Guide to supportive care in critical illness

November 8, 2018 by Josh Farkas

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High-quality supportive care consists of basic, daily management needed for any critically ill patient to prevent or surmount common problems. This isn't particularly flashy or exciting. However, it's essential for every patient passing through the intensive care unit. Minor interventions, when leveraged across thousands of patients, can have a substantial impact.

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This chapter provides an overview of issues that commonly arise in the daily practice of critical care. The goal is to rapidly provide a basic foundation of knowledge, especially for folks who don't work full-time in the ICU (e.g. residents rotating through the unit). All of these topics will be explored further in separate chapters, in much greater detail.
benzodiazepines, diphenhydramine, zolpidem

- These drugs are popular for treatment of insomnia or agitation. However, they promote delirium and should be avoided.
  - These medications will temporarily calm agitated patients, thereby appearing to work. However, they ultimately make patients worse by exacerbating their underlying delirium.
- In nearly all cases, better options exist:
  - For insomnia: melatonin, quetiapine, clonidine, perhaps trazodone.
  - For acute agitation: haloperidol, olanzapine, ketamine.
  - For sedation: dexmedetomidine, propofol, possibly quetiapine.
- Benzodiazepines should generally be restricted to the following situations:
  - Status epilepticus
  - Ketamine re-emergence, procedural sedation
  - Patients who are chronically on benzodiazepines as a home medication
  - Palliative sedation
  - Alcohol withdrawal (although phenobarbital is usually preferable here).

nephrotoxins

- The kidney is usually the first organ to be damaged by hypoperfusion. Kidney injury correlates strongly with increased mortality. The following medications should therefore be avoided whenever possible.
- NSAIDs should be avoided as a rule in the ICU (due to both nephrotoxicity and risk of GI bleeding).
  - Treatment of pain in critically ill patients is explored further below (#Analgesia & pain management). This does not involve NSAIDs.¹
- ACE-inhibitors & angiotensin-receptor blockers (ARBs)
  - ACEi/ARB generally shouldn't be initiated for control of blood pressure, except in anuric patients on chronic dialysis. For patients on ACEi/ARB solely for hypertension, consider holding these.
- ACEI/ARB should often be *continued* for patients with heart failure and reduced ejection fraction who are already on these medications chronically (but be careful).
- For patients admitted for heart failure, instead of initiating an ACEI/ARB consider using [hydralazine plus isosorbide dinitrate](https://emcrit.org/ibcc/chf/#rx_#2_-optimize_the_MAP) (similar physiologic effects with less nephrotoxicity).
- **Vancomycin** is often over-utilized. Vancomycin has no role for community-acquired abdominal or urinary sepsis. Linezolid may be preferable to vancomycin for MRSA pneumonia, especially among patients with tenuous renal function.

**Vancomycin**

![My extensive tramadol treatment decision algorithm](image)

**Tramadol**

- Tramadol is a weak opioid with a host of side effects (seizures, delirium, serotonin syndrome, hypoglycemia).
- The efficacy of tramadol is erratic, depending on genetic variation and interacting drugs.²
- Tramadol is sometimes promoted as a "non-opioid" but this isn't true. Tramadol is an opioid, it's just a really poor one.
- The only reason to consider using tramadol is if the patient is chronically on tramadol as a home medication.
- More on avoidance of tramadol from [First10EM](https://first10em.com/tramadol/) & [Tox & Hound](https://emcrit.org/toxhound/tramadol/).

**Fluoroquinolones**

- Over-utilization of fluoroquinolone has caused marked increases in antibiotic resistance.³⁴ Meanwhile, problems with clostroides difficile, MRSA, neuropathy, delirium, and tendinopathy have grown more apparent.⁵¹² The FDA has recently issued a series of [black box warnings](https://www.fda.gov/Drugs/DrugSafety/ucm511530.htm) on fluoroquinolones.
- Fluoroquinolones are almost never the best choice for critically ill patients. Previously fluoroquinolones were frequently used for treatment of pneumonia in patients with penicillin allergy, but newer evidence has shown that 3rd-4th generation cephalosporins are generally fine for such patients.¹³

**Aminoglycosides**

- There is no convincing evidence that double-coverage for gram-negative pathogens is beneficial (*even* for ventilator-associated pneumonia or pseudomonal infections – situations where double-coverage would seem most beneficial).¹⁴⁻¹⁹
- A single broad-spectrum beta-lactam is generally adequate. Addition of an aminoglycoside provides little additional coverage, but does increase nephrotoxicity.

**More Information**

- [Three reasons not to prescribe tramadol](http://empharmd.blogspot.com/2015/05/three-reasons-not-to-prescribe-tramadol.html)
- [Six reasons to avoid fluoroquinolones in the critically ill](https://emcrit.org/pulmcrit/fluoroquinolone-critical-illness/)
- [The siren's call: Double coverage for ventilator associated pneumonia](https://emcrit.org/pulmcrit/double-coverage-vap/)

**DVT prophylaxis**

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all patients should get DVT prophylaxis unless they have one of the following contraindications:

- (1) Hemorrhage
- (2) Thrombocytopenia (platelet count <30,000 or <50,000 and falling)\(^{20}\)
- (3) Planned procedure
  - DVT prophylaxis isn’t a significant problem for most ICU procedures, with the exception of lumbar puncture.
  - Interventional radiology may prefer to hold DVT prophylaxis; when in doubt this should be clarified with them in advance.
- (Note: Patients with cirrhosis and elevated INR are generally not coagulopathic and generally require DVT prophylaxis.\(^{21}\))

**DVT prophylaxis if GFR > 30 ml/min**

- Enoxaparin (low molecular-weight heparin) is the preferred agent for the following reasons:
  - Fewer injections than sq heparin, limiting patient discomfort.
  - Reduced risk of HITT compared to unfractionated heparin.
  - Enoxaparin may be more effective.\(^{22}\)
- Usual dose is 40 mg enoxaparin sq daily.
- Weight-based dose adjustments:
  - Weight <50 kg: decrease the dose to 30 mg sq daily.
  - Weight >120 kg: increase dose to 0.5 mg/kg enoxaparin q24hr.\(^{23-25}\)
- Monitoring anti-factor Xa levels
  - Consider for patients with unusual weight, pregnancy, or borderline renal function.
  - Check an anti-factor Xa level four hours after the third dose (target level ~0.3-0.5 IU/ml)

**DVT prophylaxis if GFR < 30 ml/min**

- Unfractionated heparin is used here, because it’s not cleared by the kidneys.
- Usual dose: 5,000 IU s.q. TID.
- Weight-based dose adjustments:
  - For patients weighing <50 kg, reduce the dose to 5,000 sq BID.
  - For patients weighing >120 kg, consider scaling the dose up roughly proportional to the patient's weight.

