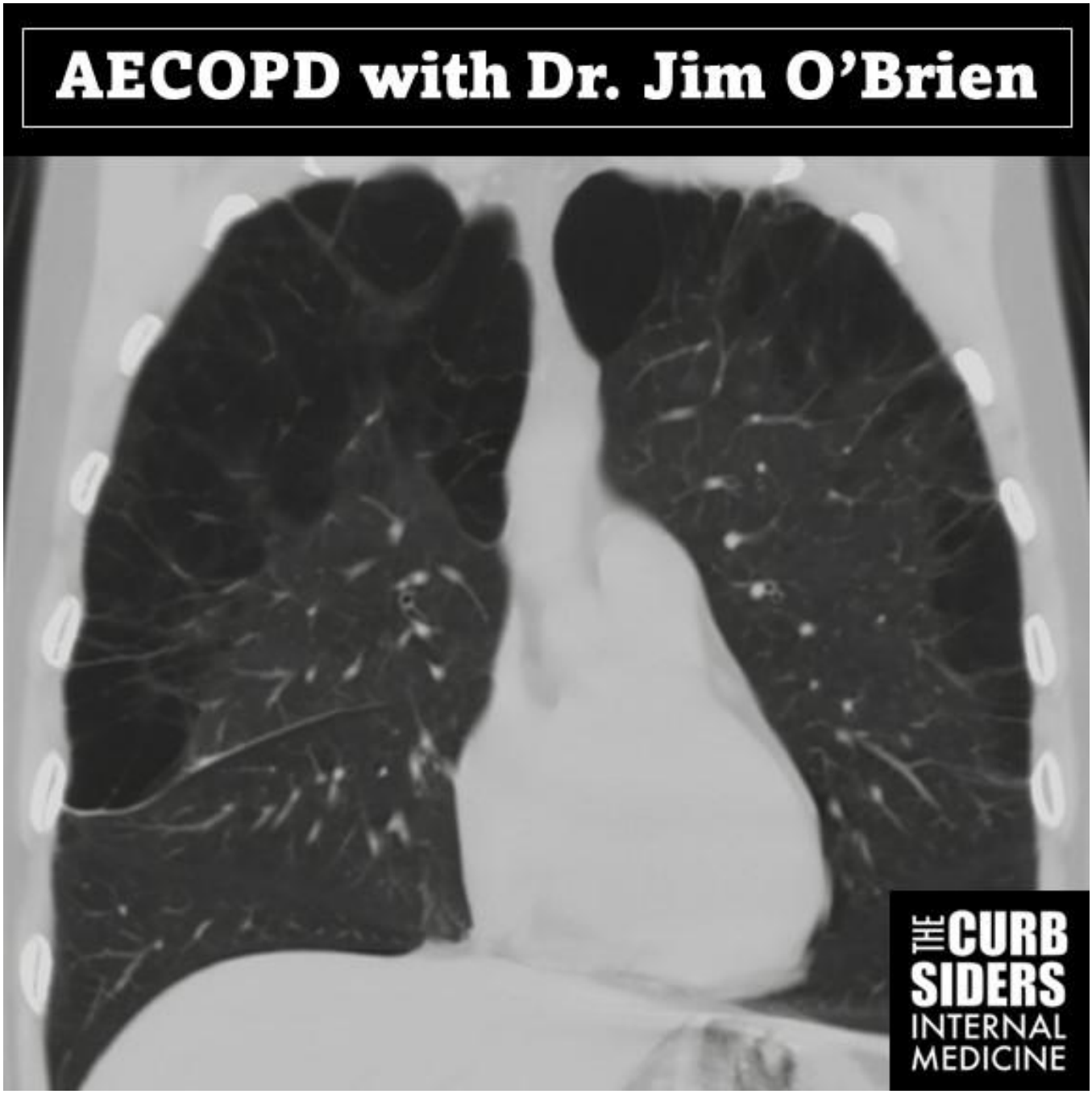
#378 Acute Exacerbations of COPD (AECOPD)



Monee: I feel like I need to do this. It's been a long night, and I feel like we are really finding it harder to breathe. Just as Maroon 5 did in 2004.

Meredith: That's the one you picked? You didn't want to go with your Destiny's Child?

Monee: Well, we did lose our breath too, so.

Cyrus: Oh, God, I love Destiny's Child.

Meredith: [chuckles] They're wonderful.

Monee: So good.

[Disclaimer]

Monee: And welcome back to Curbsiders. I'm Dr. Monee Amin, joined by my lovely cohost, Dr. Meredith Elizabeth Trubitt. How are you doing this evening?

Meredith: Doing pretty well. How are you?

Monee: You're holding up, girl?

Meredith: I am, barely. [chuckles]

Monee: Well, on tonight's show, we have a great conversation about acute exacerbations of COPD with our guest, Dr. Jim O'Brien. And in just a minute, Dr. Cyrus Askin, is going to tell you more about our guest. But in the meantime, Meredith, will you please let the good people in the audience know what this show is about?

Meredith: Sure Monee, I'd love to. We are *the* internal medicine podcast. We use expert interviews to bring you clinical pearls and practice-changing knowledge.

Cyrus: Awesome. Well, happy to be here on air with both of you and happy to welcome our guest, Dr. Jim O'Brien, with whom we had a great conversation about acute exacerbations of COPD. Dr. O'Brien is a graduate of the University of Minnesota Medical School. He did an internal medicine residency at Hennepin County Medical Center in Minneapolis. Then, he went on to do a pulmonary fellowship at NYU, followed by a critical care fellowship at Stanford. Dr. O'Brien practiced pulmonary critical care for about 15 years in the Seattle area before returning to academics at National Jewish Health in Denver. He's practiced for about 30 or more years at this point. So, a wealth of experience. In addition to continuing to see pulmonary and critical care patients, Dr. O'Brien has been part of the COPD group at National Jewish since 2013. So, we are super excited to have this expert in the field with us tonight, and we cannot wait to dive in.

