Acute exacerbation of COPD (AECOPD)

May 19, 2020 by Josh Farkas

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diagnosis & workup
### Differential Diagnosis

Patients with a history of COPD frequently present to the hospital with dyspnea. Most of them have AECOPD, but some don’t. The following are common differential diagnoses that should be considered, together with key diagnostic findings:

- **Pneumonia.** Key findings = consolidation on ultrasonography, infiltrate on CXR.
- **Pulmonary embolism (PE).** Key findings = lack of usual features of AECOPD (no sputum, no fever, etc.).
- **Pulmonary edema.** Key findings = bilateral B-lines on ultrasonography, signs of congestion on CXR.
- **Pneumothorax.** Key findings = loss of lung slide on ultrasonography, pneumothorax on CXR.
- **Upper airway obstruction** (e.g., tumor, angioedema, epiglottitis, vocal cord dysfunction). Key findings = wheeze/stridor grossly audible without stethoscope.
- **Obesity hypoventilation syndrome:** patients may present with hypercapnic respiratory failure, but without other findings of COPD (wheezing, sputum).
- **Opioid/sedative effects:** among COPD patients who are on chronic opioids, it can be extremely difficult sorting out whether hypercapnia is a medication side-effect or is due to underlying COPD. Ideally, chronic opioids should be avoided in these patients, unless the patient is on a comfort-directed plan of care (DNR/DNI).

### Basic Diagnostic Workup

- **H&P**
  - The holy trinity:
    - Ultrasonographic examination of heart & lungs
    - CXR
    - EKG
  - Basic labs (CBC, electrolytes)
  - Influenza PCR during flu season
- **ABG/VBG?**
  - Generally unhelpful and unnecessary.
  - Compared to ABG, VBG is generally fine and more humane. VBG may be obtained simultaneously with other labs.
  - ABG/VBG doesn’t generally help diagnose AECOPD or differentiate it from other diagnoses (Burri 2011). ABG/VBG is helpful in the somnolent patient, to determine whether somnolence is caused by hypercapnia.
  - Sputum GS/Cx is not helpful (discussed on section below regarding antibiotics).

### AECOPD vs. PNA

- Hardest differential diagnosis to sort out (both may cause fever, chills, purulent sputum, and leukocytosis).
- Key differentiating factor is presence/absence of infiltrate. Unfortunately, chest x-ray isn’t 100% sensitive for pneumonia. In cases which are hard to tease apart, options include:
  - Chest CT scan (although it is generally not worth getting a scan solely for this reason).
  - Procalcitonin (if <0.5 ng/ml, this argues strongly against typical bacterial pneumonia).
- AECOPD and pneumonia often occur together (“pneumonic AECOPD” – the pneumonia is causing a COPD exacerbation). Treatment of pneumonic AECOPD consists of treating both pneumonia and COPD.
- One potential approach to a patient with COPD and possible pneumonia is the following:
  - (1) Start on antibiotic coverage for pneumonia (e.g. ceftriaxone plus azithromycin) and check a procalcitonin.
  - (2) If procalcitonin is low (<0.5 ng/ml), this argues against typical bacterial pneumonia. Ceftriaxone can be discontinued, while azithromycin is continued for treatment of COPD.
  - (3) If procalcitonin is elevated, then continue combination antibiotic therapy for pneumonia (along with full-bore COPD therapy as well – the presence of PNA doesn’t exclude concomitant COPD).

### AECOPD vs. PE

https://emcrit.org/ibcc/AECOPD/
PE is found in a small, but significant fraction of patients who present with possible AECOPD (~10%). PE should be suspected in patients whose presentation is atypical for a COPD exacerbation (e.g. lack of purulent sputum, fever, chills). For atypical AECOPD presentations, it is sensible to evaluate for PE. COPD patients are at low risk of harm due to contrasted CT scans (because their age makes radiation a nonissue and contrast dye doesn’t cause renal failure). The main risk of a CT scan is finding an incidental lung nodule which will trigger a cascade of iatrogenic harm.

Patients with COPD and anxiety may fall into a cycle shown above with progressive anxiety, tachypnea, dyspnea, and gas trapping. This may cause patients to deteriorate very rapidly, but improve rapidly as well. A combination of BiPAP and anxiolytics may be very helpful in breaking patients out of an episode.