**GI prophylaxis**

**non-pharmacologic measures**

- For intubated/coagulopathic patients on aspirin for no good reason (e.g. “primary prevention”), please discontinue this.
- Always avoid NSAIDs in ICU patients.\(^1\)
- Enteral nutrition should be provided whenever possible (more on this below (#Critical_care_nutrition)).

**who needs pharmacologic GI prophylaxis?**

- Nobody really knows. The rate of stress ulceration has fallen with modern critical care practices (e.g. early enteral nutrition).\(^{26}\) Thus, the benefit of pharmacologic GI prophylaxis is probably restricted to a smaller group of patients with more risk factors.
- Our usual practice is currently to provide stress ulceration prophylaxis to the following groups of patients:
  - (1) Intubated patients
  - (2) Non-intubated patients only if they have numerous risk factors for stress ulceration (e.g. coagulopathy, profound uncontrolled shock, recent history of prior GI bleed, steroid therapy at a dose equivalent to >60 mg prednisone daily).\(^{27}\)

**what is the preferred agent for GI prophylaxis?**

- The pendulum keeps swinging back and forth between proton pump inhibitors versus H2-receptor blockers. Either one is fine.
- Currently pantoprazole 40 mg PO/IV daily may be preferable for the following reasons:
  - (1) Greater efficacy compared to H2-receptor blockers.\(^{28,29}\)
  - (2) Possibly lower risk of delirium compared to H2-receptor blockers.\(^{30,31}\)
• (3) Prior fears about increased risk of clostridiodes difficile or pneumonia have been debunked by new evidence from the SUP-ICU trial.

• Oral pantoprazole is cheaper than IV, so this is preferred if the patient is able to take enteral medications.

**more information**

- [SUP-ICU trial: The latest evidence on GI prophylaxis](https://emcrit.org/pulmcrit/sup-icu/).

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### Anemia & transfusion targets

The ICU hemoglobin drift

- Most patients in ICU will experience a gradual decrease in hemoglobin, which is caused by several factors.\(^3^3\)
  - 1) Suppression of hematopoiesis by critical illness.
  - 2) Phlebotomy from serial lab measurements.
  - 3) Subclinical loss from the GI tract (minor stress ulceration).

**approach to falling hemoglobin**

- **Evaluation**
  - *Acutely* falling hemoglobin can be caused only by bleeding or hemolysis (not reduced blood synthesis, which can cause only *chronic* anemia). Therefore, the investigation of acute anemia should focus on sources of blood loss and hemolysis. Checking iron labs, B12, and reticulocyte count is generally a waste of time.
  - Investigation may include: Repeat CBC, LDH to exclude hemolysis, ultrasonography to look for hemoperitoneum/hemothorax, CT angiography.

- **Therapy**
  - Conservative blood transfusion if needed (as below).
  - Avoid lab draws as able.
  - Treat any overt source of bleeding.
  - For patients with persistently dropping hemoglobin without any explanation, consider empiric proton pump inhibitor for therapy of probable stress ulceration.

**conservative transfusion strategy**

- Hemodynamically stable patients with anemia should be treated with a conservative transfusion strategy. Blood transfusion causes numerous complications (e.g. volume overload, transfusion-related acute lung injury, transfusion reactions). Conservative transfusion practices improve outcomes, in some cases even mortality.\(^3^4\)

- Blood transfusion should be given if the hemoglobin falls below a transfusion target.\(^3^5\)
For post-CABG patients and patients with active myocardial ischemia: transfuse if hemoglobin <8 mg/dL.

For everyone else (vast majority of patients): transfuse if hemoglobin <7 mg/dL.

Blood should generally be transfused one unit at a time (unless the patient is actively bleeding or the hemoglobin is definitely very low). Why? Two reasons:

- Hemoglobin bounces around due to random variation in the laboratory. Over-reacting to a low hemoglobin with two units of blood will often over-correct the anemia.
- Blood transfusion tends to cause pulmonary edema (more so than crystalloid). Two units of blood is a significant volume challenge for an ICU patient who will often have euvoicmic or hypervolemic anemia.

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**Glycemic control**

better sweet than sour

- Hypoglycemia may cause permanent brain damage and cardiopulmonary arrest. This is especially dangerous among intubated and sedated patients, who may develop severe hypoglycemia without any signs or symptoms (altered mental status will be masked by sedation).
- Hyperglycemia will not kill the patient. It’s controversial to what extent hyperglycemia may be dangerous. Hyperglycemia undoubtedly correlates with poor outcomes, but it’s unclear to what extent (if any) it causes harm.
- When in doubt, err on the side of avoiding hypoglycemia. Use insulin cautiously, paying attention to changes in steroid dosing and caloric intake (e.g. new NPO orders).
- ICU patients should receive occasional glucose checks to avoid occult hypoglycemia.
It's unknown when we should treat hyperglycemia. Enormous energy is spent achieving various glycemic targets, but these targets are arbitrary. No RCT has shown that treatment of hyperglycemia in critically ill patients improves outcomes (other than the Leuven 2001 study, which was subsequently debunked by VICEP and NICE-SUGAR). The most recent American College of Physicians guideline recommends a glucose target of 140-200 mg/dL (7.8-11.1 mM). Other authors have suggested 140-220 mg/dL (7.8-12.2 mM). Diabetic patients with chronic hyperglycemia may benefit from a greater degree of permissive hyperglycemia, because this is what they are used to. Among patients with hemoglobin A1C >7%, mild hyperglycemia may actually correlate with better outcomes. One pilot study demonstrated the safety of targeting a glucose level of 180-250 mg/dL (10-14 mM) among such patients.

