Patrick Kiessling:

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John Marinelli:

Hey everybody. Welcome back for another episode of ENT in a Nutshell, my name is John Marinelli. And today we're joined by Dr. Raj Dedhia who has done a fellowship in sleep medicine and is dual board certified in ENT as well as sleep medicine. Dr. Dedhia, thank you for being here today.

Dr. Raj Dedhia:

It's great. Let's do it, John.

John Marinelli:

So, in this episode, we're going to cover everything from what's involved in patients getting a formal sleep study, as well as a home sleep study and how to interpret the data, especially if you're seeing a patient in clinic and trying to sift through the sometimes confusing sleep report you get from a random sleep center. But before we get into all that, I just wanted to take a step back and ask you Dr. Dedhia, what is a sleep study or polysomnogram?

Dr. Raj Dedhia:

Yeah. So, the terms can be a little bit confusing. Sleep study is my preferred term because it's more general, a polysomnogram implies something called a type 1 study, which I'm happy to talk about, but in general, a sleep study. You're doing two things in my mind, one is you're trying to diagnose whether it's sleep apnea, periodic limb movement disorder, seizures. You're trying to diagnose something in sleep. And then for our purposes as otolaryngologists, which is generally, we do a lot of sleep apnea work, we want to estimate the medical risk of that disease so we can quantify that, how many times per hour are they having this issue? How low is your oxygen going? Those things help us quantify the risk.

So, we have really four types of sleep studies according to the American Academy of Sleep Medicine. There's really two that we're using in clinical use. The first is a type 1, that's an in-lab attended polysomnogram. And I think we'll probably have some time to go into that in some detail. In the second one that we use commonly is a type 3 test, that's a home sleep study. There are many different variations of home sleep tests. But again, if you remember, the purpose of a sleep test is to diagnose a sleep disorder. And we're talking about home sleep tests we're really focusing on diagnosing sleep breathing disorders, and also to quantify and estimate that medical risk of that disorder.

John Marinelli:

And I think we'll spend the majority of this episode talking about a formal or type 1 in-lab polysomnogram, but with a home study, that's obviously also very important in clinical practice. Why would we get a home sleep study? And what's the difference? And how do we think about that?

Dr. Raj Dedhia:

Yeah. So, the main indications for home sleep tests are you want patients who have a high pre-test probability of having obstructive sleep apnea. And I say that because home sleep tests historically will underestimate the severity of sleep apnea. Why is that? You can imagine when you have a home sleep

test, you're at home, you have a kit, you hit a button, which implies that you're sleeping. That's your sleep time. And when you wake up, you hit the button, it says you're awake. Well, I don't know about you, John, but I don't think I can hit a button while I'm awake or while I'm sleeping. So, what that tells you is that the amount of time that it estimates your sleeping is going to be likely too much. So, your denominator, total sleep time is going to be too high. As a result, we use these indices for sleep apnea, which is how many events you have per hour.

That number is going to be artificially low. Again, denominators inflated so the overall quotient there is going to be too low. So, you underestimate sleep apnea. So, if people have a high pre-test probability of having significant sleep apnea, well, you don't care if their number is 35 or 30, it still indicates sleep apnea. The other use for home sleep tests, and insurance, in truth is really dictating a lot of our ability to choose sleep tests. But let's assume in a perfect world I wanted to choose the sleep test, if I'm monitoring a post-op patient, so they've had, let's say a tonsillectomy and I want to know what was their result. A home sleep test for my purposes is pretty good. It's not perfect, but I like it. I also like it because it puts people in their own bed, and sleeping in a lab for some people is very, very unnatural. They may sleep more on their back. They may just not sleep well. So, to me, there are some advantages of home sleep tests, especially for otolaryngologists.

John Marinelli:

You started to get into this, but just for completeness, in terms of the summary that you get from a home sleep study, what are all the aspects that the data that you end up getting at the end?