One of the central problems in AECOPD is exhaustion of the diaphragm. After working hard for a prolonged period of time, the diaphragm becomes fatigued. Diaphragmatic fatigue may require 24-48 hours of rest to recover. Probably one of the key roles of BiPAP or intubation is to rest the diaphragm. Take-home messages based on this concept:

- Even if the patient recovers well after a few hours on BiPAP, it may still be worthwhile to leave the BiPAP on longer (e.g. overnight) to rest the diaphragm.
- If a patient gets intubated for COPD, it is generally wise to leave them intubated for at least ~1 day before they are extubated (assuming that they truly required intubation in the first place).

Steroid

- There is no precise evidence on how to dose steroid for COPD patients in the ICU. The following is a reasonable approach:
  - (#1) Start with 125 mg IV methylprednisolone in the emergency department.
  - (#2) If the patient remains on the verge of requiring intubation, then continue methylprednisolone 125 mg IV daily. Otherwise, proceed to...
  - (#3) Prednisone 40-60 mg daily in the morning for a few days, then taper further.
- For patients who are improving and not at imminent risk of deterioration, don’t continue high steroid doses (e.g. 60 mg methylprednisolone IV Q6, which is equal to 300 mg/day of prednisone!).

Bronchodilators

- Acutely ill patients are usually too breathless to take their home medications (metered-dose inhalers, etc.). Hold all home inhalers.
The following regimen of bronchodilators is adequate:

- Albuterol plus ipratropium nebulized Q6hr scheduled.
- Albuterol nebulized Q2hr PRN.
- For patients on BiPAP or HFNC, bronchodilators can be nebulized and administered in-line through the device (without having to remove the patient from support).

antibiotics

- Patients sick enough to be in the ICU due to COPD should receive antibiotics (even if there is no infiltrate on the chest X-ray) (Vollenweider et al 2012).
- Patients with COPD have airways which chronically grow a variety of organisms. The goal of antibiotic therapy is generally to suppress this bacterial growth a bit, not to completely sterilize the patient's lungs (which is impossible in this situation). Therefore, narrow-spectrum antibiotics are fine.
  - Avoid getting sputum cultures and ignore them if they have been obtained (these patients will grow weird stuff in their sputum chronically; there is no need to cover every single organism) (Farkas et al 2010).
  - Good choices include azithromycin or doxycycline.
    - Azithromycin is generally first-line, if the patient hasn't been exposed to it recently (don't worry, it doesn't cause Torsade de Pointes).
  - Narrow antibiotics seem to be as effective as broader antibiotics, but may cause less Clostridioides difficile.

avoid excess oxygen

- Excess oxygen may cause diffuse pulmonary vasodilation, which disrupts ventilation-perfusion matching and thereby increases PaCO2 (Abdo WF et al 2012).
- Titrate inhaled oxygen to target an oxygen saturation of 88-92% (with 85-95% being OK).

noninvasive ventilatory strategies

indications for immediate intubation

- Immediate intubation is generally the wrong move. With strategic use of various medications and noninvasive modalities, intubation can very often be avoided.
- Indications for immediate intubation may include:
  - Multiorgan failure (e.g. COPD plus cardiogenic/septic shock)
  - A patient who is truly not protecting airway (e.g. gurgling secretions in upper airway)
  - Respiratory/cardiac arrest
- When in doubt about intubation, a reasonable approach is often to prepare for intubation, while simultaneously placing the patient on BiPAP:
  - If the patient improves, that's great; you can avoid intubation.
  - If the patient doesn't improve, then BiPAP will still optimize their physiology prior to intubation.

BiPAP is the first-line noninvasive strategy

- BiPAP is supported by a very robust evidence base for the treatment of COPD. It has been proven to reduce death (relative risk 0.4), reduce intubation (relative risk 0.4), and reduce treatment complications (relative risk 0.3). This is impressive evidence which argues strongly that whenever possible, the patient should be given a real college try on BiPAP.
- Indications for BiPAP?
  - Substantial respiratory distress or tachypnea (respiratory rate >=30/min).
  - Somnolence due to hypercapnic encephalopathy, as a result of COPD exacerbation.
- Contraindications to BiPAP
  - Need for immediate intubation (see above).
  - Vomiting or increased risk of vomiting (e.g. bowel obstruction).
  - Copious secretions, difficulty with secretion management.
- BiPAP settings
Pressure: Start at 10cm iPAP/5 cm ePAP. If tolerated, may up-titrator as needed to ~18 cm iPAP/8 cm ePAP. Titrate the driving pressure (iPAP-ePAP) to achieve an adequate tidal volume. Whether to increase the ePAP slightly to cancel out autoPEEP (e.g. 8 cm) or whether to use 5 cm of ePAP is debatable and probably not clinically relevant.

