
- Overbreathing the ventilator: decreases in the set respiratory rate will not achieve a change in minute ventilation. The patient will continue to over-breathe the ventilator

5) How Long Before I Need to Get Another Arterial Blood Gas

- After changes in $F_I O_2$ or PEEP: repeat ABG is not necessary. Follow S_pO_2
- After changes in the set rate: wait 15-30 minutes before the repeat ABG

My Intubated Patient Is More Hypoxemic... Now What Do I Do?

1) Check equipment, consider calling the respiratory therapist for help

Ensure the following:

- Patient remains connected to the ventilator circuit and circuit is intact
- No inadvertent changes $F_{I}O_2$, PEEP or other settings
- Endotracheal tube is patent and remains in correct position

If the ventilator pressure alarm is sounding, consult the card on “The Peak Pressure Has Increased”

2) Examine the patient and evaluate their interaction with the ventilator

- Listen for bilateral breath sounds. If breath sounds are asymmetric, consider pneumothorax or lung collapse and evaluate accordingly
- If patient is agitated and having repeated peak pressure alarms, administer intravenous fentanyl bolus and consider increasing propofol

3) Obtain diagnostic studies

Arterial Blood Gas (venous blood gases cannot be used to assess oxygenation)
Chest radiograph

4) Adjust the ventilator to improve oxygenation

The two parameters on the ventilator that address oxygenation are $F_{I}O_2$ and PEEP

- Increase $F_{I}O_2$
- If this does not resolve the situation, increase PEEP by 5 cm H_2O
 - Expect a slow rise in oxygen saturation with increased PEEP
 - May cause paradoxical worsening of oxygenation → return to previous PEEP
 - May cause hypotension → return to previous PEEP
- **Call for Help!** if these maneuvers do not resolve the situation. Discuss other strategies with your critical care consultant

5) Treat reversible causes of hypoxemia if present

- Suction the patient to clear any mucus in the endotracheal tube or central airways
- If chest radiograph reveals lobar or whole lung collapse, start chest physiotherapy
- If worsening edema pattern on chest radiograph, consider diuresis or new diagnosis of ARDS (see ARDS sheet)

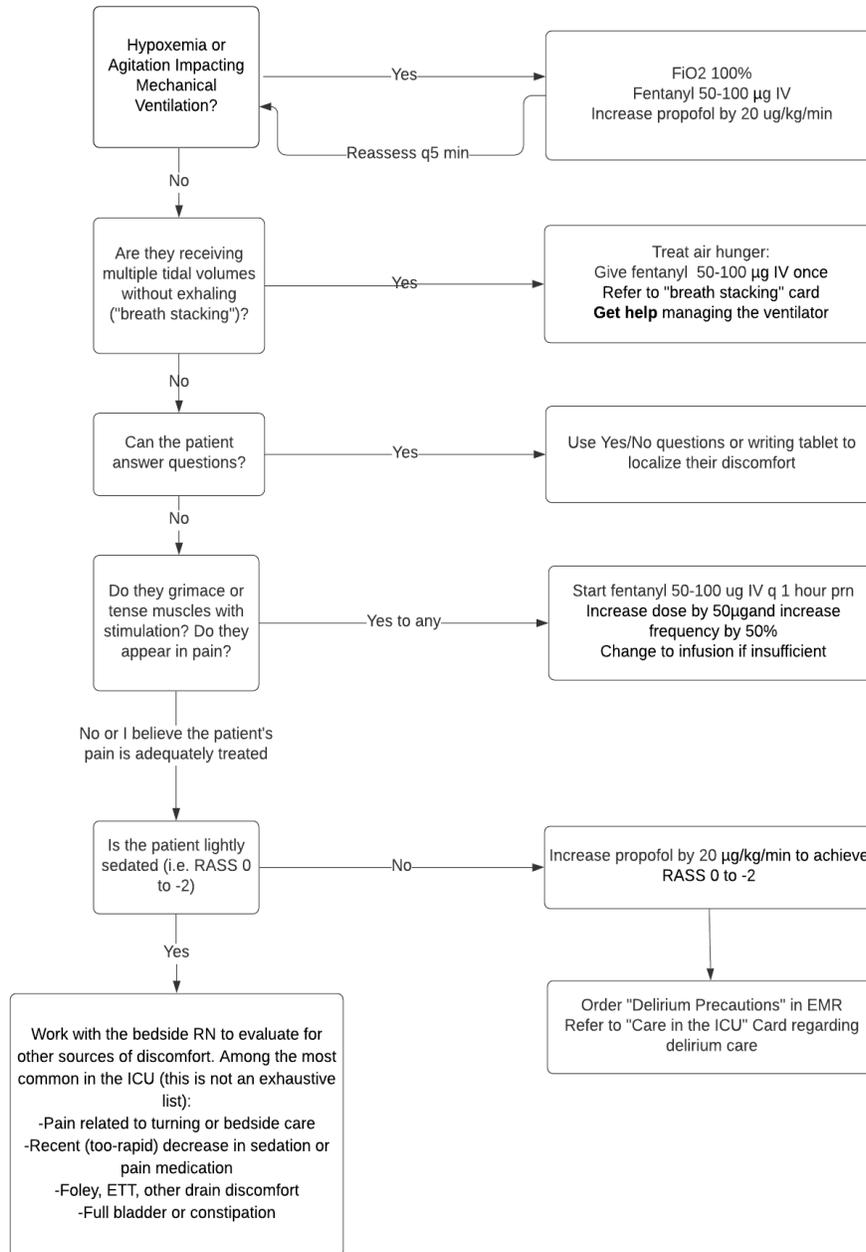
6) Optimize other factors that affect oxygen delivery