Dr. Raj Dedhia:

Yeah. So, in 2020, I think, really, two buckets of home sleep tests. You have flow-based sleep studies and then you have others. And the others really are peripheral arterial telemetry, which I'll talk about. But the flow based study, which is your traditional home sleep test, that has a bunch of sensors. And that's how I think we'll answer the question of what information do you get. Sensors you're going to have on a flow-based home sleep test are going to be an air flow signal, you're going to have a respiratory effort belt that's worn around the chest, you're going to have a pulse oximeter on your finger, you'll probably have a snoring or a body position sensor as well. So, from the air flow signal, which is like a candle around your nose and the effort belt and the oxygen, you can determine if people are having breathing events, apneas, hypopneas, which we'll talk about later I'm sure.

And so, from that, you'll get an index. How many events per hour did they have of apneas? Hypopneas? And you also get, of course, is independent information about the oxygen level. So, to me, this is really about the oxygen and about the air flow. And other study home tests, let's call it, the one that I would say is called the WatchPAT, that stands for watch peripheral arterial tonometry. So, this has different channels. This has a peripheral arterial tone measure, which is basically this cuff over your finger that can look at changes in blood flow to determine if you're having breathing pauses. It has a built in oximeter. It has an actograph to measure how much you're moving, heart rate monitor, body position sensors, snoring. And so, this will give you also an index of how many breathing events per hour, but this had nothing to do with your airflow and everything to do with how much sympathetic tone was being shifted in your finger.

So, it gives you kind of a similar printout of oxygen levels, which you can measure of course, reliably, but also it'll intimate how many breathing events you're having per hour. And that's been validated many times. So, these are both, to me, good options of flow based home sleep tests or this other one. But it's important to know that both of these are going to miss other sleep disorders. So, if

you're worried about something else, you're thinking that maybe something else is in the mix here, you should be really to be careful in interpreting that information.

John Marinelli:

And transitioning now to a type 1 sleep study or in-lab polysomnogram, I think one of the confusing things, if you've never seen this is, is just what that entails for patients, especially regarding patient counseling, when you're recommending this as the next kind of therapeutic route. So, just pragmatically speaking, what does it look like for a patient to get a type 1 sleep study?

Dr. Raj Dedhia:

It looks like Frankenstein. So, people come in and I show pictures to patients of what, especially when you have younger patients like children, you want to show them what to expect. Because the first thing is, let's say your sleep study is at 9:00 PM, you come in at 8:30, get checked in, you bet an almost one hour hookup. So, you're being hooked up to a monitor for almost one hour. You're going to get EEG leads over your head, along your scalp. So, this gooey stuff that gets in people's hair that takes a while to come out. Yeah, this is from the in-lab sleep study. You're going to get stickers, EMG stickers on your chin. You're going to get EOG stickers, which are electro-oculograms around your eyes. You're going to get these thoracic and abdominal effort belts over your chest and abdomen. You've got this sort of battery pack in the middle, that's all the wires are coming over your chest. You've got these stickers on your legs to look at your gastrocnemius and anterior tibialis, particularly anterior tibialis measurements of things like limb movements.

So, hopefully I've painted a pretty gory picture, but that's what happens. So, this is really a cumbersome study. It's called a sleep laboratory for a reason. You are a subject in the laboratory. But it gives us great data so as scientists, we love it, but we understand it's really cumbersome.

John Marinelli:

And I know there's kind of several components of the sleep study when you talked about all those data points that we're collecting and that kind of manifests in different sections of the sleep study, namely sleep architecture, respiratory summary, periodic limb movements, arousal analysis, or sleep fragmentation, and the cardiac analysis. I just wanted to start with the sleep architecture. How should we think about that section and how to interpret that?

Dr. Raj Dedhia:

Yeah. So, when you get a sleep study report and you get several sections, there's really three sections. There's the interpretation page, which is the face page by the doc, that's a one pager about what their impression was. But to me, that's like reading the abstract of a journal article. You're not going to get the meat and potatoes of that study. So, you want to go to the next two sections. The next section is going to be a bunch of tables and these tables have very valuable information as otolaryngologists that should interest us. And the third section is a hypnogram, which is a graph, which is really worth a thousand words. It shows you the entire night synchronized channels, as far as what you're seeing at various time points, are they on their back, do they have more events? That kind of thing.