Monitor tidal volume & minute ventilation on the BiPAP monitor.

Really low tidal volumes (e.g. <300-400 ml) and low minute ventilation (e.g. <5-6 L/min) suggest inadequate ventilation. In this situation try up-titrating the pressures and widening the driving pressure (with a rough maximum support level around ~20cm iPAP/5 cm ePAP).

what if the patient can’t tolerate the BiPAP mask?

- Don’t just assume that the patient needs to be intubated. The first step here is often to try some sort of sedation. If that fails, then the patient may be trialed on HFNC.

- Dexmedetomidine
  - Excellent anxiolytic to help patients tolerate the mask and rest while on BiPAP. The combination of BiPAP plus dexmedetomidine is termed ‘BiPAPidex.’ This is a powerful approach, especially for anxious patients with flash AECOPD (see figure above).
  - Strength of dexmedetomidine is that it doesn’t suppress the respiratory drive and it’s titratable, making it the safest sedative.
  - Weakness of dexmedetomidine is that it can take a little while to work. Boluses of dexmedetomidine can cause hemodynamic instability, so a reasonable approach may be to start the infusion at a high rate (1-1.4 mcg/kg/hr) and then titrate down as the patient becomes sleepy. This will take ~30-60 min to really work.

- Low-dose IV haloperidol or olanzapine is another option which may calm patients without suppressing respiratory drive or causing delirium.

- Benzodiazepines have mixed results here: occasionally they work, but they often cause the patient to get more confused/agitated. For patients who are on benzodiazepines chronically and respond well to this class of medication, this makes sense. In most cases I avoid benzodiazepines.

- Ketamine: For the acutely agitated patient this is a good option, with some bronchodilatory properties.

- Fentanyl: For patients with severe tachypnea and air hunger, small divided doses of fentanyl can be used to help them decrease their respiratory rate sufficiently to give them time to exhale properly (see: pathophysiology above). This must be done with very careful monitoring of minute ventilation & respiratory rate, by someone with extensive experience in treating respiratory failure. IV fentanyl is the way to go here because it works rapidly, so you can titrate it meticulously to hit a sweet spot where the patient is breathing at a rate of ~12-24 breaths/minute (fast enough to stay alive, but slow enough to allow sufficient time to exhale properly).

high-flow nasal cannula (HFNC)

- HFNC helps COPD patients mostly by reducing their anatomic dead space, improving ventilation, and reducing the work of breathing (‘blowing off CO2’ – more on the chapter on HFNC).

- There isn’t much evidence to support the use of HFNC in COPD (unlike BiPAP, which is supported by robust evidence). Thus, HFNC is currently a second-line therapy here.

- HFNC may be useful in the following situations:
  - Patients who are unable to tolerate BiPAP.
  - Patients in whom BiPAP is contraindicated (e.g. due to vomiting), but who aren’t sick enough to require intubation.

- Titration of settings:
  - Flow rate should be maximized to the highest level that the patient will tolerate (ideally at least 50-60 liters/minute flow).
  - FiO2 should be adjusted to target a saturation of 88-92% (accepting sats of 85-95%), as discussed above.

monitoring on BiPAP/HFNC

- Key parameters
  - Oxygen saturation: Target saturation 88-92%, tolerate 85-95%. Excessive oxygen may impair VQ matching and thereby impair CO2 clearance.
  - FiO2 requirement: COPD itself generally impairs CO2 clearance, but it shouldn’t cause profound hypoxemia. If the patient has escalating oxygen requirements, this suggests that something else is going on (e.g. pneumonia, mucus plugging, pulmonary embolism) – you need to investigate this further.
  - Respiratory rate: Significant tachypnea (e.g. >~25-30 b/m) is a bad sign that the patient may eventually tire out. Ideally HFNC/BiPAP should cause a drop in respiratory rate.
  - Work of breathing: Look for deterioration (e.g. retractions, abdominal paradoxical breathing, tripoding).
**BiPAP monitor.** BiPAP allows you to monitor tidal volume & minute ventilation as described above. Note that a mask leak may cause these measurements to be imprecise.