Monee: Hi, Jim. Thanks for coming on the show. Cyrus, can you take us to our first case from Kashlak?

Cyrus: I would love to.

Do you have certain clinical skills that you need to brush up on? Perhaps, ECG interpretation or point-of-care ultrasound? Or maybe you are interested in continuing education? Then, you need

to check out Medmastery, The Clinical Skills Academy. Medmastery is an award-winning online learning platform endorsed by the British Medical Association. They offer an affordable subscription which will give you access to their entire course library of more than 100 CME-accredited courses and workshops on topics like mechanical ventilation, fluids and electrolytes, chest x-ray interpretation, echocardiography, pulmonary function testing, ECG from the basics to advanced skills like culture monitoring and stress testing, and so much more. Medmastery's courses are taught by top-notch educators who break concepts down into easy-to-digest, short, five-minute videos that you can cram into your busy schedule.

Listeners of this show can claim a 15% discount off an annual Med Mastery Pro subscription, which is a savings of more than $80. Just go to *www.medmastery.com/curbsiders* to claim your discount. All subscriptions are backed by a 30-day money-back guarantee. So, what are you waiting for? Take advantage of the discount and give Medmastery a try. Again, that's *www.medmastery.com/curbsiders*.

Our case coming from Kashlak Memorial. You are working at a nearby safety net hospital affiliated with Kashlak Memorial, and you're called to the emergency department to evaluate Ms. Natalie Harrison. The ED physician tells you that Ms. Harrison is a 56-year-old female with an extensive smoking history who presented with shortness of breath. She started smoking at age 16 and has averaged two to three packs per day since then. Unfortunately, over the last year, she's fallen on hard times and finds herself undomiciled, moving from shelter to shelter as needed. Over the last three to four days, she's felt feverish, noticed a worsening cough which, of note, is productive of copious sputum and overall endorses easy fatiguability, and malaise in addition to her dyspnea. Ms. Harrison has never had health insurance and does not take any medications. However, she had been seen intermittently at a local free clinic where a volunteer pulmonologist did some breathing tests and diagnosed her with chronic bronchitis.

So, Jim, as you're listening to the patient's presentation, I guess to open things up, what elements of her history are immediately concerning and what other information might be worth obtaining prior to your face-to-face evaluation, while you're kind of on the phone thinking through what you might want to do for this patient?

Jim: I would say that the important things in addition to the dyspnea and cough, etc., would be the fact that she has fever making a mere acute exacerbation of COPD or even bronchitis may be less likely. More likely, she may have pneumonia or some other more infectious inflammatory process going on. I think the fact that she has copious sputum is helpful in making some decisions down the road about antibiotic therapy. In addition, if I was getting a call from a hospitalist about this, I'd ask her BMI, because that would be important given the information that's coming down the road about where she should be sent in the hospital or disposition and how concerned we may want to be with, in particular her arterial blood gas. So, that would be an important piece of information to me.

Cyrus: Can you elaborate specifically on that? The BMI element isn't something I routinely think about, except in the sense that a lot of my patients who have advanced COPD are cachectic often because of their COPD. Can you talk more about that?

Jim: Yeah, I can. There's the traditional monikers of a pink puffer and a blue bloater. A pink puffer is somebody who has hypoxemia assessment intact in their brainstem, and they will work to keep their oxygen level and their CO2 level. Their CO2 response is high as well. They do that at the loss of musculature. Somebody who is a blue bloater usually is overweight and just doesn't give a hoot about what their carbon dioxide and their oxygen are. That usually evolves

out of having sleep apnea and having changes occur in bicarb levels in CSF and affects the brainstem over time to limit CO2 and hypoxemia response. If the blood gas is poor, I get less worried about somebody who is obese or overweight and a blue bloater than I do in somebody who is a pink puffer who doesn't really have much reserve when it comes to musculature and breathing capacity. So, yeah, I'll leave it at that.

Cyrus: I see. I know we talked a little bit-- You mentioned the fever, and she has these subjective fevers. Later in the case, we'll get a little more information on that front, but certainly, that's something that could point you in one direction or another. I think you mentioned the copious sputum, the cough. That's something that's kind of worrisome and maybe leaning you perhaps in the direction of a COPD exacerbation, depending on some of the other things you're finding. Is that accurate?

Jim: Yes, I would say, first of all, COPD being highly prevalent in smokers, it's also highly prevalent in those of lower socioeconomic status and her being undomiciled is going to create like, a pretest suspicion in my mind. The fact that she was diagnosed with chronic bronchitis prior to this is also helpful in sending me down the track of thinking something related to COPD. Now, when it comes to bronchitis versus pneumonia relative to a fever, usually chronic bronchitis doesn't cause fever. I think less of just chronic bronchitis when I hear somebody has a fever and a white count and down the road, I guess we would know that.

Cyrus: Excellent. Okay.

Meredith: I might jump in for a second. I think that you also were in a couple of answers you gave and already referencing also like their blood gas being an important variable for you. What are the other important indications early on blood gas O2? Can you walk us through what are the important initial things you're asking for?

Jim: Sure. Yeah, so vital signs would be important. With vital signs, I would ask for the BMI as well. We report those with our vital signs here. Oxygen saturation is something that we can do immediately, and depending on what that is, understanding what their supplemental oxygen needs are going to be or what they are if they've already got supplemental oxygen. Then, exam would be important, and I'd probably ask if they're wheezing or not. It may be helpful in a diagnosis. If she has wheezing, it may suggest that she has a component of asthma in addition to COPD. I don't think you can get a great cardiac exam in a noisy emergency department. So, the idea of a new murmur or something, I wouldn't depend on.

And then, I think of labs. The labs that I would want would be a full basic metabolic panel so that I can get an anion gap and an ABG. I would also want to know about the CBC wanting to understand her white count and her anemia status, etc. But I think the most important labs are going to be the ABG and anion gap. Then, I'd want to know what a chest radiograph looked like.