So, that's again, an overview of a sleep study report. And now John's question was about sleep architecture. Well, that's really important in all aspects of that report. That's telling you things like stages of sleep, how long they take to fall asleep, how fragmented was their sleep. So, I break that down when I'm reading a sleep study into a few buckets, the first is sleep latency. How long did it take the patient to fall asleep? Again, I know this is an artificial setting in a sleep lab, but I usually will use a

patient history to corroborate what we find. If it takes them three hours to fall asleep in the lab, they probably have some pathologic insomnia. So, that's sleep latency. Sleep efficiency is if you take the amount of sleep time over the total recording time, how efficient were they in sleeping? In other words, maybe they fell asleep quickly, but then they were up for an hour and a half a couple of times during the middle of the night.

Well, that's not a very efficient night of sleep. Maybe there's some sleep maintenance insomnia going on there. Sleep fragmentation is really looking at the tracing of their sleep staging and seeing how many times did they pop out of deep sleep. And that could have been because of a breathing event or limb movement, but that's an idea of how fragmented their sleep was. And so, then I think we can talk about the different stages of sleep. That's really the next part of the sleep architecture analysis. Okay. You looked at sort of the big picture, but now let's talk about each stage of sleep. And so John, I'm happy if you want me to just kind of go into the different stages right now.

John Marinelli:

Yeah, that'd be great.

Dr. Raj Dedhia:

So, the first stage of sleep is called stage one, and we use terms, I guess, acronyms, N1 to non-REM1. So, you have two general buckets of sleep. Non-REM and REM. REM is rapid eye movement, non-REM is non rapid eye movement. So, non-REM sleep takes up about 80% of our night in adults. And you have different types of non-REM sleep. Stage one is really a transitional stage. Most of us should not spend more than 5% of the night in this stage one sleep, again, as we are falling asleep. And one way that we're determining sleep on a sleep study is through the EEG. The EEG is a measure of brain activity. Most of you listening have never looked at an EEG in detail. I did not until my sleep fellowship. But you can appreciate the morphology, the frequency of these waves, because as you go from awake to deep sleep, what you see is a progressive, sometimes even linear, slowing of the brain, which makes sense.

If you're active, you're going to have high frequency waves. So, awake is only about 8 to 13 hertz. I'm spitting out these numbers because they may show up on a paper someday 8 to 13 hertz is going to be your awake rhythm. Stage one is now 4 to 7 hertz. These are called theta waves. So, again, slowing down, transitioning to sleep. Then you hit stage two sleep where you spend almost half of your night. And every night stage two sleep is going to be about the same background, 4 to 7 hertz, but you have two important signatures of stage two sleep, K-complexes, which are the sharp upward waves in the frontal EEG's, and you have something called sleep spindles. These are spindles or high frequency waves that you'll see 11 to 16 hertz, often in the central EEG. That's telling you about the thalamus and that it shows some activity that's happening. Again, half your time, stage two sleep, K-complexes, sleep spindles.

We move into now stage three sleep. And this is truly a progression. You start off the night in stage one, you often get into stage two and then boom, stage three should happen in the first hour or so. Because this is, again, stage three sleep is preferentially occurring in the first half of sleep. Stage three is kind of an interesting stage. You see something called delta waves. Delta waves are slow about, 1 hertz. They're slow. These are very high voltage, high amplitude waves. This is really cortical synchrony. So, everything is slowing down, moving really nicely, you get these big spikes. So, this is where you can have snoring in stage three sleep without apnea. It's sort of, my mind, the homeostatic sleep stage where things are just right. It's typically sleep apnea resistant. If you remember from our previous podcast, this is where we see growth hormone released.

Children get a lot more of this than I do. This deep sleep, stage three sleep, preferentially is greater in younger populations. As you get older, it becomes about 20% of your sleep time. But in my one-year-old, it's about half of his sleep time. So, that's stage three sleep. And the last sleep stage is going to be REM. REM is rapid eye movement. This is really fascinating for evolutionary biologists, for ENT's, for different reasons. REM sleep, the EEG looks like you're awake, or you're in N1, it goes back to a higher frequency. So, the brain is starting to ramp up again. This is why we dream in REM sleep, because our brains are active. Our bodies are paralyzed. It's a neurotransmitter called glycine is released in our spinal cord. So, it inhibits our abilities to move. That's why you can't move, but you can dream.