Nutrition for the intubated patient

Benefits of enteral nutrition:
- Maintains gut integrity, preventing bacterial translocation into bloodstream.
- Prevents development of ileus.
- Reduces stress ulceration.
- Prevents malnutrition.

Patients should be fed unless there is a legitimate contraindication to feeding:
- GI catastrophe (obstruction, perforation, mesenteric ischemia, major upper GI bleeding).
- (Note: Pancreatitis is not a contraindication to enteral nutrition).
- When in doubt, start feeds at a low level and advance as tolerated.
- Absence of bowel sounds isn't a contraindication to feeding. There is little evidentiary support for listening to bowel sounds at all, so this is generally a waste of time.

Route of feeding
- Most intubated patients will initially have an orogastric (OG) tube placed. This can be used for feeding.
- A post-pyloric small-bore soft Kaofeed tube may be subsequently placed, for one of the following reasons.
  - (1) Post-pyloric feeding can be useful for patients with gastroparesis or vomiting.
  - (2) A smaller-bore nasal feeding tube is more comfortable and can be left in place longer (e.g. after the patient has been extubated). This may be useful in patients with hepatic encephalopathy, in whom it is important to maintain gut access following extubation (to allow administration of lactulose).
- The minor drawback of a small-bore tube is that it cannot be used to suction the stomach (e.g. to empty the stomach prior to extubation).

Initial nutrition orders
- Start with a standard 1 kCal/ml tube feed (e.g. Replete). Target a goal rate of roughly ~1 ml/hr/kg. For example, a 77 kg patient would receive 77 ml/hour. In morbid obesity use the ideal body weight.
- In severe renal failure, use a 2 kCal/ml renal tube feed (e.g. Novasource Renal) at a rate of 0.5 cc/kg/hour.
- The nutrition team will adjust your orders, but the most important aspect is to provide some enteral nutrition. The precise number of calories doesn't seem to have a huge impact – the key thing is providing a reasonable amount of nutrition.
**gastric residual volume**

- Recent nutrition guidelines recommend against checking gastric residual volume.\(^{43,47,48}\)
- Tube feeds shouldn't be held based on elevated gastric residual volumes. Feeding may be continued unless there are clinical signs of feeding intolerance (e.g. abdominal distension/discomfort, emesis).

**more information**

- [New guidelines simplify ICU nutrition](https://emcrit.org/pulmcrit/enteral-nutrition-intubated/)

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**Analgesia & pain management**

The best approach to pain is a structured, multi-modal strategy which uses several medications at low doses. Using lower doses avoids toxicity from each medication, while the use of several medications provides synergistic efficacy.

### #1 acetaminophen

- Useful agent, either as an adjunct or the sole therapy for minor pain.\(^9\)
- Most patients can receive up to 4 grams/day. It's often useful to schedule this (e.g. 1 gram q6hr) to keep ahead of pain.
- Patients with alcoholism or chronic liver disease can receive up to 2 grams/day.\(^{50}\) Acetaminophen is contraindicated in acute hepatic injury.

### #2 PRN opioids

- Often used as second line therapy, partially due to convenience.
- IV fentanyl works rapidly, but has a relatively short half-life so it may require frequent re-dosing.
- IV hydromorphone may be more effective, given its longer half-life. Note that hydromorphone is much more potent than morphine, so relatively low doses should be given (e.g. 0.2-0.4 mg IV).
- Oral opioids may be preferable for more stable patients with moderate pain who are able to take oral medications.

### #3 ketamine infusion +/- clonidine/dexmedetomidine

- Provides a mild-moderate amount of analgesia. Ketamine has been shown to reduce opioid requirements and decrease the incidence of nausea/vomiting.\(^{51-53}\) Ketamine may also attenuate the development of opioid tolerance and opioid-induced hyperalgesia, thereby making...
co-administered opioids safer and more effective.\textsuperscript{54–57}

- Ketamine is extraordinarily safe. Although sometimes feared due to its classification as an “anesthetic” agent, ketamine is actually the safest medication listed here (unlike acetaminophen or opioids, it’s nearly impossible to kill someone with ketamine).\textsuperscript{58,59}
- The dose is 0.1-0.3 mg/kg/hr. At the higher end of this dose range, some patients may experience mild psychotropic side-effects which are often pleasurable but can be scary. If psychomimetic side-effects occur, stop the infusion for 30-60 minutes and re-start at a lower dose (e.g. 0.1 mg/kg/hr). It’s generally possible to find a "sweet spot" where pain relief is achieved without side-effects.
- Dexmedetomidine or clonidine synergize with ketamine to improve analgesia.\textsuperscript{60–62} Dexmedetomidine or clonidine also prevent psychomimetic side-effects of ketamine, thereby further enhancing the safety of ketamine.\textsuperscript{63–68} Unfortunately, these are contraindicated among patients with bradycardia, heart block, or hypotension.

**#4 fentanyl infusion**

- Ideally avoided if possible, for many reasons. Over several days patients will rapidly become tolerant to opioid, which may subsequently cause withdrawal when fentanyl is discontinued.\textsuperscript{69} Fentanyl accumulates in fatty tissue, causing the drug to linger after the infusion is stopped, leading to delayed awakening.\textsuperscript{70}
- A 25 mcg/hr fentanyl infusion is equivalent to 120 mg oxycodone per day. Unless the patient has some extraordinary source of pain, 25-50 mcg/hr of fentanyl should be sufficient. High-dose fentanyl infusions can actually exacerbate pain, a phenomenon known as opioid-induced hyperalgesia.\textsuperscript{71}
- If a fentanyl infusion is used, attempts should be made on a regular basis to reduce the dose (at a minimum, every morning).

**make sure you’re targeting pain**

- Analgesics shouldn’t be used as nonspecific “calm down” medication for any agitated patient. Ideally, they should be targeted specifically at pain.
- For patients unable to communicate, a behavioral pain scale (e.g. CPOT (https://www.mdcalc.com/critical-care-pain-observation-tool-cpot)) can help determine whether the patient is in pain.