Cyrus: And you mentioned ABG. Sorry, are you--

Jim: Okay. VBG would be fine.

Cyrus: Okay.

Meredith: I'm going to ask one more question before we go on to something else. For her, I think Cyrus is painting like a nice picture of COPD. So, we've kind of gone down her need for a chest x-ray. But certainly, I don't know that everyone would come in quite so black and white and

so certainly could be thinking of a broader differential. If there are certain times where you might be more prone to wanting to do like a CT chest or kind of what helps you decide that?

Jim: Yeah, I think somebody with known COPD who presents to an emergency department, there is a broader differential than just the COPD exacerbation, obviously. And in this case, pneumonia is one. This is something that I think most of us know, is that pulmonary emboli are common in people who present to hospitals with an acute exacerbation of COPD. In some literature, 15% to 20% of those that present to emergency departments have evidence of a pulmonary embolus. Now, a pulmonary embolus may be there in a subsegmental aspect, and that may not mean that the pulmonary embolus triggered their acute exacerbation, but PE is something that one would have to consider. Others would include congestive heart failure, or in this case, I would wonder about somebody having bacterial endocarditis and having valvular dysfunction or throw in emboli. I would also then consider what else can we throw in there. We throw in pleural effusions pneumothorax.

Can I say one more thing about the role of a D-dimer in this setting? Because I would be inclined just to get a CT angiogram on her in the emergency department and I wouldn't depend on a D-dimer. There is some data that suggests that D-dimers are elevated in patients having acute COPD exacerbations without evidence of pulmonary embolus. So, if you have a negative D-dimer in this setting, again, like the CRP, I would probably consider it a spurious lab. And then, if it's positive, you'd move on to your next diagnostic regimen, which is a chest CT angiogram.

Cyrus: Well, I think probably moving on with the case so we can get to the meat and potatoes here. Unfortunately, this patient doesn't have any history in the medical records, so you don't really have anything to reference. As we stated earlier, Kashlak, paper charts, who knows where the records are anyway? But you do have some initial labs and some imaging, so the CBC gives you a white count of 17, some mild anemia, thrombocytosis. BMP is notable for mild hyponatremia 128, a bicarb of 34, a creatinine of 1.3, a BUN of 47, and a lactate that's modestly elevated at 27. Patient has a venous blood gas that's drawn. The pH there is 7.25 on the venous gas with a pCO2 of 77. A D-dimer was also drawn and it's modestly elevated. Cardiac enzymes and BNP were obtained there within normal limits. Chest x-ray was obtained, notable for hyperinflation, but no evidence of a lobar process, and no other findings there that were mentioned. At this time, no CT has been obtained.

And finally, the ED physician tells you that the patient's alert, awake and oriented, but does appear a bit tired. Other labs that were obtained include blood culture, sputum culture, and then a respiratory viral panel, which is pending as well.

With that, you arrive to the ED, you see Ms. Harrison, and review her vitals. She's currently on a 6 liters nasal cannula. Her sat is 89%. She's hypertensive and mildly tachycardic. You do note that despite her concerns regarding fever, she actually is and has been normothermic in the ER, so felt feverish but no fevers. So, that's a lot of information. I know. I guess just an open-ended question is, based on that information, are there any labs or rads that stick out to you and help you fill in your illness script for what's going on with this patient?

Jim: Right. Yeah. Thanks. First of all, I'm going to address the anemia and the thrombocytosis. I think that's probably related to iron deficiency anemia, and I would not consider that a significant diagnostic player in this case. And yet, she'll need that addressed when she's discharged. The sodium of 128 may be related to a cardiac source if she has other examination evidence like pedal edema or a history of what might be congestive heart failures symptoms like general

fatigue and malaise like she complains of. And also, SIADH is very common in lung disease generally, so somebody with an infiltrate or pneumonia often has low sodium. This isn't life-threatening sodium. So, I think we can just tuck that to the back of our heads and acknowledge it in the setting of her acute illness.

The bigger things to me include her blood gas. Since you did give me some of this information before, I put what I think is a full blood gas together. So, the pH being 7.25, her pCO2 was 77. I'm going to give her a pO2 of about 58, and her bicarb of about 34. It's a venous gas, so the CO2 might be a little bit elevated. Normal arterial to venous CO2 is 40 to 45, 40 on the arterial side and 45 on the venous side. So, it's not a huge CO2 elevation. This is a significant elevation of CO2. I would say that if you correct the pH, I'm using a correction of with every ten points of CO2, you see a change of 0.08 of pH. I can't remember all those other equations, but this one is quite helpful.

Monee: I was just saying it's really nice to hear that someone who sees these more than me feels the same way because I can never keep these straight.

Jim: Well, it's very easy now with apps and everything just to look this stuff up. But I think this is a critical part of her case in that this corrects out to her CO2 being about 60 at baseline with a calculated pH greater than 7.35, brings her CO2 down to about 60 and then her bicarb at 34. This shows that she's somebody that has a chronic hypercapnic state essentially or respiratory acidosis that's corrected.

Then, she has acute respiratory acidosis with her pH being down and her CO2 being elevated. So, this would, for me, kind of suggests that she may be overweight and she may be a blue bloater or somebody that just doesn't have CO2 or hypoxia response in their brainstem and they just kind of don't care, in so many words, about what their CO2 and their O2 is doing.

Cyrus: I think that's a super helpful walkthrough of those labs and how you interpret them and how you paint a picture, so to speak. I guess one question we should probably get to is, now that you're there with her and you're doing your exam, things like auscultation, point-of-care ultrasound, assessing the extremities and work of breathing, how do you marry all of those skills together when you're doing your initial assessment?

Jim: Yeah, this is something that brings out the experiential aspect of medicine, I think. I would walk into a room and know in a second if someone needed-- where they needed to go in the hospital if they needed noninvasive ventilation or mechanical ventilation. To analyze that, I think I would look at their breathing first and I would look for accessory muscle use. I would look for paradoxical breathing, chest down, abdomen up, abdomen down, chest up. I would look at her eyes and see how distressed he or she is. I would get a sense of what their work of breathing was based on that, I would see or decide if they were in respiratory distress. With respiratory distress comes usually elevated work breathing.

