Talking Sleep Season 4
Episode 18
Oct. 7, 2022
Dr. Safwan Badr, Guest

Episode Transcript

DR. SEEMA KHOSLA: Thank you for joining us for Talking Sleep, a podcast of the American Academy of Sleep Medicine. I'm your host, Dr. Seema Khosla, medical director of the North Dakota Center for Sleep in Fargo.

So we had an episode almost two years ago that's been our most popular episode at over 4,000 downloads. It was about the diagnosis and treatment of central sleep apnea.

So today we would like to revisit central sleep apnea, especially because it can be a confusing and complex disorder. To help walk us through it is Dr. Safwan Badr. Dr. Badr is a past president of the AASM and currently serves as professor and chair of internal medicine at the Wayne State University School of Medicine and is staff physician at the VA Medical Center in Detroit. Dr. Badr currently serves on the board of directors for the American Board of Internal Medicine. Thanks for joining us today, Dr. Badr.

DR. SAFWAN BADR: Glad to be here.

DR. KHOSLA: So you published your review on central sleep apnea a few years ago, and I'm hoping you can take us through it and help us to understand when it is a problem versus an innocent bystander. So what is the relationship between central sleep apnea and obstructive sleep apnea?

DR. BADR: Great question. Well, we tend to think of central sleep apnea as a footnote or or a small separate disorder. And I would posit that central sleep apnea should be viewed as a headline. And that the relationship between central and obstructive sleep apnea is very tight from a pathophysiologic standpoint. These two conditions are very much intertwined. Now, I'm going to take you back a little bit in history, but go into the early eighties, and there was the pioneering work of Onal and Lopata at the University of Illinois in Chicago, where they actually looked at periodic breathing and they noted that upper airway obstruction happened at the nadir of ventilatory drive.

So as you decrease drive, and again central apnea is the extreme form of decreased drive, the airway obstructs. Several groups of investigators tried to induce that experimentally by inducing periodic breathing with hypoxia, which is what happens to us when we go to high altitude. Individuals who have unfavorable upper airway anatomy developed upper airway obstruction at the nadir of drive. So clearly it says that as you decrease drive the upper airway tends to narrow.

We've done some experimental work on this one. We give hypercapnia or hypoxia and upper airway patency improved. But then years later, we did another study where we looked at patients who had pure central apnea with fiberoptic endoscopy of the upper airway. And lo and behold, the upper airway was obstructed in these individuals during pure central apnea. When we

induced the central apnea, we noted the same phenomenon, that the airway was progressively narrowed even in healthy individuals. So I think drive and upper airway patency do go hand-in-hand. That's why I like to think of breathing instability and upper airway patency as two interrelated phenomena and not as two separate parallel tracks.

DR. KHOSLA: Oh, so that's really interesting. I think I remember reading something about sort of the accuracy of diagnosing central sleep apnea in somebody with a BMI, I think greater than maybe 45 or something. And it had something to do with the inability to move the chest and abdomen. But then when they looked at the airway, it was obstructive, even though on on the study, right on the polysomnography, it looked central. So this is so this is interesting. I thought that was maybe an isolated population. So tell me a little bit more about the sort of the basics, like the basic mechanisms of what happens with central sleep apnea.

DR. BADR: So so central apnea is there are multiple pathways for central apnea, but the one that's most relevant to us is the dependance on CO2 levels. So during wakefulness, if we hyperventilate and lower our CO2, we will not stop breathing because we have a cortical drive on top of all of this. But during non-REM sleep, and this is specifically to non-REM sleep, if we decrease the CO2 by a certain amount, we will go apneic.

And I always tell when I when I talk about central apnea, I can induce central apnea in every human in non-REM sleep if I lower their CO2 enough. What varies is how much we need to lower the CO2 and and that magnitude of hypercapnia is what's now called the CO2 reserve. Now, the original study on this was done by Skatrud and Dempsey in 1983. And basically exactly what they did, they used nasal mechanical ventilation to lower the CO2 and determine what's called the apneic threshold, which is reproducible the same way we talk about a pcrit in obstruction we can talk about apneic threshold in central apnea. It's a parallel concept.