**ABG/VBG or mental status**
- If the patient has an intact mental status, I don't think you need serial ABG/VBG values. If the patient is arousable and able to report how they are feeling, then just follow the clinical exam. Ideally the patient will report that they are feeling better. If the patient starts getting progressively more sleepy/confused, then you may be in trouble (check an ABG/VBG to exclude severe hypercapnia).
- If the patient is sedated, then you do need to follow ABG/VBG values to make sure the patient isn't becoming dangerously hypercapnic (sedation prevents you from using mental status to exclude severe hypercapnia). While either ABG or VBG is fine, serial VBG monitoring using a peripheral vascular catheter that allows blood withdrawal is usually the most humane approach. More on ABG versus VBG differences here [here](https://emcrit.org/pulmcrit/vbg-abg/).

**indications for delayed intubation**

- **Delayed intubation** is defined here as a patient who stabilized on BiPAP or HFNC, but subsequently is intubated a couple hours later (usually due to failure to improve). In my opinion this is usually a mistake, because it represents a misunderstanding of the goals of noninvasive ventilation.

- **Goals of BiPAP/HFNC in COPD:**
  - (a) Maintain adequate oxygenation (>85-88%)
  - (b) Reduce the work of breathing, so that the patient doesn't develop progressive diaphragmatic fatigue. This is probably the most important goal. Therapeutic targets here include improvement in tachypnea and in the patient's subjective sense of breathlessness.
  - (c) Keep pCO2 low enough that the patient doesn't develop complete obtundation/coma. The goal is *not* to immediately improve the ABG/VBG. Ideally the blood gas will improve, but this may take a while (several hours).

- **Key points:**
  - The decision to intubate is ultimately a *clinical decision*. If the patient seems to be stable or improving but the blood gas remains unchanged, then it's often best to continue BiPAP/HFNC with careful monitoring.
  - Serial ABG or VBG values will vary randomly by as much as ~0.03 differences in pH and ~5 mm differences in pCO2 ([Umenda 2008](https://www.ncbi.nlm.nih.gov/pubmed/19091262), [Sasse 1994](https://www.ncbi.nlm.nih.gov/pubmed/8020270), [Thorson 1983](https://www.ncbi.nlm.nih.gov/pubmed/6407807), [Hess 1992](https://www.ncbi.nlm.nih.gov/pubmed/1583545)). So if the pCO2 increases slightly, that doesn't necessarily indicate that the patient is worsening – it may simply be noise due to repeated laboratory measurements.
  - In summary: If the patient is *clinically deteriorating*, then intubation is indicated. However, if the patient is overall stable, then it's often best to continue noninvasive therapy with careful monitoring (with the ability to immediately intubate if warranted). It may take some hours for the bronchi to open up and clinical resolution to occur. Wait for it.

**how long should BiPAP/HFNC support be continued?**

- For most patients, ~12-24 hours of support may be reasonable.
- Avoid premature discontinuation of support. Diaphragmatic fatigue and bronchoconstriction take time to resolve. Even if the patient looks beautiful after 1-2 hours on BiPAP, it's often a mistake to discontinue it prematurely (assuming that the patient truly needed BiPAP initially).
  - One potential exception is a patient with pure flash-COPD exacerbation (see figure above). This refers specifically to a patient who was doing perfectly fine, then suddenly developed anxiety/tachypnea and fell apart. If the patient was really doing great before this episode, they may require only transient BiPAP support to stabilize them and return to their baseline.
- Don't keep patients on BiPAP for too long. Over time, BiPAP can cause ulceration of the nose. It's probably a bad idea to leave a patient on continuous BiPAP for >48 hours. If the patient is unable to be freed from BiPAP after 48 hours of intensive therapy (e.g. even unable to tolerate HFNC), then you probably need to consider intubation.
- For patients who are very tenuous and require a prolonged duration of support, the following strategies may be considered:
  - HFNC can be continued indefinitely, because this allows for adequate nutrition.
  - Many patients can be weaned from BiPAP to a combination of nocturnal BiPAP plus HFNC during the day. Over time, as they recover, they can be transitioned to nocturnal BiPAP plus a standard low-flow nasal cannula during the day.