You lose chin EMG tone. That's reflective of that, again, that general paralysis. And this is preferentially happening in the second half of the night, whereas stage three sleep is happening in the first half of the night. This is about 20% of our time across all ages. REM sleep is preserved at about 20% of our night. And again, I mentioned evolutionary biologists because we want to know why do we have REM sleep as far as memory and dreaming, but then as otolaryngologists, you have to remember, this is where we see respiratory instability. Not just because we have this paralysis that's invading our skeletal muscles, but also because you have a decrease in your response to chemoreceptors. So, you can imagine, one of the most sensitive chemoreceptor we have is carbon dioxide. And our carotid bodys can detect when carbon dioxide is low and it tells our body, okay, we got to ventilate. We got to get rid of this carbon dioxide. During REM sleep, there's a blunted response to this. So, that same breathing off of carbon dioxide is not happening. So, that coupled with this muscle inhibition really promotes respiratory instability and worsening sleep apnea.

John Marinelli:

And transitioning to the respiratory summary, can we talk a little bit about apneas, hypopneas? How those are defined, AHI, that sort of thing.

Dr. Raj Dedhia:

Do you have a cup of coffee this morning?

John Marinelli:

I did not, no.

Dr. Raj Dedhia:

Okay, I'm not sure you're ready for this, but I'll do my best. So, this is where it gets in a bit of this alphabet soup and for a resident coming on service and trying to understand all these things. I understand it's not part of our day-to-day otolaryngologists experience or exposure, but these are important definitions when it comes to reading a sleep study, particularly for sleep breathing disorders. So, let me start by defining a couple events. The first is an apnea. An apnea is defined, and this is current 2007 ASM definition, is it defined as a decrease in the air flow by more than 90% for at least 10 seconds. And if you have respiratory effort, that's going to be called an obstructive apnea. If you don't have any effort, it's called a central apnea. Again, central apnea means that your body's not telling you to breathe.

Obstructive apnea is when you're actually trying to move air, but you can't. As otolaryngologists we want to treat obstructive events. So, that's the obstructive apnea definition. Hypopneas, which are generally obstructive, have two definitions. And this is where it gets confusing and this is where it gets to be the case that things can change from decade to decade in the sleep field. But as it stands as of 2007, our definition of hypopnea for the ASM is as follows, a decrease in the nasal pressure by more than 30% for at least 10 seconds with either a drop in your oxygen by 3% or an EEG arousal. An arousal

is a disruption in the EEG where you have three seconds of a really generally very fast frequency that tells you that the brain is sort of waking up. So, that definition contrasts with the Medicare definition, again, think of it as ASM hypopnea definition and the Medicare definition, the Medicare is more strict.

So, the center for Medicare and Medicaid services requires that you have a 30% drop for 10 seconds in airflow, plus a 4% desaturation. So, the importance here, the clinical relevance for those of us that are looking at a sleep study with a patient in front of us in the clinic is, if you have somebody that had a sleep test using a Medicare definition of hypopnea, they may show a much less severe AHI than somebody that had an ASM hypopnea definition. You want to look in the fine print at the sleep lab, what they're using to define hypopneas. The same person can have really mild sleep apnea if they had Medicare, but they have moderate or severe sleep apnea with the ASM definitions. So, that's just hypopneas, apneas. So, the apnea and hypopnea index, the AHI, which is really our currency when it comes to sleep apnea care. This is really the number that people come to my office with. That is a composite of the number of apneas and hypopneas that the person had per hour of sleep. That's our AHI.

I'm going to get into now some more alphabets. So, that is the high yield stuff right now, the AHI. But the other thing to keep in mind is something called a RERA, R-E-R-A. That's a respiratory effort related arousal. These don't meet criteria for apnea or hypopnea, but what they do show is some perturbation in you're breathing, often less than 30%, that is accompanied by an arousal. And so that, you can imagine, is some restriction in your nose or your palate that's causing you to wake up over and over again if you have many of these RERAs. When you add apneas and hypopneas with RERAs, now you don't have an AHI, but you have something called an RDI, which is the respiratory disturbance index.