- Check hemoglobin and transfuse red blood cells if $[Hb] < 7$ g/dL
- Check $S_{c}vO_2$ and call for help if $< 60\%$
- Review medications for those that can cause pulmonary vasodilation (e.g., calcium channel blockers) as this may worsen ventilation-perfusion matching

Critical Care Skills for Non-Critical Care Providers
Worsening Hypoxemia on the Ventilator

“My Patient is Agitated While Receiving Mechanical Ventilation... Now What Do I Do?”

Usually, patients who are not severely hypoxemic will be maintained in an alert or lightly sedated state. The degree of sedation is measured by the Richmond Agitation Sedation Score (RASS). The goal RASS is usually 0 (alert) to -1 (sedated but easily arousable). When the patient is out of this range (RASS > 0), the following step-by-step process can be used to resolve the issue.

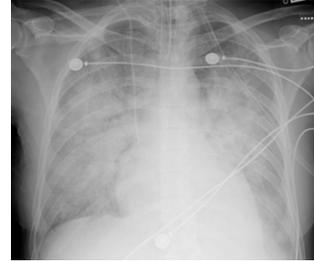


My Patient is Developing ARDS... Now What Do I Do?

1) Confirm the Presence of ARDS and Classify Severity

Patients are deemed to have ARDS if they meet all 4 of the following criteria:

- Acute onset (<7 days) from known cause (e.g., COVID-19 infection)
- Bilateral opacities on chest radiograph
- $P_aO_2 / F_I O_2$ (P/F ratio) < 300 while on PEEP of 5 cm H₂O
- Not *entirely* due to pleural effusions, volume overload or cardiogenic edema



Classification of Severity: (P_aO_2 obtained from ABG; $F_I O_2$ expressed as a decimal)

- Mild: $200 \leq P_aO_2 / F_I O_2 < 300$
- Moderate: $100 \leq P_aO_2 / F_I O_2 < 200$
- Severe: $P_aO_2 / F_I O_2 < 100$

2) Initiate low tidal volume ventilation (often referred to as lung protective ventilation)

Change tidal volume (V_T) to 6 ml/kg *predicted* body weight (PBW)

Goals:

- Plateau Pressure < 30 cm H₂O :
 - If $P_{plateau} > 30$ cm H₂O: consider decreasing V_T further, to as low as 4 ml/kg PBW
 - If $P_{plateau} < 30$ cm H₂O: maintain 6 ml/kg
- S_aO_2 88 – 95% (or P_aO_2 55 – 80 mmHg):
 - Use the PEEP/ $F_I O_2$ ladder. Start with the low ladder
 - Monitor for hypotension due to increased PEEP
 - **Call for Help! with persistent or worsening hypoxemia**
- pH >7.20 (Tolerate increases in P_aCO_2 , “permissive hypercapnia”)

3) If $P_aO_2 / F_I O_2 < 150$ consider prone positioning

Note this requires substantial personnel to safely perform, so consider available resources

Protocol: Prone for 16 hours, then return to supine position

Repeat daily

Stop when P/F >150 on PEEP >10 cmH₂O and $F_I O_2 < 0.6$ or if ineffective

4) If $P_aO_2 / F_I O_2$ and patient is not synchronous with the ventilator, start neuromuscular blockade

48-hour infusion of cis-atracurium

Ensure deep sedation (RASS -4 to -5)

5) Call for Help! if hypoxemia persists despite prone positioning and neuromuscular blockade

The Peak Inspiratory Pressure Is High... Now What Do I Do?