On physical exam, like I say, pulmonary auscultation would be important. I wouldn't depend that much on cardiac auscultation. I would want to know if they have pedal edema or not. And then also I'm looking for if they're blue anywhere, lips, fingernail beds, etc. Echo? Do you want me to go through that?

Meredith: As the work of breathing, especially, like, signs, are there ones that you would treat are more concerning to you, where you triage to the unit may be faster than where you're more

comfortable with them being on the floor for at least a little bit? Can you walk through that thought process?

Jim: Sure. Paradoxical, breathing is threatening, but I would also ask her if she's getting tired. Patients usually know exactly what's going on, and they often say, "Yes, I'm going to crap out here." It's not necessarily just the exam. It's the discussion with the patient.

Meredith: I interrupted before. So, if you wanted to talk about echo and ultrasound, we could go back to--

Jim: Okay. Alright. Bedside echo in this case might be helpful. We could get a sense of what her right ventricular appearance is. If she did have right ventricular dilatation, then I would be even more inclined to get a chest CT angiogram, understanding also that somebody in acute distress may have pulmonary hypertension and acute right ventricular dilation. I would get a sense of what their left ventricle looks like and if they are at risk for left ventricular failure in the setting of right ventricular dilatation, which may contribute to pulmonary edema. We'd see any wall motion abnormalities, we'd see pericardial infusion.

Then, I would move to the lungs and look for evidence of pneumothorax. With the stratosphere appearance of bedside echo, I would look for B-lines to give us any sense if they have pulmonary edema or if they're developing acute lung injury. That actually would be quite helpful in the early evaluation of her. And yet, I still want the chest CT angiogram.

Cyrus: I guess with that, we'll go ahead and say, okay, all signs at this point are leaving you with a leading diagnosis of COPD exacerbation. We'll say that while she does look like she's having of difficulty breathing, after putting the oxygen on her, she is feeling better. She's starting to feel a little more comfortable. You're not seeing that belly breathing. You're not seeing that tripoding or those retractions that would make you think, "Okay, this person needs to go to the intensive care unit for very close monitoring and potentially impending intubation." This is actually someone who are going to say is appropriate for perhaps a progressive care unit for closer monitoring, but maybe not ICU-level care.

As you're putting in your admission orders and that sort of thing, I guess maybe before we get to the actual like what are you going to order and what kind of treatments are you going to administer, I did some research and did some reading and obviously, a pulmonary fellowship is not too far away from me. Different people seem to have somewhat different definitions of what an acute exacerbation of COPD actually is. So, I was curious if you might have an operational definition for what constitutes an exacerbation to you and then kind of a follow-up to that would be if there's a common pathophysiology that we should be familiar with in regard to those exacerbations.

Jim: What I've been telling patients, and I know my colleagues are, is that if they have a change from their baseline of shortness of breath, cough, or sputum, then they're having an acute exacerbation of COPD. By sputum, I mean whether they have more sputum or purulent sputum. Most frequently this is something that occurs at home over a few days, and they call clinic and we make decisions on whether we want to see them or how to treat them over the phone. If someone has enough shortness of breath or cough or purulent secretions that would require that they go to a hospital, that's considered a more severe exacerbation. The GOLD guidelines allow stratification of COPD by greater than or equal to two exacerbations a year or one hospitalization.

Cyrus: My other question was having that operational definition basically increased dyspnea, increased cough, increased production of sputum, those sorts of things, I think that's very helpful. But then, is there an underlying pathophysiology that drives these exacerbations? Something that we as clinicians, our listeners, wherever they are in their training or in their lives, should be aware of.

Jim: Yeah, things that precipitate acute exacerbation. It's about a third viruses, a third bacteria. Somewhere in there, we can include both because we know that viral illnesses predispose to bacterial pathology. But then, a third go undiagnosed and we think that it's exposure to external pollutants. But I would also suggest from our experience that GERD and aspiration are very important to think of, gastroesophageal reflux and aspiration. If someone has acute symptoms and a history of COPD, really acute symptoms, that's something to also think about. Once if you think that the patient had GERD and aspiration, then bacteria, either enteral bacteria or whatever he or she is colonized with in their oral pharynx may be helpful when you're choosing antibiotics. I think of MRSA in this case. A certain number of people have oropharyngeal MRSA, or they're colonized with MRSA. If somebody is aspirating and having exacerbations of their COPD, I think one would empirically want to include MRSA in your treatment. So, that would be the precipitants of a COPD exacerbation.

The physiology, I think, is really based on airway edema. There are many precipitants of changes in airway edema. The airway edema is most likely at the small airway level, 2 mm or less. If somebody has bronchitis and worsening of their bronchitis, that usually suggests they have larger airway inflammatory changes. But the larger airway inflammation is an indicator of lower or smaller airway edema as well, usually.

Monee: I think we've established a good foundation in the basics of history, labs, and then what exactly is a COPD exacerbation? So, the moment has arrived. I think it's a good time to maybe start talking a little bit about treatment. I guess we think about a lot of the mainstays. I think the first thing a lot of us think about are steroids. Talk us through dosing and choice and all that stuff.

Jim: This is still an issue that's up in the air, I think. I think most that are younger than I am use oral steroids in somebody who's admitted to a hospital. When it comes to-- I'll back up a little bit and when it comes to outpatient steroids, I think it's pretty clear. Steroids are beneficial. They increase the time to the next exacerbation. They contribute to people's improvement or faster. They limit failures to therapy. Usually, with any exacerbation, we use systemic steroids. Now, if somebody can't get systemic steroids, using inhaled budesonide has been shown to be helpful in this case. The literature shows usually hospitalization, inhaled steroids or budesonide. What's out there, they use nebulized budesonide up to about 4 to 8 milligrams, which usually we use 0.5 milligrams three or four times a day for asthmatics, etc. So, that's a lot of budesonide. But it's been shown to be helpful if somebody can't get systemic steroids.