**more information**

- [Analgesic ladder for critically ill patients](https://emcrit.org/pulmcrit/analgesic-ladder/)
- [Why high-dose fentanyl infusions are evil](https://emcrit.org/pulmcrit/pulmcrit-fentanyl-infusions-sedation-opioid-pendulum-swings-astray/)

**Sedation of the intubated patient**

Intubated patients will generally require a sedative infusion. Propofol or dexmedetomidine are front-line choices, without any solid evidence that one is superior to the other. In practice, propofol is generally used initially. Dexmedetomidine is often useful once the patient is within a few days of extubation.

**propofol**

- Easily & rapidly titrated.
- May cause hypotension, but this can be counteracted with an infusion of low-dose phenylephrine (e.g. 0-80 mcg/min) or norepinephrine (e.g. 0-8 mcg/min).
- Use of high doses for prolonged periods of time may cause hypertriglyceridemia and a risk of propofol infusion syndrome. Over extended periods of time, it’s ideal to wean the dose down to <50 mcg/kg/min.

**dexmedetomidine**

- The major advantage of dexmedetomidine is that it doesn’t suppress respiration, making it safe to use in a non-intubated patient. Therefore, dexmedetomidine may be continued throughout the weaning process (unlike propofol, which must be shut off prior to extubation). This is an excellent option for patients who develop anxiety and tachypnea whenever sedation is lifted, making it difficult to extubate them.\textsuperscript{72}
- Dexmedetomidine may cause hypotension due to bradycardia, but this can be counteracted with an infusion of low-dose epinephrine if the use of dexmedetomidine is critical.
  - **Boluses of dexmedetomidine should be avoided**, as these can cause bradycardia in hemodynamic instability. Instead, the infusion can be started at a relatively high rate (e.g. 1-1.4 mcg/kg/min) without a bolus, and then down-titrated within 30-60 minutes.
Dexmedetomidine can cause tolerance over ~4-5 days, with subsequent withdrawal when it is discontinued. It may be inadvisable to continue dexmedetomidine infusions for longer periods of time. If tolerance occurs, dexmedetomidine may be transitioned to oral clonidine.

**sedating atypical antipsychotics**

- Olanzapine or quetiapine have sedating effects and efficacy in anxiety disorders. These agents may be used for their sedative properties, in order to allow a reduction in the dose of propofol or dexmedetomidine that is required.
- More information on antipsychotic pharmacology [here](https://emcrit.org/ibcc/delirium/#antipsychotics).

**benzodiazepines**

- Sedative of last resort, should be avoided whenever possible.
- May occasionally be needed for patients with profound hemodynamic instability, in whom propofol or dexmedetomidine are contraindicated.

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**Delirium prevention**

Delirium is extremely common in the ICU (prevalence of ~50%). This makes it worthwhile to deploy preventative measures broadly.

**general delirium prevention measures**

- Scheduled melatonin agonist before sleep (melatonin 3 mg or ramelteon 8 mg).
- Ear plugs and eye shades at night if tolerated.
- Avoid unnecessary sleep interruption (e.g. frequent blood pressure cuff monitoring, neurochecks).
- Use of glasses & hearing aids during the day.
- Aggressive physical therapy and early mobilization.

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**insomnia treatment strategy**

- Quetiapine 25-50 mg is often effective, but ideally should be given *early* in the night (to prevent somnolence the following day and day/night reversal).
- Dexmedetomidine infusion is an excellent option for insomnia and nocturnal agitation. The main limitations are logistic (this is an expensive intervention which cannot be used on the general ward).
  - If dexmedetomidine is used for insomnia or nocturnal agitation, it should be discontinued or aggressively down-titrated the following morning. Reserving dexmedetomidine for nocturnal use may promote preservation of a normal circadian rhythm.
  - Oral clonidine (0.2-0.3 mg) may be considered as an alternative to dexmedetomidine for mild insomnia.

**more information**

- IBCC chapter: [Delirium](https://emcrit.org/ibcc/delirium/)
Melatonin agonists for delirium prevention

Volume status & diuresis

**avoid volume overload**

- Critically ill patients will generally tend to retain fluid and become volume overloaded. Without attention to volume status, this may lead to profound volume overload. Numerous studies correlate positive fluid balance with mortality. 83,84
- Follow your patient's volume status carefully. If the patient is consistently gaining fluid or becoming >4-5 liters net positive, this is a problem which requires management.
  - Allowing patients to develop highly positive fluid balance (e.g. >6 liters positive) is a hallmark of poor-quality critical care.
- Avoid maintenance fluids. Most patients will receive plenty of fluid via medications, infusions, and enteral nutrition. 85 Maintenance fluid should be restricted unless there is a specific indication (e.g. rhabdomyolysis and diabetic ketoacidosis).
  - Exception: For healthy patients with normally functioning kidneys and heart (e.g. young intoxicated patients), maintenance fluids are reasonable. However, you should still follow input/output to ensure that the patient isn't retaining fluid (healthy patients will balance input/output on their own).

**don’t blindly chase low urine output or elevated lactate with fluid**

- It is quite uncommon for patients in the ICU to be truly volume depleted:
  - Most patients will be adequately fluid resuscitated in the emergency department.
  - Patients will usually retain fluid, so they will generally tend to develop fluid overload.
- Fluid should be given only if, after careful examination (often with ultrasound) and review of the clinical history, there is evidence of true volume depletion.
- Lactate elevation may occur for numerous reasons, most of which have little to do with volume status (e.g. albuterol or epinephrine administration). A rising lactate level calls for global re-evaluation of the patient, not knee-jerk fluid administration. It's doubtful that cycling lactate is beneficial anyway, because it usually doesn't reflect tissue perfusion.
- Don't check central venous pressure or use this to guide volume resuscitation. Central venous pressure doesn't correlate with volume status or fluid responsiveness. 86,87

**if you're going to give fluid, LR is generally superior to normal saline**

- LR is generally the fluid of choice in the ICU with the following two exceptions:
  - 1) Patients with elevated intracranial pressure (plasmalyte may be preferable).
  - 2) Patients with metformin-induced lactic acidosis (isotonic bicarbonate may be preferable).
- Don't be afraid to use LR in hyperkalemia, since it's a great fluid for hyperkalemic renal failure. 88–91
- Normal saline causes a dilutional acidosis (pH =5) and may also impair renal perfusion. 92,93 RCTs have demonstrated that NS may increase the risk of renal failure. 94,95
  - The use of normal saline should be restricted to patients with metabolic alkalosis.