DR. KHOSLA: So is that why central sleep apnea is more common in non-REM sleep?

DR. BADR: Yes. Well, non-REM sleep. See, this is the difference and this is a way sometimes I can tell that events such as hypopneas are more likely to be central — when they disappear in REM.

DR. KHOSLA: Oh, sure. Yeah.

DR. BADR: Because REM sleep ventilatory motor output is actually higher. And and there is the problem is that it's very difficult to do the same type of experimental paradigm in in REM sleep because it's a finicky sleep. If I tried, the way we induce central apnea is we hyperventilate someone for 3 minutes using nasal mechanical ventilation with a BPAP machine. And I cannot if I do that in REM, people will not stay in REM. So I've been trying it for about 30 years and I can't get enough trials. People don’t stay in REM, it’s a finicky sleep state.

DR. KHOSLA: Oh, that's really interesting. So. Okay, so then why is central sleep apnea positional?

DR. BADR: Well, this is this is a fascinating area and there are multiple ways of thinking about it. The closest to the most likely, in my view, and I don't have the answer. Okay. The closest I

think of is that when we are supine, our lung volumes are smaller. And this is especially in in people who are obese individuals. And when when lung volumes are smaller, what we call the plant gain is higher.

So there are two ways of to get into this idea. If we think of it as a as a controller system, if our plant gain is higher, that means for a given degree of a minute ventilation, our CO2 will go down further. The closest way I can look at this idea of blood gain, think of a room you are trying to cool or to heat. A smaller room is more likely to respond to a change in the furnace activity.

DR. KHOSLA: Oh, sure.

DR. BADR: Okay. And smaller lung volume is more likely to change the CO2. So that's one possibility, is that it's it's increase is higher planting. And this may also explain why giving CPAP in about 50% of central apnea patients will treat the condition because you're increasing lung volume.

Now, there are other mechanisms that are a little bit peculiar. One of them that has not been shown in humans, but it's at least in animals. If you deform the upper airway, you induce central apnea in animals. So applying negative pressure in the up to the upper airway. Whether this works in humans or not, I don't know. But I haven't done the experiment. But there are multiple pathways to lead to this. But I think the supine dependent is real. By the way, this was described for the first time by the same group that described or developed CPAP by Sullivan and Issa in 1986.

DR. KHOSLA: Oh, wow. I didn't realize that.

DR. BADR: They actually they're the ones who first identified that central apnea responds to CPAP and has a positional dependance.

DR. KHOSLA: So what else then impacts central sleep apnea? I mean, is there is it in older individuals, younger individuals, males, females?

DR. BADR: So this is that this is the beauty of this is that the central that propensity to central apnea varies across populations. That's one. And that variation seems to parallel the epidemiology of sleep apnea in general.

DR. KHOSLA: Oh, does it really?

DR. BADR: If you take and this was, when we were doing some of these studies on fiberoptic imaging, the upper airway, we're trying to induce central apnea. So we weren't really particularly paying attention to how easy or difficult it was, but we noted that in women it was taking us a lot longer to induce the central apnea. And we're anxious. We just wanted to image the airway and move on, and then we decided to test it systematically. And sure enough, we found that premenopausal women are more resistant to developing central apnea than men of comparable age. And that was there was about a 1.3 tau difference between men and women. And initially we said, well, that means is probably everybody says progesterone. So we studied women in the luteal and follicular phase, but it wasn't different.

DR. KHOSLA: Oh, wow.

DR. BADR: But then we said, okay, then it must be testosterone. So we took women, and we gave them testosterone.

DR. KHOSLA: And?

DR. BADR: Sure enough, the apneic threshold moved exactly like men.

DR. KHOSLA: Huh.

DR. BADR: And then we took men and gave them Lupron…

DR. KHOSLA: And?

DR. BADR: And it moved exactly like women.

DR. KHOSLA: Oh, for goodness sakes.