So, I wanted to sort of end with saying that you have different indices and stratifications for what are disease severity. So, if you have an AHI or RDI less than five that's normal, mild sleep apnea is going to be between 5 and 15, moderate, 15 to 30 and over 30, severe. That is our current stratification for adults with sleep apnea. In kids this is different. Kids normal AHI is less than 1, mild, 1 to 5, moderate, 5 to 10, and severe is more than 10.

John Marinelli:

I know another aspect of the respiratory summary is the patient's oxygen saturation throughout the night and it can be reported in a couple different ways. How should we think about that?

Dr. Raj Dedhia:

So, the oxygen saturation is important because I think of the oxygen as our marker for cardiovascular risk. So, when we look at the oxygen time below 90%, for example, we want to see, that's called a hypoxic burden. So, if patients are having a lot of time below 90%, there is some data to suggest that that may bear an impact on their cardiovascular risk. The other one that I think is, well, that has been more substantiated, is how many times per hour of sleep is their oxygen dropping by 4% or more? We call that the ODI4. So, if somebody has a high ODI4, that tells me they had, let's say, 20 events per hour of 4% drops in oxygen that puts them at excess cardiovascular risk for things like high blood pressure, diabetes, stroke, heart attack. So, we want to be very careful of that. So, that's oxygen.

And I think John, the other thing that we think about with oxygen, we talk about a gas exchange disturbance is going to be carbon dioxide. Historically adults, and when I'm reading studies, I don't get carbon dioxide levels unless I'm worried about hypoventilation syndrome, but kids, that's used commonly. It's more sensitive in kids where we can get carbon dioxide levels in addition to oxygen levels to figure out, are they hyperventilating? Which would be potentially a reason to do something like a tonsillectomy in a child.

John Marinelli:

And transitioning kind of to the next section I wanted to ask you about, we've talked about this briefly before with the limitations of a home sleep study and kind of what a type 1 in-lab sleep study offers, but this section on periodic limb movements, what's important to grasp from that section?

Dr. Raj Dedhia:

Periodic limb movements, I think the first thing I will say is the knee jerk reaction, pun intended, is to think of periodic limb movements like restless legs. And there's a difference here. Restless leg syndrome is a clinical diagnosis. You ask the patient four questions. Do you have an urge to move your legs or arms at night? Is it worse with inactivity? Is it better with movement? And does it prevent you from falling asleep? So again, there's a circadian component. Does it happen at night? Is there this urge that really makes it hard to fall asleep? Is it worse with rest, better with movement? And if they say yes to those four questions, you have your diagnosis of restless leg syndrome. Periodic limb movement is a polysomnographic diagnosis.

If you remember, I said that there was EMGs on your anterior tibialis and that's done so that when you're moving your legs often in a flection pattern, you can see that patients are having these limb movements. And in kids, if they're more than five an hour, it's abnormal, and adults more than 15 an hour. As we think of it similar in some ways to the sleep apnea stratifications.

But this is important because if they have a lot of these movements and they're having arousals with these movements, we might think this could be causing their sleepiness. And the overlap is real. So, restless leg syndrome, when we see adults with abnormal PLMs, about 30% are going to have restless leg syndrome. The other way around, if somebody has restless leg syndrome, about 75%, 80% are going to have PLMs. So again, not the same, but they are related. And so, just think about this when you have patients that are having sleep apnea, potentially treated, but still having other sleep problems. This is one thing that I want to rule out with an in-lab sleep test.

John Marinelli:

And transitioning now to the arousal analysis. We've kind of talked about this intermittently, but could you specifically talk on the arousal analysis involved in a type 1 sleep study?