**balanced diuresis**

https://emcrit.org/ibcc/guide/
It's not uncommon to encounter patients who are 5-10 liters positive, often due to over-resuscitation at an outside hospital. These patients will benefit from aggressive diuresis if tolerated.

- Use of furosemide alone tends to cause excretion of dilute urine, causing hypernatremia. This hypernatremia must then be treated by administration of free water, so the net result is ineffective diuresis.
- Addition of a thiazide diuretic (e.g. indapamide 2.5-5 mg PO daily) will enhance renal excretion of sodium, facilitating effective diuresis without the development of hypernatremia. For patients with severe bowel edema, IV thiazide may be needed (e.g. 500 mg chlorothiazide IV q12hr).
- If contraction alkalosis is developing and the patient remains hypervolemic, IV acetazolamide may be needed to prevent worsening alkalosis. For patients who are undergoing active diuresis, a reasonable dose is 500-1000 mg IV q12 hr.
- Follow electrolytes, with aggressive repletion of potassium and magnesium as needed.

Hypernatremia should never be tolerated, because this will cause the patient to be miserable (they will be profoundly thirsty yet unable to drink).

- Untreated hypernatremia is an under-recognized and under-treated cause of agitation among intubated patients. Too often it's treated with sedatives, rather than water.
- The vast majority of hypernatremia in the ICU is due to inadequate administration of free water. A diagnostic workup usually isn't needed (unless the patient has brain injury and there is concern for central diabetes insipidus).
- Hypernatremia should be treated by administration of free water (preferably via the gut, or otherwise in the form of intravenous D5W).
- Free water deficits are generally under-estimated. It's useful to calculate them (e.g. using MDCalc here).
- Don't just arbitrarily give some random amount of water – calculate exactly how much water is required to achieve the desired drop in serum sodium.
- For patients with chronic hypernatremia (stable elevation >48hr), target reducing the sodium by 12 mM per day. Don't worry about overshooting this a bit unless your patient is <30-40 years old (the risk of cerebral edema seems to be very low).
- If the sodium is creeping upwards, be pro-active in treatment of hypernatremia. If mild hypernatremia is ignored, it will generally get worse over time (hypernatremia usually represents a free water deficit, which cannot improve on its own).
- Don't treat hypernatremia with half-normal saline (this is an inefficient treatment of hypernatremia which will tend to cause sodium/volume overload).
- For patients with simultaneous volume overload plus hypernatremia, both processes should be treated simultaneously:
  - Furosemide plus high-dose thiazide may be used to promote sodium excretion (natriuresis; discussed in the section above).
  - Free water administration is provided to treat hypernatremia.
  - Follow electrolytes, with aggressive repletion of potassium & magnesium. Follow input/output balance and target a negative fluid balance.
- More on hypernatremia: see the full chapter [here](https://emcrit.org/ibcc/hypernatremia/).

**hyponatremia**

- Mild subacute or chronic hyponatremia is common in critical illness (e.g. sodium ranging 125-135 mEq/L). This doesn't require aggressive management or evaluation.

**hypokalemia**

- Traditional treatment with kayexalate is ineffective and shouldn't be utilized. The most effective treatment for hyperkalemia is usually kaliuresis: stimulation of potassium excretion by the kidneys using a combination of diuretics and fluids. However, patients with end-stage renal disease on chronic dialysis will simply need to be dialyzed.
- Diuretic:
  - Mild hyperkalemia: Furosemide alone may be fine.
  - Severe hyperkalemia: A more aggressive diuretic regimen may be indicated, especially for patients with severe hyperkalemia who will require urgent dialysis if they don't respond. A maximally aggressive diuretic combination is the nephron bomb which consists of 160 mg IV furosemide, 500-1000 mg IV chlorothiazide, and 500-1000 mg IV acetazolamide.
- Fluid:
  - The goal here is to bring the patient to a point of euvolemia and keep them there. Patients who produce copious amounts of urine in response to diuresis should have this fluid volume replaced.
  - For patients with significant metabolic acidosis (serum bicarbonate <20 mEq/L), the preferred fluid is isotonic bicarbonate (D5W plus three 50-mEq ampules of bicarbonate to generate a 150 mEq/L solution of sodium bicarbonate).
  - For patients without metabolic acidosis, the preferred fluid is lactated ringers, plasmalyte, or normosol.
  - Normal saline is contraindicated in hyperkalemia, because it will worsen it.
- Fludrocortisone (0.2 mg PO) may be useful to promote potassium excretion, particularly among patients taking ACE-inhibitors or ARBs.
- More in the chapter on hyperkalemia [here](https://emcrit.org/ibcc/hyperkalemia/).
Hypokalemia isn’t as dangerous as commonly perceived (mild hypokalemia is generally well tolerated in the absence of digoxin use or hypomagnesemia).

When possible, potassium should be repleted via the gut. Administration of enteral potassium is easier, cheaper, and safer than IV potassium. It’s hard to kill someone with enteral potassium, whereas this isn’t necessarily that difficult with IV potassium.

Check the magnesium level and replete if necessary. Hypokalemia and hypomagnesemia often coexist. Hypomagnesemia will cause ongoing potassium wasting, so successful repletion of potassium may depend on fixing the magnesium as well.

Be very cautious about treating hypokalemia in patients with severe renal failure:
- The target potassium might be around 3-3.5 mEq/L in this scenario (don’t shoot for a stone-cold “normal” potassium level; the potassium level will rise over time).
- Check and replete the magnesium level (target >2 mg/dL), as this will protect against Torsade de pointes and increase the safety margin for mild hypokalemia.
- Discontinue PRN potassium orders.