1) What Determines Peak Inspiratory Pressure and What Is a “Normal” Value?

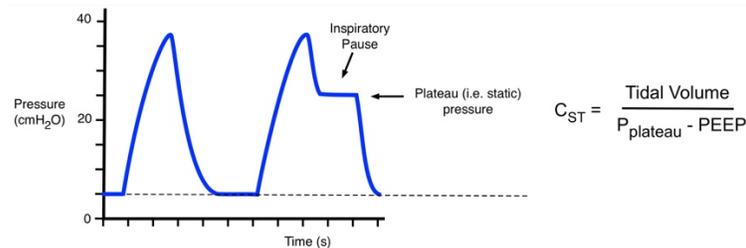
The peak inspiratory pressure (PIP) reflects how hard the ventilator must “work” to deliver a breath and is a function of three variables: (1) the inspiratory flow rate and flow pattern; (2) airway resistance (including the endotracheal tube and circuit); and (3) the compliance of the respiratory system. Although it can vary based on the delivered volume and the inspiratory flow rate and pattern, a normal value is typically < 40 cm H₂O. Values above this threshold indicate the patient has high airway resistance and/or low compliance.

2) What Are Common Factors That Increase Resistance and Decrease Compliance?

Increased Resistance	Decreased Compliance
Airway secretions	Abdominal distention
Bronchospasm	Pleural effusions
Kinks in circuit tubing	Pneumothorax
Patient biting on ET tube	Pulmonary edema / ARDS

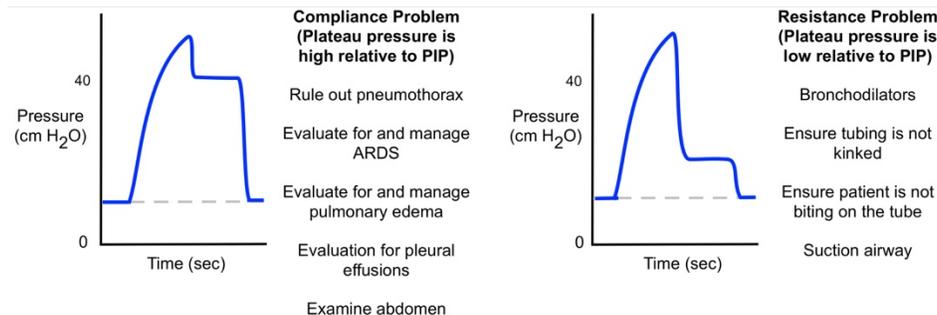
3) How Do I Distinguish Between Resistance and Compliance Issues on the Ventilator?

Perform an inspiratory pause maneuver. During the pause, there is no airflow and, therefore, resistance is no longer a factor. The pressure measured during the pause (referred to as the “plateau” or “static” pressure) is the pressure needed to keep the system open at that volume and reflects the compliance of the respiratory system. Static compliance (C_{ST}) is then calculated using the formula in the figure below. In healthy, non-intubated individuals C_{ST} is about 100 ml/cm H₂O. Values < 20 ml/cm H₂O are indicative of very low compliance.



4) Management

Management varies based on whether you have a primary resistance or a primary compliance problem. This can be identified by looking at the pressure vs. time curves



Call for help if unable to determine the cause or the plateau pressure > 30 ml/cm H₂O.

The Peak Inspiratory Pressure Has Increased... Now What Do I Do?

1) What Determines Peak Inspiratory Pressure?

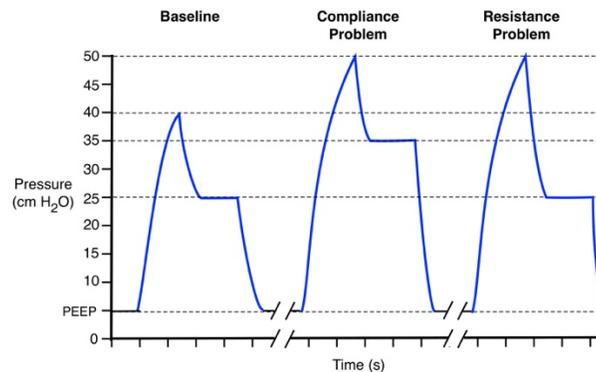
The peak inspiratory pressure (PIP) reflects how hard the ventilator must “work” to deliver a breath and is a function of three variables: (1) the inspiratory flow rate and flow pattern; (2) airway resistance (including the endotracheal tube and circuit); and (3) the compliance of the respiratory system. If the flow rate and flow pattern have not changed, any change in PIP is due to either a change in resistance or a change in compliance.