DR. BADR: So this tells us that the apneic threshold is actually dynamic, it has plasticity, which means it responds to manipulation. So the idea that central apnea is not common in women is not it's not new. I's actually was found in the large epidemiologic study in Hershey, the Hershey study, which was I think it was published 99 or 2000. There are two of those. And clearly, in the abstract of it, it says paucity of central apnea in premenopausal women. So clearly there is there is a difference between men and women.

Now, as we age, our propensity to central apnea increases. And that's why we see central apnea in older adults. But in women, it's even more complicated because there is a menopause effect. And menopausal women tend to have more central apnea than premenopausal women. So so the story is a little bit variegated and complicated. It defies simple linear analysis, say this or that. But clearly there are demographic changes that that affect us with with at least with age and gender.

DR. KHOSLA: Huh. Okay. So then what about post-arousal centrals? Do you think of them any differently?

DR. BADR: Well, I so I make a distinction here between the scoring stage and the review stage.

DR. KHOSLA: Okay.

DR. BADR: I view my role as interpret the polysomnography in light of all the available clinical literature, clinical findings. So I pull their PSGs and imaging or anything else I find in the record to actually interpret it. So I capture all of these when they're being scored, but then I when we decide what they mean, I make a distinction in my mind between two types.

So there are types where we have an arousal, by the way, arousal after event is a normal physiology. It's if it's an expected physiological response. Now, is it contributing to the subsequent central apnea? If I see a period of hyperventilation. And again, you need you need some time of this hyperventilation. Think of we're decreasing the arterial CO2, that low CO2 has to travel all the way to the medulla. It's not going to get all the way to the medulla in five breaths.

It needs somewhere between 2 to 3 minutes to get there. So if I see that pattern, which is what you see in Cheyne-Stokes respiration, then the arousal is contributing. There are times when I see, and I think this is the one with at least many of my colleagues and our trainees identify where they talk about the post arousal. They see a huge breath, and there is an apnea after that. Now, that is a sigh and a sigh is a pause, is a is a is a large tidal volume. And it's maybe that inhibition after that is vagal and not necessarily certainly related to CO2 that is probably physiologic. Now, if that's all I see, I'm prepared to downgrade it and not consider it contributing to the overall picture. But again, it all depends on how many of these we see.

DR. KHOSLA: Huh. So then what about then the sleep onset central apnea. So you you taught me something really interesting that you talked about how when it was paper records and you would score events, all of the events were or all of the events were counted. Right. Even during wakefulness. But now with our digital platforms, we don't score them during wakefulness. So are we missing something if we're only scoring them during sleep? I mean, what do you think about those sleep onset centrals?

DR. BADR: Well, first of all, the whole concept of an epoch is a contrived concept.

DR. KHOSLA: Sure. Yes.

DR. BADR: So. So if I can shift the epoch by half a second and I change it, whether that's, you have 14 and a half seconds, or 15 and a half seconds of wakefulness, shift it by half second and now that central event is captured in that epoch of sleep.

The way I look at it and I know that the system doesn't capture them. As I'm reading studies, I look at those. And if I see a lot of them, that tells me that the person is probably has a high propensity to develop central apnea. And I know our textbooks tend to call it, quote unquote, physiologic. But if it's physiologic, why don't I see that in the majority of patients. So the fact that I don't see it in everyone tells me that I'm not sure it's physiologic, and these are the ones. I capture them, and then I start looking. And quite often I see they have other central events later. So to me, it's a marker.

Now, how do we capture those? I don't really have an answer, except that I view it at least in a holistic fashion. And you're right, they can't be scored because the epoch is scored as as wakefulness.

DR. KHOSLA: Mm hmm. So let's say you have a patient that has primary central sleep apnea. So how do you work them up? Do you have an algorithm? I mean, do you do a urine drug screen?