**Hypomagnesemia**
- Common in critically ill patients.
- Should be treated with IV magnesium sulfate (oral magnesium causes diarrhea and is ineffective at improving serum magnesium levels).
- Patients with total body magnesium depletion (e.g. alcoholics) may require ongoing therapy to achieve eumagnesemia.

**Hypocalcemia**
- Critically ill patients are very commonly hypocalcemic. This is an epiphenomenon of critical illness, which doesn’t seem to actually cause harm (with certain exceptions, most notably massive transfusion).
- Treatment of mild hypocalcemia is generally ineffective and might actually be harmful.
- Unless the calcium is very low (e.g. ionized calcium < 0.7 mM) and/or the patient seems to be demonstrating symptoms of hypocalcemia, it’s generally best *not* to administer calcium.
- Overall, it’s probably best not to check calcium levels, with the exception of admission labs or a specific reason to expect hypocalcemia (e.g. massive transfusion protocol).

**MI & ischemia evaluation in the non-cardiac patient**

This section describes the approach to troponin elevation in the “non-cardiac” patient – that is, a patient admitted to the ICU for a non-cardiac problem (e.g. septic shock).
do not check troponin routinely in all ICU patients

- About ~40% of ICU patients will have troponin elevation, but the vast majority of these patients are not having a plaque-rupture (type-I) MI.\(^99\)
- EKGs should be routinely obtained on all ICU patients (if nothing else, this serves as a baseline for comparison throughout the patient’s hospital stay).
- Troponin should be checked only if there is a genuine clinical suspicion for myocardial infarction based on the EKG and clinical presentation.

### Sorting out three categories of troponin elevation

- Unfortunately, troponin levels are often obtained when they aren't actually indicated. When this occurs, we need to sort out what is causing the troponin elevation.
- A key concept here is the **universal definition of a myocardial infarction**, which requires:\(^100\)
  1. Dynamic rise/fall in troponin levels
  2. At least one of the following: clinical history suggests MI (e.g. anginal chest pain), new ischemic EKG changes, or new wall motion abnormality on echocardiography.

Using this definition, troponin elevation can be sorted into three categories:

- **Non-MI troponin elevation**
  - These patients have troponin elevation without other features of MI (they don't meet the definition of MI above).
  - This is extremely common among critically ill patients and requires no specific treatment. Treatment should focus on management of the underlying disease.
  - There is no therapeutic benefit to ongoing cycling of the troponin in this situation.

- **Type-I MI (plaque rupture MI)**
  - These patients have classic MI due to plaque rupture. Most patients with type-I MI will initially present to the hospital with cardiac-related signs or symptoms. Type-I MI can arise as a complication of critical illness, but this is rare.
  - Type-I MI should be treated with the typical treatment regimen generally associated with MI if possible (e.g., aspirin, P2Y12 inhibitor, possibly cardiac catheterization). However, the ability to provide such treatments is often limited by bleeding, hypotension, and acute kidney injury.

- **Type-II MI (demand MI)**
  - These patients have MI due to a fixed coronary lesion plus physiologic stress (figure above).
  - Sorting out type-I vs. type-II MI can be tricky (the table below may help guide this). When in doubt, urgent echocardiography can be helpful.
  - The treatment for type-II MI focuses on treatment of the underlying disease (e.g. anemia, sepsis, hypoxemia). Aspirin is reasonable, but otherwise these patients don't require specific treatment directed towards plaque stabilization.
use a heparin infusion only if truly indicated

- Troponin elevations are scary and we may feel compelled to do something. However, initiating a heparin infusion is rarely wise here. Heparin is indicated only if both of the following two conditions are met:
  - (1) The patient has a type-I (plaque-rupture) MI. This can occur in patients who present with non-cardiac diagnoses, but it's uncommon.
  - (2) There is a plan to take the patient for urgent/emergent cardiac catheterization.
    - The traditional practice of using a 48-hour heparin infusion for "medical management" of MI without PCI is not evidence-based. The only clear role for a heparin infusion is to stabilize plaque as a bridge to cardiac catheterization.

more information

- IBCC chapter on troponin & MI evaluation (https://emcrit.org/ibcc/troponin/)

Noninvasive respiratory support (HFNC & BiPAP)

Approach to selecting mode of respiratory support

1. Needs immediate intubation?
   - Yes
     - Intubate
     - Note: consider BiPAP trial for COPD/catech or flash pule edema even if patient looks bad
   - No

2. Contraindication to BiPAP?
   - Significant secretions
   - Risk of aspiration
   - Facial abnormality/trauma
   - Yes
     - HFNC (although chest drain or Heliq may also be used, per below algorithm)
   - No

3. Probable / definite diagnosis?
   - Yes
     - HFNC
   - No

4. Heart failure
   - Small airway obstruction (COPD, Asthma)
   - Diaphragm failure
     - Obesity hypoventilation syndrome
     - Neuromuscular weakness (ALS, MG, etc.)
   - BIPAP
   - BIPAP + sedation

5. Pneumonia
   - Interstitial lung disease
   - ARDS
   - HFNC

6. Pleural disease
   - Pleural effusion
   - Pneumothorax
   - HFNC

7. Upper airway obstruction, e.g.:
   - Post-extubation laryngeal edema
   - Undifferentiated stridor
   - Drain
     - HFNC

8. CNS cause of low respiratory drive
   - Drug intoxication, encephalitis, etc.
   - Antidote (esp. narcan)
   - HFNC

https://emcrit.org/ibcc/guide/
• Contraindications to BiPAP
  - Secretions (BiPAP generally impairs expectoration)
  - Aspiration risk, altered mental status
• Diseases that are exquisitely BiPAP-responsive
  - COPD, Asthma
  - Cardiogenic pulmonary edema
  - Obesity hypoventilation syndrome
• Sedation to facilitate BiPAP tolerance
  - Generally worth trying for patients with COPD/asthma or heart failure who have difficulty tolerating BiPAP.
  - Dexmedetomidine is ideal, given its titratability and lack of respiratory suppression.
  - Avoid benzodiazepines (they occasionally work, but often cause patients to be agitated/confused thereby worsening the situation).
  - Some reasonable options may include: haloperidol, ketamine, or carefully titrated fentanyl for patients with severe air hunger.