2) What Are Common Causes of Changes in Resistance or Compliance?

Increased Resistance	Decreased Compliance
Airway secretions	Abdominal distention
Bronchospasm	Pleural effusions
Kinks in circuit tubing	Pneumothorax
Patient biting on ET tube	Pulmonary edema / ARDS

3) How Do I Distinguish Between Resistance and Compliance Issues on the Ventilator?

Perform an inspiratory pause maneuver. During the pause, there is no airflow and, therefore, resistance is no longer a factor. The pressure measured during the pause (referred to as the “plateau” or “static” pressure) is the pressure needed to keep the system open at that volume and reflects the compliance of the respiratory system. In comparison to measurements done before the change in PIP, one of two situations can be seen:



4) Management

Resistance Problem: (Peak pressure increased, plateau pressure unchanged)

Suction the ET Tube

Bronchodilators

Check endotracheal and circuit tubing for kinks or patient biting on the tube

Compliance Problem: (Peak pressure and plateau pressure increased)

Sudden change: rule out pneumothorax with chest radiograph and/or chest ultrasound

Less sudden changes:

- Chest radiograph
- Review fluid balance
- Examine the abdomen. Consider bladder pressure measurement if tense

The Respiratory Therapist Said My Patient Has AutoPEEP... Now What Do I Do?

1) What Is AutoPEEP and Why Is It a Problem?

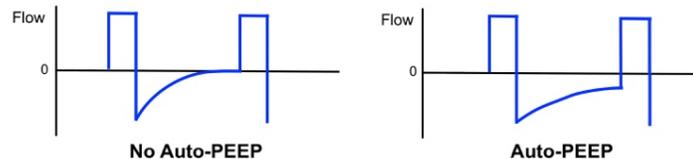
Under normal circumstances, the entire delivered tidal volume is expired during exhalation. If expiratory time is insufficient (see below), some portion of the previously delivered breath may remain in the lungs at the time the next breath is delivered. If this happens on a repeated basis, the lungs become hyperinflated. This can lead to increased intrathoracic pressure which decreases venous return and impairs cardiac output. In severe cases, people become hypotensive and can even go into pulseless electrical activity.

2) When to Look For This?

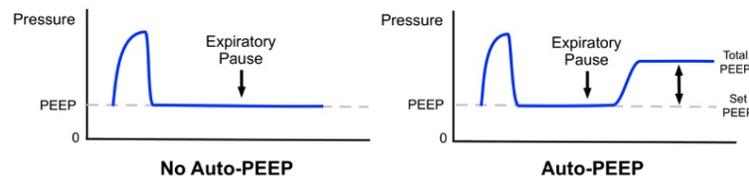
- Patients with obstructive lung disease (e.g., COPD, asthma)
- Patients requiring a very high respiratory rate to compensate for severe metabolic acidosis
- Patients who are spontaneously breathing at a very high rate
- Unexplained hypotension or cardiac arrest while on mechanical ventilation

3) How to Recognize AutoPEEP on The Ventilator?

Step 1: Check the flow vs. time curve. Flow should return to zero before the next breath. This tells you if auto-PEEP is present but does not quantify the magnitude.



Step 2: Perform an expiratory pause. The pressure should remain at the set PEEP. A rise in pressure confirms auto-PEEP. The bigger the increase, the more auto-PEEP.



4) Management

Initial Steps:

- Decrease minute ventilation by lowering rate and/or tidal volume (this is ineffective for patients breathing above the set rate on the ventilator)
- Increase the inspiratory flow rate; change to a square wave flow pattern
- Increase sedation
- Initiate bronchodilators and consider steroids for patients with asthma and COPD

Severe Cases:

Call for help!

Initiate neuromuscular blockade

Hypotension and/or bradycardia progressing toward PEA arrest: disconnect from ventilator (the loss of cardiac output is a greater risk than a transient cessation of ventilation). Readjust ventilator settings using the above-noted steps with reconnection.