Now, in the hospitalized patient who is admitted with an acute exacerbation of COPD, I still think the way I read the literature and the way some of my colleagues read the literature, that we just were at an impasse and there really has been no prospective randomized control trial comparing low-dose steroids to high-dose steroids. So, we are shackled with an inability to fully understand to give us this really good data. The last best data out there was from 2010. I think the author's name was [unintelligible [00:36:28]. He's a big data researcher at Tufts in Boston. He showed us that the conclusion from the paper was that oral systemic steroids are as good as high-dose IV steroids. There is a lot of editorial concern about that conclusion. One of them is

that nobody knew what the patient was like before they were admitted to the hospital. This was data that was gleaned from insurers and not from hospital charts.

The patients that were treated with prednisone were older, did not have private insurance, most of them, and it suggested that they may be of lower socioeconomic class. It could be my speculation that they were hospitalized as if they were having an outpatient acute exacerbation perhaps because their socioeconomic conditions, etc., didn't allow them to be treated at home, they were hospitalized possibly.

Now, the other thing is that many of these prednisone patients crossed over from the low-dose prednisone arm to the IV, high-dose steroid arm, about 20% of them, 15% to 20%. It was an intention to treat trial, and that wasn't taken into the final statistics. So, I don't belabor it.

But I think what I would choose is what I have been doing is starting people on IV Solu-Medrol 60 milligrams two to three times a day, which comes close to overlapping with what's considered low-dose steroids, which is often 50 to 80 milligrams.

Meredith: One of the questions that I think sometimes comes up for me is for those who are maybe on the more severe spectrum of their underlying COPD, I think kind of classically. we're all taught five days burst steroids and then they're done. But for those that either seems to have slower improvement, maybe because of the severity of their disease, I've heard rumors about maybe if they're like asthma-COPD overlap, you might consider tapering. But I don't know anything.

Monee: You went there with the taper, girl.

Meredith: I did. I always wonder. My residents always ask, and I don't know what to do.

Jim: Neither do I.

Meredith: Okay, cool. [chuckles]

Jim: Okay, so that's the answer. The literature shows that five to seven days of the same dose, just a burst of steroids, is not inferior to longer periods or tapering. And yet, this comes into some of the philosophy of medicine, is how to look at what is a very good study and how that actually applies to an individual patient situation. The best studies are really valid relative to the subjects that are studied in that paper, but they exclude so many people that it's sometimes hard to legitimately apply the conclusion from these good papers to an individual patient. If they're not ready at five days, they're not ready. You need to either continue your steroids or attempt to taper, etc. I would be with you, Meredith, on judging each individual patient based on their merits, etc.

Monee: Yeah, I think that's a pretty good overview of the steroids. I'm not sure I ever thought that hard about the studies that had been out, so I'm always glad to be reminded that you have to put them in the context of the patients that were studied, and then does that actually meet the patient you're treating.

The next piece is maybe just me being an ignorant hospitalist. If someone's being admitted to the hospital for COPD exacerbation, I just assume they need to be on an antibiotic. Please tell me I am right so that we can move on and then just be done with it.

[laughter]

Jim: Yeah. Antibiotics in somebody with severe COPD exacerbations are probably helpful. The question is, what kind of antibiotic to start? You have your sputum cultures cooking, but you want to decide, firstly, I think, on whether somebody's going to be colonized with Stenotrophomonas or Pseudomonas, etc. Those risk factors are out there if they have a previous culture. If you do know the previous cultures and the patient has shown Pseudomonas, then you have to cover them for pseudomonas. Other risks include if they have bronchiectasis and if they've received broad-spectrum antibiotics within the last three months, they may be at risk both for Pseudomonas and MRSA. Those would go into your decision-making. Let's say the patient doesn't have any of those risks for pseudomonas. Then, you'd have to decide on an antibiotic regimen. Third-generation cephalosporin is very good. Levaquin as a lung-related fluoroquinolone is helpful. Moxifloxacin isn't used anywhere. Or I don't know, do you all use moxifloxacin?

Meredith: Not a ton, but some, yeah.

Monee: At one of the previous Kashlaks I worked at, we did.

Jim: Okay Alright. Anyway, so I haven't seen it use that much. I don't know why we do this, but we often add azithromycin in, and I speculate that it's thought to perhaps have immune modulatory aspects to it and not necessarily broaden out the spectrum of an initial regimen with the third-generation cephalosporin. But that's kind of how institutions and communities gain a fingerprint of management that's one of ours.

Monee: Ideally, yes. I think getting culture results would be helpful. What happens in the situation where you either never get culture results to come back and/or you get a respiratory panel that comes back? I guess start with the first one. You don't get culture results. What do I do with that, with a severe COPD exacerbation?

Jim: Without you ever expecting to get cultures, I wouldn't be very worried about it. About 50% of individuals with pneumonia never grow out a known bug from their sputum, and that may be related to pneumococcus being difficult to grow out. But at least half, if not most, aren't going to give us an answer in the sputum culture. So, we have to empirically treat or hone our antibiotics to the risk factors that the patient has and wanting to be broad enough to limit the risk to those that have severe COPD or comorbidities that are going to make them at high risk for mortality. Age, which is a high risk for mortality. So, you don't want to miss it but you also to be a good steward of antibiotics, don't want to be too broad. Does that get to the point? And then, you have the respiratory panel.

Monee: Respiratory panel. Yeah.

Jim: Is that the viral panel you're talking about?

Monee: Yeah, how does that play in, say, maybe you don't get cultures back, but you do have this positive-- pick-- virus NOS on your RSV [crosstalk]

Jim: What about influenza? What do you think? That would be really the only one that, to me, would make a difference.

Meredith: Just to clarify, the reason you're saying it would be helpful to know about the specific virus is if there was treatment for it. If you found out that it was another virus, would you ever peel off the antibiotics, or are you trying to get the anti-inflammatory effect from them that you would keep it on regardless of the viral panel?