**how to titrate BiPAP**

• Simply sticking the patient on BiPAP isn't enough – you need to titrate settings to obtain optimal benefit. The FiO2 may simply be titrated against the patient's oxygen saturation. The tricky part is adjusting the inspiratory and expiratory pressures.

• **Cardiogenic pulmonary edema**
  - Key here is the expiratory pressure (this increases the mean airway pressure and thereby decreases preload & afterload).
  - Ramp up expiratory pressure until the patient improves (e.g. 10/5 ==> 15/10 ==> 18/15).¹⁰⁴

• **Everything else**
  - The key here is driving pressure (inspiratory pressure minus expiratory pressure), which supports the work of breathing.
  - Ramp up the driving pressure until the patient improves (e.g. 10/5 ==> 15/5 ==> 18/5).

**when to choose HFNC**

• Any patient with significant respiratory distress who doesn't have specific indications for BiPAP (above).
  - The most common indication is pneumonia.¹⁰⁵
• Can be used in situations where BiPAP is preferred, yet patients cannot tolerate BiPAP (e.g. COPD, pulmonary edema).
• HFNC is the safest mode of noninvasive support, so when in doubt this is a reasonable choice.

**how to titrate HFNC**

• FiO2 is titrated against the patient's oxygen saturation.
• Initially when the patient is in distress, flow rate should be increased as high as tolerated, usually 50-60 liters/minute (this provides the maximal amount of ventilatory and PEEP support).
• After the patient is improving, flow rate can be weaned down. If there are signs of tachypnea or dyspnea, this may indicate that the patient isn't ready for weaning.

**post-extubation prophylactic HFNC**

• Extubation directly to HFNC has been shown to reduce the risk of reintubation, even in patients who are low risk for reintubation.¹⁰⁶,¹⁰⁷
• With the exception of patients intubated very briefly for procedures, post-extubation HFNC is worthwhile.
• For maximal benefit, HFNC should be increased to the highest flow rate tolerated (ideally 50-60 liters/min) and continued for ~24 hours.
• The mechanism of action seems to be that HFNC reduces the work of breathing, thereby preventing patients from developing respiratory fatigue. In order for this to be effective, it should be started promptly following extubation (not in a delayed fashion after patients develop severe dyspnea).

**more information**

- [Dark art of noninvasive ventilation (HFNC vs. BiPAP)](https://emcrit.org/pulmcrit/bipap-hfnc/)
- BiPAP
  - [Dexmedetomidine to facilitate BiPAP](https://emcrit.org/pulmcrit/dexmedetomidine-to-facilitate-noninvasive-ventilation/)
- HFNC
  - [HFNC in immunocompromised patients (HIGH trial)](https://emcrit.org/pulmcrit/pulmcrit-does-the-high-trial-debunk-high-flow-nasal-cannula/)

https://emcrit.org/ibcc/guide/
Respiratory monitoring (including ABG, VBG and etCO2)

The best way to monitor oxygenation is pulse oximetry. Pulse oximetry is preferred over ABG monitoring because pulse oximetry directly measures oxygen saturation (whereas portable ABG meters calculate oxygen saturation).

**Oxygen saturation** (not pO2) is the most important variable because this most directly measures oxygen delivery to the body's tissues.

Shooting for a target oxygen saturation of ~90-96% is generally optimal, for a few reasons:

- Excessive amounts of oxygen (hyperoxia) can be dangerous, especially following cardiac arrest.
- If an unnecessarily high amount of oxygen is used, this will mask early clinical deterioration (the patient will need to worsen a lot before they desaturate).
- For patients with chronic hyperapneic respiratory failure (e.g. COPD), target a saturation of 88-92%.

**Lower pO2 in the lungs may improve ventilation/perfusion matching, thereby improving removal of CO2.**

**ventilation: what are we targeting ??**

- Honestly, nobody really knows. This implies that we shouldn't be dogmatic or obsessive about adjusting/monitoring ventilation.
- Upper safe limit for pCO2 ??
  - Hypercapnia is generally tolerated extremely well (for example, there are COPD patients living in the community right now with a pCO2 >80 mm).
  - **Permissive hypercapnia** refers to the concept that allowing some hypercapnia is fine. Among patients with COPD/asthma or ARDS, some hypercapnia is preferred because this facilitates lung-protective mechanical ventilation.
  - The lower limit of pH below which hypercapnia becomes harmful is unknown. pH >7.2 is definitely fine, and pH >7.1 is probably fine as long as hemodynamically tolerated. There doesn't appear to be any specific pH cutoff below which the patient will turn into a pumpkin.
- **Lower safe limit of pCO2 ??**
  - Respiratory alkalosis (hypocapnia) may be less well tolerated than hypercapnia. There is a potential risk of seizure at pH >7.5
- **So:** for most patients we're targeting a pH of roughly 7.2-7.5
  - The target range of pCO2 will depend on the bicarbonate (using the **Henderson-Hasselbach equation** [http://ebmcalc.com/HendersonHasselbach.htm]).
  - For example, for a patient with bicarbonate of 24, a safe pCO2 would be 30-65 mm.
  - For patients with elevated intracranial pressure, pregnancy, or severe pulmonary hypertension it might be safer to target normocarbia (pCO2 35-45 mm). More attention will be needed to blood gas parameters in these patients.

**when to check ABG/VBG?**

Overall these are checked far too often, with the vast majority of results having minimal/no effect on clinical management.

ABG/VBG values generally provide no diagnostic information regarding why a patient is in respiratory failure (with the exception of hyperventilation due to anxiety). Checking a blood gas for a patient in respiratory distress of unknown cause is unlikely to yield a diagnosis (whereas tests such as chest X-ray and lung ultrasound are far more likely to help).