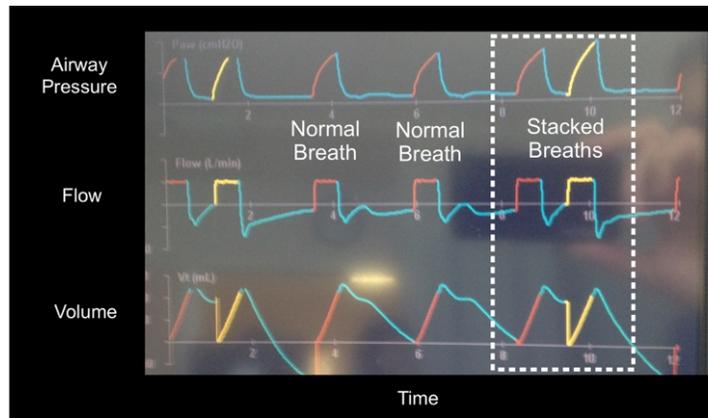
The Respiratory Therapist Said My Patient is Breath Stacking... Now What Do I Do?

1) What Is Breath Stacking and When Does It Occur?

In volume assist control, the ventilator will attempt to deliver a full breath (i.e., tidal volume) every time a patient starts to inhale. If the patient initiates a breath before full exhalation of the preceding one (due to their own intrinsic drive to breathe), the machine senses the inhalation effort and delivers another breath. This results in a larger than desired tidal volume and higher peak inspiratory pressures.

You will most commonly see this in patients receiving low tidal volumes as part of lung protective ventilation.

2) What Does It Look Like on the Ventilator?



3) Is This a Problem That Needs to Be Addressed?

It depends:

- It is not a problem if: Breath stacking is sporadic (e.g., once every 5 breaths) and/or the patient does not have severe oxygenation problems
- It is a problem if: Breath stacking is frequent (e.g., every breath or every other breath) and/or the patient has severe oxygenation problems. This needs to be addressed

4) A Step-wise Approach to Management

Step 1: Increase sedation with propofol and fentanyl to decrease respiratory drive

Step 2: Increase the inspiratory flow rate

Step 3: Add an end-inspiratory pause (0.25-0.3 second)

Call for Help if Steps 1-3 Do Not Fix the Problem

Step 4: Consider increasing tidal volume as long as the plateau pressure < 30 cm H₂O

Step 4: Initiate neuromuscular blockade

My Patient May Be Ready to Come Off the Ventilator... Now What Do I Do?

1) Assess Readiness for a Spontaneous Breathing Trial

Is the primary problem getting better?

Are they requiring an $F_{I}O_2 \leq 0.4$ and a PEEP ≤ 8 cm H₂O?

Is the minute ventilation ≤ 15 L/min?

If the answer to all questions is "Yes," proceed to the next step. If the answer is "No," continue volume assist control

2) Lighten Sedation (often referred to as Spontaneous Awakening Trial, SAT)

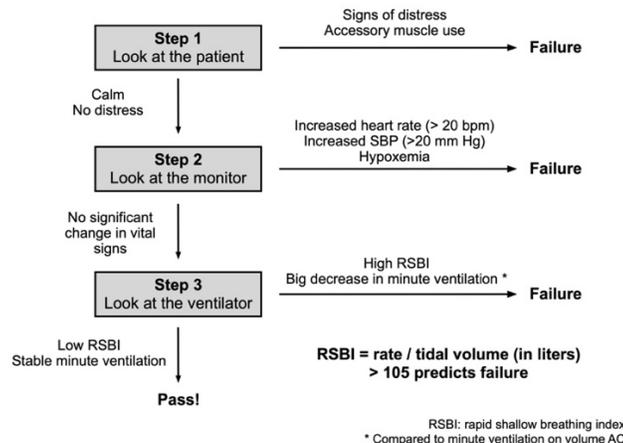
Turn propofol off. Some patients may require a low-dose of propofol due to anxiety around the time of the spontaneous breathing trial

3) Start Spontaneous Breathing Trial (SBT)

Place the patient on Pressure Support of 5 cm H₂O with a PEEP of 5 cm H₂O for 30 minutes

4) Assessing the Spontaneous Breathing Trial

Stepwise Approach to Assessing Patients on Spontaneous Breathing Trials



5) Assess Mental Status and Ability to Protect Airway

Normal mental status: Extubate patient

Altered mental status: Extubate patient if they have:

- A good cough
- A gag reflex
- No-to-minimal airway secretions

My Patient's Creatinine Rising or Urine Output is Decreasing... Now What Do I Do?