**indication for intubation**

https://emcrit.org/ibcc/AECOPD/
These are explored in more detail above. To summarize:

(a) Indications for immediate intubation
- Multiorgan failure (e.g. COPD plus cardiogenic/septic shock).
- The patient is really not protecting airway (e.g. gurgling secretions).
- Respiratory/cardiac arrest.

(b) Indications for delayed intubation
- Patient clinically deteriorating despite optimized BiPAP/HFNC support.
- Patient stabilizes on BiPAP but is completely BiPAP-dependent for >48 hours.

**Intubation procedure itself**

- Consider use of a relatively large-size ETT (e.g. 8.0 for larger people, 7.5 for smaller people). Use of a small ETT may increase airway resistance, hindering your ability to ventilate.
- Resist the urge to aggressively bag patients following intubation. COPD patients may rapidly trap gas in their lungs (due to impaired airflow), leading to pneumothorax or hypotension. Bag these patients gently and slowly.

**Ventilator settings**

- Ventilating COPD patients is generally much easier than ventilating asthmatic patients, despite the fact that both have airflow limitation.
  - COPD patients: Respiratory failure is usually due to a combination of diaphragmatic fatigue and bronchospasm. Once they are on the ventilator, diaphragmatic fatigue isn’t a problem — so ventilation is fairly easy.
  - Asthmatic patients: Respiratory failure is due primarily to intense bronchospasm. The degree of bronchospasm is more severe, which can create major challenges in ventilator management.
- The major concern with ventilation is autoPEEP
  - Patients have difficulty with expiration. If the tidal volume and/or respiratory rate are too high, this causes gas trapping inside the chest at end-expiration (autoPEEP).
  - AutoPEEP can be problematic because it can impair venous return to the heart (causing hypotension) and it can make it difficult for the patient to trigger the ventilator (leading to ventilator dyssynchrony).
  - AutoPEEP can be diagnosed by persistent expiratory flow at end-exhalation (airflow never goes to zero before the next breath).
  - The treatment for autoPEEP is to reduce the respiratory rate and/or tidal volume. Decreasing the respiratory rate is generally the most effective intervention. If the patient is over-breathing the ventilator, suppression of their respiratory rate may be necessary (e.g. with propofol or an opioid).
  - Increasing the set PEEP slightly (e.g. from 5 cm to 8 cm) may stent open airways during expiration and make it easier for patients with a little autoPEEP to trigger the ventilator.
- Reasonable starting settings:
  - Volume-cycled vent: Tidal volume 8 cc/kg, respiratory rate ~14 b/m, 5-8 cm PEEP
  - Pressure-cycled vent: Pressure 30 cm/8 cm, respiratory rate ~14 b/m.

**Target blood gas?**

- Many COPD patients have chronic hypercapnic respiratory failure, with a chronic compensatory metabolic alkalosis. In this case, ventilation to a normal pCO2 (40mm) is problematic for two reasons:
  - (1) Ventilation to a normal pCO2 will cause alkalemia (pH >7.45), which probably isn’t awesome.
  - (2) Over time, the kidney will respond to alkalemia by excreting bicarbonate until the serum bicarbonate level is ~24 mEq/L. This will cause problems with trying to get the patient off the ventilator. Stripped of their chronic compensatory metabolic alkalosis, the patient now needs to blow their pCO2 down to ~40 mm in order to achieve a normal pH. This will increase their work of breathing, making it harder for them to pass a spontaneous breathing trial or be liberated from the ventilator.
- The best approach is generally to target a pCO2 close to the patient’s baseline value:
  - If you know the patient’s baseline, you can use that.
  - In most cases you won’t know the patient’s baseline. In this situation, targeting a lowish pH (shoot for pH of roughly ~7.25-7.35) will get you close to the patient’s baseline pCO2. Mild acidemia will stimulate the kidney to retain bicarbonate, which keeps the patient near their baseline bicarbonate level (which will eventually facilitate extubation).
  - If there is difficulty achieving this pH, then lower pH may be entirely acceptable as well (i.e., a strategy of permissive hypercapnia).
- Unfortunately, severe COPD is one situation where end tidal CO2 may be misleading.
### weaning off ventilation