ABG/VBG occasionally can reveal the presence of hypercapnic encephalopathy in a somnolent patient with no obvious respiratory compromise. Such patients generally have a history of chronic hypercapnia (e.g. severe COPD or obesity hypoventilation syndrome).

ABG/VBG can be helpful to monitor ventilation, especially among patients who are intubated or on BiPAP/HFNC. However, this is only one piece of information which should be carefully integrated with the overall clinical picture (e.g. the patient's overall appearance is generally more important than blood gas values).

**VBG vs. ABG?**

- For most patients oxygenation can be measured with pulse oximetry. Therefore, the only information we get from VBG/ABG is the pH and pCO2. If we already know the patient's bicarbonate from serum chemistries, then VBG/ABG provides only one independent piece of information (because the pCO2 will determine the pH based on the Henderson-Hasselbach equation).

- VBG values are extremely close to ABG values as long as the oxygen saturation from the venous blood gas isn't extremely low (VBG oxygen saturation >75%)
  - Most patients have a peripheral vein cannula that allows blood removal. A small amount of blood can be removed, allowing for painless and accurate monitoring of blood gas parameters. If venous blood is obtained without a tourniquet and analyzed immediately, it will generally have a saturation >75%, yielding accurate information.

- If VBG oxygen saturation is very low (<50-60%), then pH and pCO2 values will be off slightly. However, they won't be far off and the direction of deviation is predictable:
  - VBG pCO2 > ABG pCO2
  - VBG pH < ABG pH

- Clinical management should never hinge on tiny differences in ABG/VBG parameters. Therefore, it's generally fine to use VBG parameters. Remember: the decision to intubate is based on clinical judgement, not numbers.

**end tidal CO2 (etCO2)**

- For intubated patients, etCO2 can be used to estimate and trend the patient's arterial pCO2.

- etCO2 will always underestimate blood pCO2, because gas in the trachea dilutes CO2 as it travels from the alveoli to the ventilator.
  - If the etCO2 is >45 mm, then the patient is definitely hypercapnic.

- The gap between etCO2 and pCO2 depends on how healthy the lungs are:
  - In healthy lungs, this gap is usually <15 mm.
  - In patients with lung disease (e.g. COPD, pneumonia, ARDS, PE, pulmonary contusion) the gap will widen.

- For most patients, after intubation the respiratory rate should be adjusted to achieve an etCO2 of ~30 mm. This should put the pCO2 in a safe range (~35-65 mm).
  - One exception would be in patients with severe metabolic acidosis, in whom the respiratory rate should initially be maximized in efforts to provide a compensatory respiratory alkalosis.

**minute ventilation**

- Minute ventilation is the volume of gas moving in/out of the lungs every minute. A normal minute ventilation is ~6-7 liters/min.

- Patients with ineffective ventilation (ventilation/perfusion mismatch, for example, due to COPD or ARDS) will need a higher minute ventilation to achieve a normal pCO2 level (e.g. 10-14 liters/minute).

- Minute ventilation is displayed on the screen of the ventilator of BiPAP machine. For patients on BiPAP, minute ventilation is accurate only in the absence of an air leak around the mask.

- Paying attention to minute ventilation can be extremely helpful, especially for patients on BiPAP. For example:
  - A COPD patient with minute ventilation of >9 liters/min: this patient is probably ventilating OK.
  - A COPD patient with minute ventilation <6 liters/min: this patient is probably hypercapnic.
Oxygenation monitoring: pulse oximetry is preferred. https://emcrit.org/pulmcrit/pulse-oximetry/

How to convert a VBG into an ABG https://emcrit.org/pulmcrit/vbg-abg/

ABG unhelpful in diagnosing cause of respiratory failure https://emcrit.org/pulmcrit/approaching-undifferentiated-cardiopulmonary-failure-which-tests-are-most-useful/

Approach to fever in the ICU

common causes

- Infection (~50% of cases)
  - Pneumonia
  - Clostridiodes difficile, Acalculous cholecystitis
  - Line infection
  - Surgical site infection

- Non-infectious (~50% of cases)
  - Procedure-related (hemodialysis, bronchoscopy, 1-3 days post-surgery)
  - Drug fever
  - Febrile transfusion reaction
  - Sterile inflammation (pancreatitis, aspiration pneumonitis, ARDS)
  - Pulmonary embolism
  - Alcohol withdrawal
  - Central fever (intracranial hemorrhage)

Exam for febrile ICU patient

- Physical examination as above.
- Chest X-ray if intubated or symptoms of pneumonia.
- Blood cultures
  - At least two, with at least one peripheral culture.
  - Any line in place >48 hours should be cultured.
- Clostridiodes difficile testing if diarrhea.
- Further testing per clinical judgement (e.g. abdominal CT scan, CT angio for PE).
test to usually avoid

- **Urinalysis & culture from foley catheter**: In the absence of urinary obstruction or recent urinary tract manipulation, cystitis is rarely the cause of new-onset fever. Routinely culturing urine in all febrile ICU patients will tend to cause false-positive diagnoses of urinary tract infection.
- **Tracheal aspirate cultures**: These will generally be positive due to colonization in anyone with COPD, cystic fibrosis, or prolonged intubation. Obtain sputum cultures only if there is a genuine clinical suspicion for pneumonia (e.g. based on increased sputum production, hypoxemia, or chest X-ray infiltrates).

**avoid antipyretics (acetaminophen)**

- It's generally best to avoid antipyretics, as these will obscure the true fever curve.
- **Indications for antipyretics**:
  - Neurologic injury (e.g. stroke, anoxic brain injury)
  - Severe fever (e.g. >40°C/104°F)
  - Fever appears to cause clinical deterioration (fever can cause some patients to become delirious or tachycardic)

**avoid empiric antibiotics**

- Fever isn't an indication for antibiotics.
- Antibiotics may be indicated in specific situations:
  - Neutropenic fever (absolute neutrophil count <500 -or- <1,000 and falling)
  - Septic shock
  - High index of suspicion for a specific infection (e.g. clostridiodes difficile) – antibiotics may be started while awaiting test results.

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**Questions & discussion**

To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/guide/).

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**References**


https://emcrit.org/ibcc/guide/