DR. BADR: Actually, this is one of, I wish we do drug screen on 100% of people who have central apnea unexplained because we're otherwise struggling by labeling primary by definition is idiopathic meaning you have it's a fancy word of saying way of saying, you know, central apnea but I have no idea why. And in that case, I'd like to know more and possibly the same way we do urine toxicology screen before an MSLT, if it’s central apnea we should do it. But but again, that has to be done in a standardized fashion. I don't really have an algorithm, and I've oscillated over the years and pardon the pun, of using oscillation.

DR. KHOSLA: I'll I'll give you that one. That was pretty good.

DR. BADR: And and so for a period of time, I got wanting to do more imaging. I've done brain MRI and that, but I found nothing. I mean, there's no other symptoms. Probably. I don't want to waste resources. I'm not finding anything. But if somebody has a headache or has something to make me suspect a chiari malformation, then it's probably worth doing.

I think in the vast majority of patients, we we find we find nothing. And I've seen I've seen central apnea in young, healthy men whom we're screening for research studies. We will know anything and we find central apnea in them. And what does that mean long term? I don't really know. So I, I don't really have one. But most of the time, central apnea that I see happens in the context of, broadly speaking, sleep-disordered breathing. They have other events. So I rarely have to make that pause and say, okay, I. I have to decide whether to treat it or not. There's another reason to treat it, usually.

DR. KHOSLA: Mm hmm. Okay, so that's really interesting. Tell me more about the young male with central sleep apnea and no symptoms that is just being tested for. For what? Research?

DR. BADR: Yeah, we do research studies that for healthy adults. And for years, most of our volunteers were college students or medical students. And occasionally we find someone who's perfectly healthy and they have central apnea and they are scared. And they come and he came and talked to me and said, Well, you're not hypertensive, you don't have any other medical condition, you're asymptomatic. And this is something to just simply be aware of. And I don't know what the natural history of something like this is.

DR. KHOSLA: Well, that's what I was wondering. Do you see them then? Have you followed any of them out? Do they develop?

DR. BADR: I haven't. What I told my one of them, I told them. Okay, well, I guess healthy living is your way forward. Maybe if you don't gain weight and develop obstructive apnea in the process and just healthy living, that's all I can say and just be aware of it. And again, since this was not triggered by a health concern, I don't know what it means.

DR. KHOSLA: That's fair. That's true. That's true. Because how how do you know what you know, quote unquote normal is across a population.

DR. BADR: Yep.

DR. KHOSLA: Oh. So then. Okay, so they don't have narcotics. They don't have heart failure. They don't have any sort of symptoms that make you think of a chiari malformation. Then what do you just sort of…

DR. BADR: I just left them alone. I mean, they weren't there wasn't there wasn't a huge number. But this particular individual was very concerned. But it wasn't it wasn't a large number to make him concerned, because otherwise I probably would have pursued a more a more systematic evaluation.

DR. KHOSLA: That's fair, huh? So let's take a short break. And when we come back, we'll talk more about the diagnosis and treatment of central sleep apnea. You're listening to Talking Sleep from the American Academy of Sleep Medicine.

AD BREAK

DR. KHOSLA: Welcome back to Talking Sleep. Our guest today is Dr. Safwan Badr and we're talking about central sleep apnea.

So then if we were to look at outcomes or clinical relevance or maybe maybe clinical importance is central sleep apnea as worrisome as obstructive sleep apnea?

DR. BADR: Well, this all depends on the the definition. To me, I think central apnea, because of the way we classify it, is it's been viewed as kind of a minor side note. I do believe that it's very significant and and it merits investigation and we shouldn't stop breathing at night for whatever reason. And we need to know in every particular individual. And I think we need to approach it methodically and systematically. I do believe in a large number of patients we will find coexisting obstructive sleep apnea. So I think they go hand-in-hand.

DR. KHOSLA: Huh. And you described it as the Cinderella. What do you mean by that?

DR. BADR: Well, because it's kind of ignored. It's like it's like it's it's like the stepsister of the attention goes to the loud obstructive apnea and the quiet little sister just doesn't get any attention. And and I and I think we need to pay more attention to it. I think it's a driver of sleep-disordered breathing, not a not a minor footnote.

DR. KHOSLA: So not just a marker, but a physiologically important.