1) Gather more information about the issue by asking three questions:

- In the last 24 hours: has the creatinine risen to 1.5 times the baseline?
- In the last 24 hours: has the creatinine risen by >0.3 mg/dL?
- In the last 12 hours: has the *average* urine output been lower than 0.5 ml/kg/hr?

If “**Yes**” to any of the above, the patient has acute kidney injury; Go to Step 2 below
If “**No**” to all of the above, monitor serum creatinine, potassium, and magnesium daily.

2) Rule out post-obstructive nephropathy:

- Flush foley catheter and inspect for obstruction.
- Obtain bladder scan: If bladder is decompressed, post-obstructive nephropathy unlikely.
- If concern remains, obtain renal ultrasound to evaluate for hydronephrosis

3) If no evidence of post-obstructive nephropathy, pursue further evaluation

- Measure urine sodium and creatinine
- Obtain urinalysis with microscopy
- Calculate fractional excretion of sodium [$FE_{Na} = (\text{Urine Na}/\text{Serum Na})/(\text{Urine Cr}/\text{Serum Cr})$]

4) Evaluate and treat for pre-renal nephropathy:

Pre-renal etiology is likely with urine sodium < 10 and/or fractional excretion of sodium $< 1\%$
If present:

- Assess for hypovolemia and cardiac dysfunction
- Treat hypovolemia with fluid boluses; start with 1 liter lactated ringers in most patients
- Treat hypotension using the approach laid out in “My patient is hypotensive...”

5) Evaluate and treat for intrinsic nephropathy:

An intrinsic renal process is likely if urine sodium > 20 or fractional excretion of sodium $> 1\%$
or urine microscopy reveals muddy brown casts, granular casts, red or white cell casts

- Avoid repeated fluid boluses
- Review medication list for nephrotoxic medications
- Monitor potassium, magnesium, calcium, phosphate

Consult nephrology if:

- Severe or worsening metabolic acidosis (bicarbonate < 15 mEq/l)
- Hyperkalemia unresponsive to medical interventions (> 5.5 mEq/l)
- The patient is anuric
- Ongoing increases in creatinine and/or BUN

6) Work with the pharmacist to adjust the dose of medications based on the estimated glomerular filtration rate.

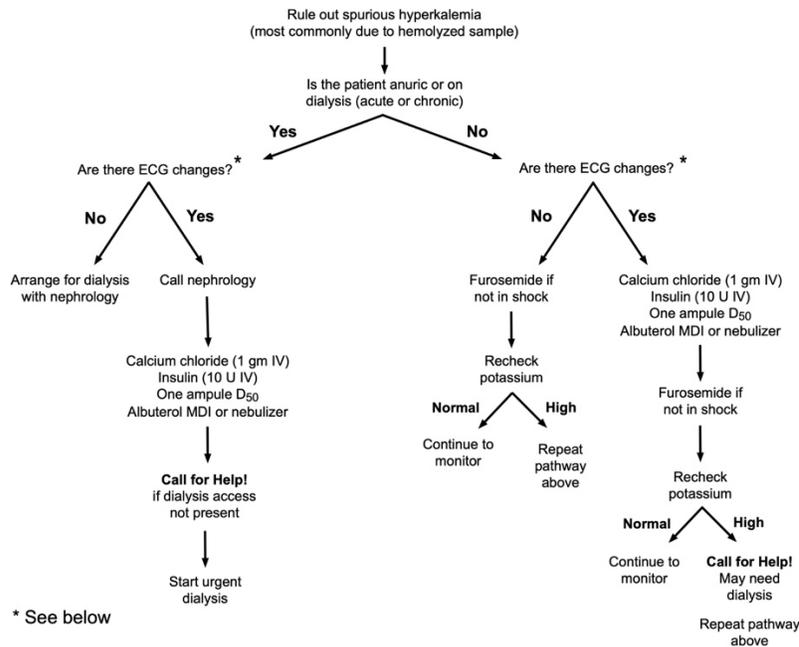
Critical Care Skills for Non-Critical Care Providers
My Patient's Renal Function is Getting Worse

My Patient is Hyperkalemic... Now What Do I Do?