- **Timing**
  - It’s generally a reasonable idea to rest the patient on the ventilator for at least ~24 hours in order to allow for diaphragmatic rest. (Even if the patient looks terrific after a few hours on the ventilator, it’s generally not a great idea to extubate at that point in time.)
  - After ~36-48 hours, bronchospasm and diaphragmatic fatigue really ought to improve, so efforts to wean should be quite aggressive in that time-frame.
  - Prophylactic extubation to HFNC or BiPAP reduces the risk of extubation.
    - HFNC is easier to tolerate, potentially making it superior here.
    - For patients with chronic hypercapnia, consider transitioning to chronic nocturnal BiPAP.

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### checklist

**COPD exacerbation**

- **Diagnosis**
  - Cardiopulmonary POCUS
  - Review CXR
  - Review EKG
  - Further tests as needed (e.g. PE workup if atypical symptoms)

- **Respiratory support**
  - 1st line is BiPAP
    - Remember to titrate settings to optimize level of support.
    - Trial sedation (e.g. dexmedetomidine) if unable to tolerate.
    - Contraindicated if nausea/vomiting.
  - 2nd line is HFNC
  - Intubation only if clinically necessary

- **Steroid**
  - Start with IV methylprednisolone usually (e.g. 125 mg IV)
  - Rapidly de-escalate to lower doses (e.g. prednisone 60 mg qd)

- **Bronchodilators**
  - Hold all home bronchodilators.
  - Albuterol/pratropium nebulized Q6hr, scheduled.
  - Albuterol nebulized Q2hr PRN.

- **Antibiotic**
  - Use narrow-spectrum (azithromycin or doxycycline).
  - Choose something the patient wasn’t recently exposed to.

- **Monitoring**
  - BiPAP: Follow tidal volume & minute ventilation.
  - Respiratory rate: <10 suggests hypoventilation, >30 suggests high work of breathing that may be unsustainable.
  - Oxygen saturation: Shoot for 88-92%, accept 85-95%.
  - Mental status/CO₂: If patient is arousable and mentating, follow mental status. For patients who are sedated, follow A&G/VBG.
  - Gestalt: Probably most important. Look at the patient! Discuss with nurse and respiratory therapist. Follow over time.

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### podcast


The Podcast Episode

Want to Download the Episode?
Right Click Here and Choose Save-As (https://trac.libsyn.com/secure/ibccpodcast/IBCC_EP_80_-_AECOPD.mp3)

questions & discussion
(back to contents)

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

Over-use of antibiotics: Chasing sputum cultures with broad-spectrum antibiotics. As discussed above, COPD patients will always grow strange pathogens from their sputum, even when healthy (e.g. pseudomonas).

Under-use of antibiotics: Failure to provide any antibiotic therapy to a patient with severe AECOPD.

Under-utilization of BiPAP: Even patients who look terrible (and may seem like they require intubation) will often improve rapidly on BiPAP.

Inadequate sedation for BiPAP: BiPAP is proven to reduce mortality in COPD, so it’s worth taking a little time and trying to sedate the patient so that they can tolerate it (e.g. with dexmedetomidine). Immediately concluding that an anxious patient “can’t tolerate BiPAP” and proceeding to intubation often isn’t in the patient’s best interest.

Going further:

- IBCC chapter on noninvasive respiratory support (https://emcrit.org/ibcc/support/).
- EMDocs: Acute COPD Exacerbation (http://www.emdocs.net/em3am-acute-copd-exacerbation/) (Brit Long)
- EMDocs: Chronic Obstructive Pulmonary Disease Exacerbation: When it isn’t just your classic exacerbation... (http://www.emdocs.net/chronic-obstructive-pulmonary-disease-exacerbation-when-it- isn’t-just-your-classic-exacerbation/) (Sarah Iosifescu and Jennifer Beck-Esmay)
- Is too much supplemental oxygen bad? (https://rebelem.com/is-too-much-supplemental-o2-harmful-in-copd-exacerbations/) (RebelEM by Allan Guiney, MD)

The Internet Book of Critical Care is an online textbook written by Josh Farkas (@PulmCrit), an associate professor of Pulmonary and Critical Care Medicine at the University of Vermont.