DR. BADR: I think it's physiologically very important. And it and it may tell us a lot about ventilatory control and other aspects any given individual.

DR. KHOSLA: So you kind of talked earlier about why CPAP works in central sleep apnea and you suggested that maybe it was by increasing lung volumes. Is that the only reason?

DR. BADR: Probably multiple reasons. Increasing lung volume would be one of them. The other one is CPAP opens the upper airway. And we mentioned something about upper airway narrowing and obstruction. And and by opening the upper airway, it may dampen the after shoot. The overshoot that happened after an event. The other thing, which is equally important, I don't know if it's equally important, but this is important is is the fact when you increase lung volume, especially in someone whose oxygenation dropping a little bit, you increase oxygenation. When you increase oxygenation, you're turning off the carotid body. And the carotid body, the peripheral chemo responsiveness is maybe one of the most destabilizing factors in respiration. Because the carotid body responds quickly for people to talk about system one and system two. This is a system one. It's a survival thing. My oxygen is dropping. Within 10 seconds I'm going to, I have to breathe. So. So by doing so, it lowers the CO_2 very quickly. So it's destabilizing. So I think CPAP increases lung volume, opens the upper airway, increase oxygenation and hence dampens the carotid body response. So decreases decreases all of these factors. So I think it works on more than one pathway.

DR. KHOSLA: So what are the other treatment options that we should consider for our patients with primary central sleep apnea?

DR. BADR: Well, we have we have a variety. The other treatment that have been tried and they every most of these studies are fairly limited. And most of the studies in central apnea, just to frame them, were in patients who have central apnea and heart failure. This is where the bulk of the studies were. So CPAP is the very first one that would describe people tried BiPAP and it and it works but there's one careful word of caution with BiPAP is that BiPAP in and of itself may cause central apnea.

DR. KHOSLA: Yes.

DR. BADR: So I actually all my experiments that we do in our lab to induce central apnea uses BiPAP, that's how we do it. And and and it's the same idea because that pressure support level is what lowers your CO2. So BiPAP would probably have to be done in the context with with it with a backup rate.

The third modality, which is adaptive servo ventilation, which originally was described for or developed for heart failure, is another mode that we use again. And now we know that we shouldn't be using it in patients who have central sleep apnea, heart failure with reduced ejection fraction below 45%. So now now we know from from the literature, but it can be in in primary central apnea or somebody who had normal ejection fraction. It can be used.

One of the things I'm becoming more and more interested in is combination therapies. And we've done this in in patients with used oxygen with CPAP. Now, the problem with oxygen is that if there is no hypoxia, you may not be able to to get it covered because you need a certain level of hypoxemia Sso you can actually use it. Hypoxia is very appealing because it works on multiple pathways. One of them is it dampens the carotid chemo reflex sensitivity, that's one. But the other one, which is fascinating literature, is that if you give if you give oxygen, you increase brain CO2, slightly effect, you displace that CO2. So so it has multiple ways of responding. I think it's a very nice physiologic response. And I know there's been a lot of interest in in oxygen for central apnea, but we've used it whenever whenever payer allows us combination with with CPAP. And the last one that I've actually been using is acetazolamide.

DR. KHOSLA: I wondered about that. How do you use that? How does it work for you?

DR. BADR: Well, I use it. I use a is a low dose, 250 milligram only. And I use it with CPAP.

DR. KHOSLA: Oh, you do?

DR. BADR: Yeah, so I do not. Again, the vast majority of patients with central apnea have coexisting obstructive disease. That's just reality in epidemiologic studies. And so I give them CPAP, and then add acetazolamide to it. Again, I do it on a case by case basis and and evaluate them carefully, monitor their bicarb. I've I've had a reasonable success rate, and I'm actually trying and we're studying it now experimentally in a in a clinical trial, looking at the combination of acetazolamide plus CPAP in patients with central apnea.

DR. KHOSLA: Oh, that's interesting. What about the implanted device?