Jim: Well, there's a lot of coinfection because a classic presentation for pneumococcal pneumonia is somebody saying, "I had this viral syndrome, like a cold. The cold kind of went away, but then I got really sick two or three days after that." That's like a classic bimodal presentation of someone with pneumococcal pneumonia. So, I would say I would think about it. If somebody didn't have grossly purulent secretions and we grew a virus out, I would think of stopping my antibiotics more so than if we had no microbiology information, but I wouldn't necessarily on a uniform level, just stop antibiotics.

Meredith: Okay. I think the other mainstay of treatment that a lot of us think about are bronchodilators. Do you want to just start by walking us through what kind of dose, treatment, all that good stuff?

Jim: Yeah. For someone who's hospitalized, we want to start short-acting bronchodilators. We usually do both of them. None of this is based on any data, really. There's not great high-grade data for short-acting bronchodilators, but they seem to help, so we use them. In the outpatient setting, we use short-acting bronchodilators as kind of an indicator of whether somebody is becoming more short of breath or not. So, I ask patients to tell me how frequently they're using their LABA or their LAMA because I use that as an indicator of where their COPD is going.

Cyrus: Do you mean their SABAs or their SAMA?

Jim: Oh, sorry, the short-acting bronchodilators, yes. Not the long-acting. So, albuterol use or ipratropium use, if it starts increasing in frequency, then I have some idea that they're more short of breath, and I may be prone to treat them as if they're having a slow burn or an exacerbation of COPD. When it comes to long-acting bronchodilators, I think the GOLD criteria spells it out pretty well. If we're going to pick one to use, it would be a LAMA over a LABA or LAMA-LABA for GOLD 3 or GOLD 4 for patients. The LAMA seems to have a better track record with limiting exacerbations, etc.

Cyrus: What do you do with their home inhalers? Do you just hold them?

Jim: I would want to see them on a LAMA and LABA in the hospital certainly if their home meds include that, even if they don't include that. But how they're delivered, usually, I'd like to see them delivered through a nebulizer in the hospital. That's addressing the issue of whether an MDI is as helpful as a nebulizer. Yes, in the outpatient setting, the literature shows they're equivalent in medication delivery. But in a patient who is maybe a little delirious, they're very short of breath, they're maybe not be that cooperative with an MDI system, I would like to see them on a nebulizer.

Cyrus: Okay, I think that's a great overview of steroids, antibiotics, and bronchodilators, which I think are all mainstays of therapy. But with the time we have left, obviously being respectful of your time, Jim, I do want to talk about a couple of other things, in particular, oxygen therapy. Obviously, there's an array of options that we can use, but I'm particularly interested in how you use PAP therapy, whether it be continuous or bi-level, and how you apply that on the inpatient setting. Who are your candidates for PAP therapy? What are your endpoints? That sort of thing. So, maybe you could walk us through your process.

Jim: Sure. First of all, I think you need to give enough oxygen for the patient to survive. One of the reasons I say that is because it sometimes can be mistaken that by giving oxygen, you may contribute to hypercapnia, which may be true, but you also need to give enough oxygen for the patient to survive. So, you do want to target a goal of 88% to 94%, let's say. You don't want to go above that because of the Haldane effect and ventilation and perfusion mismatching issues. So, given that there's enough oxygen. The second thing that you didn't mention, Cyrus, and I want to add, is heated high flow.

Cyrus: Yes. Huge fa. Thank you. Yes.

Jim: Yeah, because that's been shown to be quite helpful. It allows for higher flow for O2 where patients need higher FiO2 or O2 flow. But, also it does actually diminish the work of breathing, and it does that probably by pressurizing the airways by about 5 to 10 cm of H20. And it's quite helpful. Also, I don't know the mechanism behind this, but it's been shown in some correlative studies that it limits readmission. So, it's a good thing to use on the floor. When it comes to PAP therapy, I think there are some things that one wants to pay attention to, to try to prevent somebody from needing the ICU. One of them is certainly if they're complaining of being tired or short of breath and tired of breathing, probably need the ICU, maybe would do okay on a step-down unit with BiPAP or CPAP.

But then, others, hypercapnia, respiratory acidosis, just as treatment of COPD, this isn't necessarily on the respiratory side. We know that people with COPD who get noninvasive partial pressure ventilation actually do better. So, it's kind of a treatment as well in the COPD setting. They get out of the hospital earlier, they feel better, they turn around better. I'll say this, even CPAP can diminish work of breathing and be helpful in patients with acute exacerbations of COPD. Most of the studies have been done with BiPAP or a volume-targeted PAP of some kind. But you can imagine somebody having airtrapping and their alveolar pressure at the end of exhalation is about, let's say, plus 10 cm of water. They have to generate negative 11 cm of water to get any movement of air into their lungs. If you put a CPAP mask on them and you match that end-expiratory pressure at the alveolar level with CPAP, that means they have to pull negative 1 cm of pressure to get movement of air. So, that essentially diminishes the work of breathing significantly. CPAP can sometimes be easier for a patient to sync with as opposed to BiPAP. But in the end, if you're going to choose one or the other, I'd probably choose BiPAP but I wanted to go through the CPAP thing.

Cyrus: I think it's helpful because we often don't think about-- I think a lot of folks are dogmatic in that it's like, "Okay, we're going to use CPAP for heart failure. We're going to use Bi-level for COPD because of the Delta P," if you will. So, I think it's helpful to look at it from that perspective. The other thing that I want to make sure that we're clear on is really the purpose of in this case, let's say, bi-level therapy is to decrease work of breathing, enhance minute ventilation, and then hopefully by doing so, improve that acute hypercapnia. Is that a fair statement?

Jim: Yeah. The Delta P will help with minute ventilation, which will help blow off more CO2. The CPAP part of that is the one that diminishes the work of breathing, which then diminishes the generation of CO2. So, by using BiPAP. Yes [crosstalk]

Cyrus: Got it.