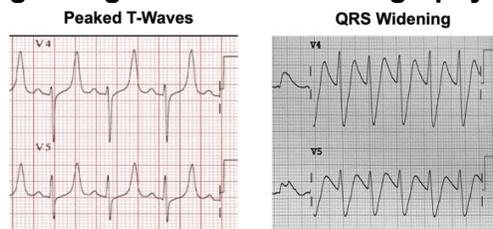
1) What Is a Normal Serum Potassium?

The normal range for serum potassium is 3.5 to 5.0 mEq/L. The higher the value above 5.0, the greater the risk of complications. Patients with chronic kidney disease can typically tolerate greater increases in serum potassium above normal than patients without chronic kidney disease. Most patients do not experience problems until the potassium rises above 6.0 mEq/L.

2) Follow the steps below:



3) What are the concerning changes on electrocardiography?



If the problem is not addressed, the patient can progress to a sinusoidal pattern followed by ventricular tachycardia or fibrillation

4) Identify and address contributing causes hyperkalemia

As you manage the problem, consider and address factors that may be leading to the rise in potassium:

- Acute kidney injury
- Cellular shifts due to metabolic acidosis
- Muscle breakdown in rhabdomyolysis
- Ischemic tissue injury
- Tumor lysis syndrome

“My Patient Has a Fever... Now What Do I Do?”

1) Perform a targeted history and exam to identify potential sites of infection

- Examine central lines, urinary catheter, drains for erythema, pus
- Intubated patients: assess for change in amount / quality of secretions, or worsening oxygenation
- Abdominal exam for focal tenderness or rigidity (pancreatitis, acalculous cholecystitis.)
- Assess surgical sites for purulence or erythema
- Assess for recent diarrhea
- Review medication list for sources of drug fever (e.g., antibiotics, anti-seizure meds)

2) Start work-up for infection:

- Blood cultures x 2 (peripheral and central)
- Chest radiograph
- Endotracheal aspirate if chest radiograph and clinical data suggestive of pneumonia
- Urinalysis with reflexive culture
- Consider liver panel (cholecystitis, drug reaction) and lipase (pancreatitis)
- If diarrhea, send stool for C. Diff PCR if patient has not recently received laxatives
- **Call for Help!**
 - If specific procedures indicated (paracentesis, thoracentesis)
 - CT scan necessary for further evaluation as this will mandate patient transport and create infection control issues.

3) Evaluate need for antibiotics:

Start broad spectrum empiric antibiotics if: hemodynamic instability, high clinical suspicion and no clear source: Cefepime and vancomycin

Start targeted antibiotics if a likely source is identified.

- HAP/VAP: vancomycin + either piperacillin/tazobactam, cefepime or meropenem. If recent MRSA swab negative, may hold vancomycin unless severely ill
- Central line: vancomycin + cefepime
- Intra-abdominal: piperacillin/tazobactam
- Catheter associated UTI: ceftazidime, change the catheter
- C-difficile: oral vancomycin. Add intravenous metronidazole for severe disease

If patient is already on antibiotics, **Call for Help!** with antibiotic selection.

4) Evaluate for non-infectious etiologies of fever:

Drug fever: rash, peripheral eosinophilia
Transfusion-related (can check a direct antiglobulin test)
Venous thromboembolism

5) Treat the fever

Acetaminophen 1000mg every six hours unless liver dysfunction. Avoid non-steroidals which may worsen COVID-19 and may contribute to worsening kidney injury.

My Patient Has Altered Mental Status... Now What Do I Do

1) Initial Assessment

- Check vital signs: assess for shock (hypotension), hypoxemia, or infection (fever)
- Stat fingerstick glucose: if hypoglycemic (FSG < 70), give 1 ampule of D₅₀
- Perform neurologic exam:
 - Focal neurologic findings: **Call for Help!** Consult neurology. Must weigh infection control risks of traveling for brain imaging
 - If history of seizures or exam concerning for non-convulsive status epilepticus (roving eyes): **Call for Help!** Consult neurology for electroencephalogram (EEG)
- Review medications (opiates, sedative/hypnotics), recent events/procedures:

2) Laboratory Studies

- Arterial blood gas to rule out hypercarbia
- Serum electrolytes to assess for hypo/hyponatremia, hypercalcemia, increased BUN
- Serum ammonia level in patients with cirrhosis
- Evaluate for infection with fever and/or leukocytosis:
 - Urinalysis with reflexive culture
 - Blood cultures x 2 (peripheral and central)
 - Chest radiograph
 - Consider respiratory viral panel / COVID-19 (if not already checked)
 - Lumbar puncture generally not indicated as risk of meningitis in hospitalized patients is low unless they underwent a neurosurgical procedure with violation of the dura