DR. BADR: Implanted devices? Again, it's also all of these things will probably end up every one of those may end up being a a subsegment of the of the population. I think I think it can be tried, but these are the kind of things that have to be in a specialized center where the expertise is there. This is this is this is not everywhere. And again and again, all of these are going to be until we have long term outcome data. One important note, in most of the things that we have, what we have are intermediate outcomes. And so my, my, my opinion is that these things can be tried and we should consider them on a who is the appropriate patient and understanding that at the present time we are still at the stage of collecting long term outcome data.

DR. KHOSLA: Okay. So let's talk about treatment-emergent central apnea. So tell me about the physiology, like what is happening here.

DR. BADR: Well, treatment-emergent sleep apnea. Somebody once told me that if you want new ideas, look at old literature. And treatment-emergent sleep apnea was actually described in the seventies by Weitzman.

DR. KHOSLA: Oh, no way. I didn't know that.

DR. BADR: And then. And then Onal and Lopata published it as well. So there was and actually there is another somebody told me there's even an older Italian study.

DR. KHOSLA: Oh, for goodness sake.

DR. BADR: It goes back into the seventies that people describe. Because, remember, the only treatment for sleep apnea in the Jurassic era of medicine was tracheostomy. So. So people would find that you tracheostomize patients and they. Voila, they still have central apnea. They had obstructive apnea last week. This week its central apnea, flavor of the week. And after you study them later and it goes away.

So so central sleep apnea, why does it persist and why this develop? So there are again, there are multiple potential pathways. The question is, are we inducing is the disease inducing it? Are we inducing it or has it always been there? And we're not seeing it?

Kind of three different pathways. Are we inducing it? Rapid decrease increase in CPAP, a leak or the CO2 is being lowered. The brain is used to a certain level of CO2 and we are lowering it very rapidly. That's one we're inducing it. Is is the disease inducing it? One thing we know that with sleep apnea, you have chronic intermittent hypoxia and and this phenomenon sensitizes the carotid body and increases your propensity to develop central apnea.

In one of our studies, we took patients with pure obstructive apnea, and we saw that their propensity to central apnea, their CO2 reserve, was very narrow. We treated them with CPAP and it normalized. So just having exposure to intermittent hypoxia would make you more susceptible to central apnea. So this is the second one that the disease is causing it.

And the third possibility is that it was always there in a subtle way that we missed it. And I'm gonna tell you how one tells me. For example, you look at this study and you find that there was central apnea at sleep onset, and now you're not seeing it. Two, most clinical laboratories do not have the ability to distinguish central from obstructive. Not because they lack the ability. It's time

consuming. So it's not being done. So if you look at many of the older studies, the hypopneas are always lumped under the rubric of obstructive events. While these hypopneas may be central for all we know, and then many times I find that there are a small number of central apnea and a whole bunch of hypopneas.

So. So, again, we are inducing it. The disease caused it or it's actually always been there. So strictly speaking, if it's treatment emergent, it should not have been there to begin with. But. But it's hard to make that distinction. But, yeah, there are multiple pathways. But at the end of the day, it's there. And we need to, we need to be cautious and not over treating it.

DR. KHOSLA: So then how do you treat it?

DR. BADR: So my approach is and this is one area when data are few, experts are many. Our approach has been to be a cautious one. So I look at the level of CPAP that eliminates the obstructive apnea and treat it, because the idea here is let's look at the fit, make sure that the fit is appropriate, make sure there is no leak and treat them with with CPAP. And what does this do? The most important thing this will do is that it will eliminate if the disease is of obstructive apnea is causing it. We eliminate that, and then we monitor them frequently, probably for several weeks. We used to bring them back for another sleep, study, another polysomnogram. But many patients don't like to have to come back for this, so we actually rely on tele monitoring, on our remote monitoring, because it's I think it's pretty good. And and if after three months or so this thing went away, we're done, which is probably the natural history in a good number of patients. Now, there are a number of people who would continue to have some. And this is where we could debate and there could be different approaches. Our approach has been on not over treating not not and how am I defining this? Probably an AHI of about 15 or so.