Jim: -used, both of them.

Cyrus: And just for maybe [crosstalk]

Jim: Mechanism.

Cyrus: To be clear, when I talk about Delta P or we talk about Delta P, we're talking about that end-exploratory pressure, for that baseline pressure and then your inspiratory pressure is going to be higher than that, resulting in "Delta P". Just for the sake of clarity, I guess it probably would be helpful since we're talking about a patient in the progressive care unit, is there something, in particular, you look at when you have a patient on, let's say, bi-level, and you're sort of worried, like, "Okay, this could go one way or the other," in regards to mechanical ventilation, invasive ventilation versus, "Okay, this person is going to get better." Is there a time course that you're like, "Okay, I'm going to give this person 2 hours, and after 2 hours. That's that"? What's your, I guess, endpoint of treatment for Bi-level on the inpatient side?

Jim: Good question. I would work with the respiratory therapist to start BiPAP and the one thing I would be most mindful of at that time would be their anxiety, their ability to synchronize with the BiPAP because that's frequently a hurdle and we're not really equipped on the step-down units for the amount of sedation that we can give somebody in the ICU. But it may help in trying to avoid benzodiazepines or minimizing benzodiazepines to help the patient with sedation a bit. That would be the first thing.

Then, as I'm moving through the hours, at about an hour or two, I would want to check their arterial blood gas or venous blood gas and I would want to know that it's trending the right way or at least staying the same and not worsening. If it is worsening at that point, I would just-- and it is like an hour or two. I wouldn't let them languish overnight without reevaluating their acid-base status and their CO2 and their O2 status when I'm thinking that I have to make a decision on whether they go into the unit or not.

Cyrus: Thank you. I think that's very helpful. I think that's what I've typically done too, when I'm either consulting on the inpatient side or when I'm doing internal medicine is like an hour or two hours, and you either buy yourself endotracheal intubation or probably you're moving in the right direction. But sort of along those lines, as we move towards discharge, let's say we're getting towards the end of the hospitalization, things have gone well, we're getting ready to send our patient home. I know that Meredith and Monee are going to talk about some pearls here, but I was curious. There is some conflicting data regarding PAP therapy, specifically bilevel therapy, on discharge and how effective it can be in preventing hospitalizations or recurrent hospitalizations. I was wondering if you could speak to, let's say, noninvasive ventilation post COPD exacerbation, what your thoughts are on it? And then, if we're able to use it, what should the goal be? What are we trying to accomplish? Are we trying to normalize the bicarb? Are we just trying to make them feel better? How do you approach that?

Jim: There is a suggestion that there is a benefit after hospitalization with newer literature. The older literature, they didn't really show the positive effect of diminishing CO2 and diminishing readmission for COPD exacerbations when patients need it or qualify medically to get NIV at their home. There are home problems as well, and this goes into the socioeconomic aspects of COPD exacerbations and who gets them. Usually, it's lower socioeconomic-- or often lower socioeconomic patients for many reasons. They develop acute exacerbations and, in this case, one of those might be there's not great home accommodation for having a complicated machine to help a patient with doing something at home that we've been taking care of in the hospital.

They may not have family or friends support or the social support. Often, it's difficult to transition somebody to a skilled nursing facility or a rehab center with noninvasive ventilation.

I don't have a clear answer for this. I think it really is kind of a social problem with medicine vis à vis our COPD patients and patients who have acute exacerbations of COPD. It's a hole in our coverage.

Meredith: Yeah, I think it's a good reminder too sometimes, the difference between what you practically want to do and what you are able to do with the resources provided in the system.

Cyrus: Before we kind of move on, let's suppose it's a kind of different patient. They have private insurance, gold plated, platinum plated. Here is your ventilator at home. Now, I'm the friendly neighborhood internist or pulmonologist, let's say, that's managing it on the back end. What am I looking for? What are we trying to achieve?

Jim: I would say we're trying, over the long run, to work on somebody's CO2. Like you say, Cyrus, their bicarb will change, and that will give us the confidence that we're being successful with that intervention. What I do believe, there is a signal for as well is that it will limit the risk for readmission for COPD, and that seems to be what we all are looking at. And then finally, it will, in a broad sense, probably make the patient feel better.

Cyrus: Thank you. That's helpful. I appreciate that.

Meredith: I guess along those lines, I think one of the things that I often get caught up in also as you're approaching discharge is who should that patient follow up with? Specifically, I feel bad referring everyone to Pulmonology, but then you were just hospitalized with pretty significant COPD. So, who should see pulm? Who should follow up with their primary care? What does that timeline look like? Or is it both?

Jim: What I would say is it would be very reasonable, and we actually want to see this where I practice, that we see the patient as a pulmonary group within two to four weeks after discharge, and then we would make adjustments, make recommendations for follow-up with us, but then hopefully coax the patient back to their primary care physician. The most risky part for a patient is about four weeks out from discharge, and early follow-up is probably the most significant intervention. In some cases, pulmonary rehab may be helpful after discharge, but I think the literature is still a little iffy on that. We're actually trying to contribute to the literature in that regard.

Monee: I think in the interest of making sure that we all get to sleep at some point this evening, I think it'd be good to kind of just do some take-home points because I think we've covered a lot of ground that's been great, but just maybe three take-home points that you would want us to take away at the end of this.

Jim: Oh, okay. I would say that prevention is very important, and education is important. We kind of put prevention towards the end and it may be more effective towards the beginning. When we're talking to our patients in the office, talking about smoking, talking about the use of inhalers and appropriate, effective use of inhalers, the right inhalers, and then how you try to jigger our ability to limit their risk for exacerbations based on the GOLD guidelines.

Secondly, when it comes to hospitalizations, look for PE. I think that's a pretty common thing that sends an older former smoker with comorbidities into the hospital with an acute

exacerbation of COPD. When admitting them, broad-spectrum antibiotics, keep in mind pseudomonas and MRSA and risk for colonization. If that risk exists, then we want to cover the patient initially with antibiotics that would cover those bugs.