3) Consider Head Imaging

- Primary Indications: focal neurologic findings, increased risk of bleeding, sudden onset severe headache, or sudden change in level of consciousness
- Must weigh benefit of imaging with infection control risk of traveling for imaging

4) Management

- Assess ability to protect airway. **Call for Help!** Intubate if airway protection in question
- Suspected opiate overdose: naloxone 0.4 mg IV. May repeat up to 2 mg total
- Discontinue sedating medications
- Consider ventilatory support for patients with hypercarbia not related to opiates
- Address electrolyte disturbances
- Treat identified sources of infection

My Patient Has Delirium... Now What Do I Do?"

1) How Do I Recognize Delirium

- Symptoms range from a withdrawn state ("hypoactive delirium") to severe agitation ("hyperactive delirium"), with irritability, delusions, day-night reversal, and increased sympathetic activity (hypertension, tachycardia)
- ICU Nurses screen for delirium using a standardized bedside tool (**CAM-ICU**)

2) Evaluate for Contributing Factors

Delirium typically results from the combination of severe primary illness and the ICU environment.

Look for other contributing factors including:

- Untreated infection
- Hypoxemia
- Inadequately treated pain
- Altered day/night cycle and poor sleep
- Medications: Top culprits are benzodiazepines, antihistamines, antiemetics

3) Management

- Non-pharmacologic interventions focused on orientation and the environment
 - Frequent reorientation during the course of the day
 - Encourage visitation from family (as able based on infection control restrictions)
 - Lights on in the room during the day, off at night
 - Minimize noise and stimulation at night
 - Remove catheters and physical restraints when able
 - Mobilize and do physical and occupational therapy as able
 - Ensure patient has their glasses and/or hearing aids
 - Consistency in the nursing staff as able
 - Pharmacologic interventions
 - Stop benzodiazepines, anti-histamines other contributing medications, particularly those with anticholinergic side effects
 - Dexmedetomidine or propofol for sedation in intubated patients with target of alert or mildly sedated (RASS 0 to -1)
 - Consider pharmacologic intervention for sleep (e.g. trazodone, quetiapine) if non-pharmacologic measures are ineffective
- Severe agitated delirium: Call for Help!**
- As needed haloperidol (2.5-5 mg IV in repeated doses) if severe agitated delirium poses a risk of harm to the patient (e.g. discontinuation of lines or tubes) or the staff
 - May consider scheduled oral quetiapine if severe agitated delirium persists despite prn haloperidol

I Need To Protect My Patient From Harm In the ICU... Now What Do I Do?

Deep Venous Thrombosis (DVT) Prophylaxis

- A single form of prophylaxis is indicated in all critically ill patients
- Rely on pharmacologic over mechanical (sequential compression devices) unless contraindications to pharmacologic prophylaxis present (active bleeding, HIT, platelet <50k)
- Options for pharmacologic DVT prophylaxis:
 - Unfractionated heparin: usual dose 5000 U SQ Q8 hours
 - Low-molecular weight heparin (enoxaparin): usual dose 40 mg SQ daily
May be preferred for COVID-19 due to daily dosing
Contraindicated in eGFR < 30
- **Call for Help!** Consult the pharmacist for patients at the extremes of body mass

Stress Ulcer Prophylaxis

- Primary indications:
 - Intubated, not receiving enteral nutrition (discontinue once on full nutrition)
 - High risk for gastrointestinal bleeding (prior history, coagulopathy, high dose steroids)
- Primary options: proton pump inhibitor (PPI) or H₂ blocker
- If patient is on a PPI or H₂ blocker prior to admission, continue it

Other Harm Reduction Strategies

- Review indications for central venous catheters on a daily basis and whether they can be removed.
- Ventilator Associated Pneumonia (VAP) Prevention Bundle for all intubated patients
 - Semi-upright positioning (Head of bed >30°)
 - Oral hygiene with chlorhexidine
 - Careful monitoring of endotracheal tube cuff pressures
 - Daily assessment of readiness to extubate
- Review indications for foley catheter and remove as soon as able.
 - Invasive mechanical ventilation does not mandate placement of catheter
 - Risk of discontinuing catheter must be weighed vs. risk of skin break down and nursing care
- Review "ICU Checklist" every day on rounds to ensure these and other quality safety dashboard items are addressed.

Critical Care Skills for Non-Critical Care Providers Harm Reduction