DR. KHOSLA: Oh, wow. Okay.

DR. BADR: And I think this is consistent with with what's with other guidelines. But the question is, around 15, they're not symptomatic. Leave them alone. If it's more than 15 and it's persist and they're symptomatic, then I think then we need to bring them back. And we do either ASV or BiPAP. So this is kind of our approach.

But I'd like to give them time to get them on CPAP, get them to accept the CPAP, use the CPAP, make sure it's still there despite all of these things. Make sure there is nothing else, heart failure, whatever, and then reassess them and have a bit of a high threshold. Now. That's that's probably the a bit of a conservative approach and maybe some others who like to be more aggressive and long term. I have no idea which one would be more correct, but that's our view.

DR. KHOSLA: Hmm. And so symptomatic. You mean persistent sleepiness.

DR. BADR: Persistent sleepiness, disturbed sleep? Yes. So something to tell me that these people are not getting the quality sleep that they that they deserve a need. Yes.

DR. KHOSLA: That's interesting. So one of the conversations I had with my colleagues around SERVE-HF. So we learned from SERVE-HF that we don't use ASV to treat primary central sleep apnea in people with heart failure and reduced ejection fraction, as you mentioned earlier.

But what about then treatment-emergent central apnea is I mean, should we be applying this logic to a different entity? You know, should we be getting echos in our patient?

DR. BADR: My approach is that if they have ejection fraction, 45% or below, I don't distinguish TECSA from central apnea.

DR. KHOSLA: Oh, you don't?

DR. BADR: To me to me, it's central apnea. So to me, it's the device that I would not be using in this population. If it's if they're ejection fraction above 45%. Yes. If ejection fraction below 45%. No, I do not use it. I don't view TECSA as different than central apnea. I think it's a it's simply manifested because now that we open the airway.

DR. KHOSLA: That's really interesting. So you just draw a hard line.

DR. BADR: If it's persistent, that means you've had it. It's no longer treatment emergent if it's continued, if it's related to the treatment or the condition and you treat them, eventually it will go away. But three months later, it's still there. That means you have you have an increased propensity to central apnea.

DR. KHOSLA: So three months is your cutoff. Three months are sort of your threshold.

DR. BADR: Yeah, exactly. If it persists, that means that means you've had it before.

DR. KHOSLA: That's actually that's a really interesting way of looking at it. And I wonder if you then go back to the PSG and you look at sleep onset. Right. And do you see that sort of instability in their in their respiratory pattern? That would be really interesting to look at.

DR. BADR: In many instances, I have. But I have to also be cognizant of the fact that the eyes will see what the mind wants to see or have selectivity in what we remember.

DR. KHOSLA: Okay, that's fair. That's funny. So if you have one message about central sleep apnea that you want to share with our sleep medicine colleagues, what would it be?

DR. BADR: The message is that we need to think of ventilatory drive of central apnea as the headline, not a footnote. It is really a driver in in the whole condition under the rubric of sleep-disordered breathing. And that morphologic distinction that we draw in international borders or firewalls between central apnea and obstructive apnea, I think it's arbitrary and artificial and may not be benefiting our patients.

So if we think of it as an unstable system with an unfavorable upper airway, it will manifest as obstructive apnea, an unstable system with a favorable upper airway. It will manifest a central apnea. It's one system that that is showing you instability.

DR. KHOSLA: Well, thank you so much for talking with us today. So while central sleep apnea isn't as common as obstructive sleep apnea, it is really an important conversation for us to have so that we can better understand the signs and symptoms and understand treatment options.

DR. BADR: Well, thank you.

DR. KHOSLA: Thanks for listening to Talking Sleep, brought to you by the American Academy of Sleep Medicine. For more podcast episodes, please visit our website at aasm.org. You can also subscribe through your favorite podcast service. And if you enjoyed this episode, please take a moment to leave a rating or review. For more feedback or suggestions email us at podcast@aasm.org. I hope you'll join us again for more Talking Sleep. Until next time this is Seema Khosla, encouraging you to sleep well so you can live well.