When it comes to systemic steroids, I think there is still what we call equipoise out there where we don't know exactly where to land on the steroid issue. We do know that people do better with systemic steroids when they're hospitalized as opposed to placebo. It's just the dosing. We've seen a trend in dosing in 40 to 80 milligrams a day, which would be a lower dose. Some of us, including myself, think perhaps that a higher dose may be more effective. Yet there is a risk for diabetes and suppression of inflammatory processes and things like that, but we still don't know.

On discharge, low threshold for noninvasive ventilation if people meet criteria. Frequently, it's if they present with a PCO2 of 45 or greater. In some of the test literature, I believe that Medicare is reimbursing with 52 cm water or greater. One would have to check that with their local Medicare system since each state is a little bit different.

Cyrus: And we will be back with our lightning round.

Monee: We're going to just start by getting to know you a little bit. Would you mind starting with your one-liner?

Jim: You got it. I'm a 62-year-old physician who loves medicine, seeing patients and enjoy my family and friends. Also, I've been quite a traveler through my life. Just recently, I got a master's degree in the philosophy of medicine at King's College in London.

Monee: Man, that's like taking that whole lifelong learner thing to the next level, I have to say.

Jim: Yeah, I'm never going to quit.

Meredith: [laughs] What's next then?

Jim: I don't know. Actually.

[laughter]

Jim: I think staying at work for a while.

[laughter]

Meredith: All right, well, I guess with the lifelong learning mentality, do you have any favorite advice or feedback that you've ever gotten from someone during your training or career?

Jim: First of all, I've gotten a lot of advice and most of it has been very helpful. The one that sticks out is from an attending I worked with when I was a resident who just said, "Never forget about the patient." I think that's important. It has been to me because through my career, and I know others will encounter this, your academic pressures, social economic pressures can take your mind off what we're really there for, which is patient.

Meredith: Wonderful advice. I love it. That's usually my guiding principle too. So, I support it. I think, just for sake of time and because Monee and I have been hanging on to this pick of the week for a while, we're going to jump to pick of the weeks.

Monee: Meredith, it's all you this week.

Meredith: It is. [chuckles]

Monee: Please tell. This is so good. We're going to definitely have a link for this one.

Meredith: Yeah. So, we'll put the link in the show notes. Actually, Cyrus and I were talking about this before the air that I'm from Texas, and I don't know if anyone's had the extreme pleasure of watching the Officer Big B Fort Worth Police Department recruiting video. I don't know if either of you guys have, but it's fabulous. It is one of the funniest things I have seen in a long time. They essentially spoofed like a used car salesman commercial.

Monee: Complete with neon chirons.

Meredith: Yeah. People pretending to be the balloon people that go up and down. This is going to be great on video, I just realized. Up and down. Monee and I were having a very bizarre day at work, and I just had seen it the night before and so were watching it on repeat for a full day, so it's brought much joy to us and we'll put in the show notes for everyone else too.

Monee: Because it hasn't happened in a while, I will point out that this is so viral that Kelly Clarkson talked about it on her talk show.

Meredith: Yeah.

Monee: I just have to say.

Meredith: And it wouldn't be a complete show tonight if we didn't reference Kelly Clarkson at least once.

Monee: Because it's been a few episodes. I've been pretty good about it, I have to say.

Meredith: Yeah.

Monee: Okay, enough of that. Cyrus, do you have a pick of the week for us?

Cyrus: Picks of the week? Oh, man. Yeah, I have a lot. Well, I guess on the heels of taking the critical care boards yesterday, which was probably one of the most painful experiences of my life, I'm enjoying an adult beverage, a high noon. Big fan. Drink responsibly, drinking in moderation please, guests and listeners. But I feel like I earned it after going through the pain and suffering that was the ten-hour ordeal of the critical care boards, I could just go on and on, and on about the experience, but I will spare everyone that. I guess if I can throw in a bonus pick of the week, I did take some time to redo some pleasure reading, which I haven't had a lot of time for recently. I'm a big music person. I'm a big Chili Peppers fan and I actually got to see them at ACL this past time.

Prior to that, I read Anthony Kiedis' *Scar Tissue*, his autobiography. It is a true miracle that man is still alive. That's all I can say. [Monee laughs] Worth the read, if you like the Chili Peppers.

Monee: For sure. I saw them live a few years ago and remember thinking, "I think maybe the touring thing maybe should slow a little bit for real." [chuckles]

Cyrus: It's crazy. It is crazy. What an insane life that man has had. Again, just like the fact that he is still alive is mind blowing. But there you go.

Monee: I'm pretty sure.

Cyrus: The human body is tremendously resilient.

Meredith: So, this has been another episode of The Curbsiders, bringing you a little knowledge food for your brain hole.

Monee: Yummy.

Meredith: It's great. That was excellent. Get show notes from *thecurbsiders.com* and sign up for our mailing list to get our weekly show notes in your inbox. Plus, twice each month you'll get our Curbsiders Digest recapping the latest practice-changing articles, guidelines, and news in internal medicine.

Monee: We're committed to providing high-value practice-changing knowledge and to do that we need your feedback. So, please subscribe, rate, and review the show on Apple Podcasts or on Spotify, or email us at *askcurbsiders@gmail.com*, a reminder that this and most episodes are available for free CME credit for all healthcare professionals through VCU Health at *curbsiders.vcuhealth.org*.

Special thanks to our writer-producer today, Dr. Cyrus Askin, and to our whole team. The Curbsiders is produced and edited by the team at Pod Paste. Elizabeth Proto runs our social media. Stuart Brigham composed our theme music. And until next time, I've been Dr. Monee 'Not Money' Amin.

Cyrus: I've been and hopefully will continue to be Dr. Cyrus Askin.

Meredith: And as always, this has been Dr. Meredith Trubitt. Thank you and goodnight.

[Transcript provided by SpeechDocs Podcast Transcription]