Dr. Raj Dedhia:

Yeah. In general, like type 1 sleep studies are really where we're going to see the arousals because they do require an EEG for cortical arousals. There is some interest, there's actually a lot of interest, but not great ways to determine sub-cortical arousals that we think probably are occurring throughout sleep apnea when you don't see an arousal in the EEG. But again, standard manuals are going to look at EEG arousals. So, an EEG arousal, you need some preceding stable sleep for several seconds. And then after that, if you see a change in the EEG pattern, again, typically a high-frequency change for at least three seconds, followed by, again, returning back to sleep for 10 more, more seconds, we consider that to be an arousal. Again, they didn't wake up, eyes weren't open. They didn't wake up fully, but the EEG changed enough to tell us about that.

And so, you can see arousals after sleep breathing events, after limb events, you can have spontaneous arousals, patients who have, for example, some fibromyalgia chronic pain. We see more of this arousal signature, but this to me is still academic. We have not seen arousals actually correlate with sleepiness maybe because of the phenotypes of who's getting arousals. But we are aware of arousals, we understand conceptually what they are. And so, we use these clinical practices, again, with a grain of salt.

John Marinelli:

And the last section of the sleep study I wanted to ask you about was a cardiac analysis. How should we think about that?

Dr. Raj Dedhia:

Yeah. So, the cardiac analysis, and when you're reading a sleep study on the actual screen, in the montage, you can look at it and look for things like PVCs, PACs are commonly seen throughout sleep. But if you have a piece of paper with a question saying, what's the most common arrhythmia that you're going to see during sleep apnea? It's going to be this brady tachy arrhythmia. And this is kind of a neat teaching point, that during the event, when you are obstructed, when you're asphyxiating, your body has a high vagal tone and you actually have a bradycardia. Once that's relieved, often with, again, the arousal that promotes your body to wake up and sort of just get with it and you have that nice big breath. You then see this sympathetic tone. So, you have brady arrhythmia, then tachy arrhythmia. And this brady tacky phenomenon throughout the night can lead to things that can upset the cardiac milieu, potentially, things like arrhythmias, these are arrhythmogenic causes.

So, we think about this and if we see a lot of this back and forth, some people have more of this action than others, we do worry about potential cardiac repercussions of the untreated sleep apnea.

John Marinelli:

And last question I wanted to ask you, if we just step away from kind of the nuts and bolts and we just try and bring this all together. You're about to see a patient in clinic, how do you recommend approaching reading a sleep study?

Dr. Raj Dedhia:

Yeah. So, John, I give myself, and I've been doing this for about five years in practice, plenty of time to do this. So, I have the luxury of maybe perhaps being in academics and then having one clinical week where I'm seeing patients for sleep apnea. So, I spend about two hours before every clinic going through all the old sleep records of each of the new patients, because if you have labs reporting in one way, another lab reporting in another way, it can be pretty confusing. And I have a table that I've made that I am able to extract what I think are the relevant and the high yield pieces of information. So, I would encourage somebody else to do this in the same way.

You start with looking at the hypnogram. And again, I know on a podcast, it's hard to teach somebody how to look at a hypnogram. But if you look at a couple of hypnograms, you'll get an idea of the general layout, where you can look at sleep architecture over the course of the night, like we talked about earlier. You can look at breathing events, they often are marked by hypopneas and apneas. And the height of the bars tells you the length of the events. So, you can get an idea. Is this guy having two minute apneas or ten second apneas? And that's important. The next line you'll see is the oxygen tracing. And you want to make sure that you're having sawtooth desaturations, up and down. If you're seeing sustained hypoxia, that is not sleep apnea. Think about other disorders that cause sustained hypoxia, COPD, for example. So, the oxygen tracing can tell you that. Then you have the body position sensor and that'll tell you were they on their back or their side.

And often you can see very nice correlation when you looking across the hypnogram that, oh, they're on their back. Look at that. Look at the oxygen levels, drop, look at these apneas and hypopneas happen. Look at their sleep fragmentation, and then, oh, they flip on their side and it's all better. So, that's another example where you can look at that body position to determine that on the hypnogram that you may not get from the tables. And then there's some cardiac information that you can get as

well. But with those things you can get on most sleep studies it's really quite valuable. So, I start with interpreting the hypnogram and I make my own comments. Then in a tabular format, I'm going to go through and extract the AHI, AHI overall, supine AHI, non-supine AHI. I want to make sure I know what the obstructive apnea hypopnea index is.

I can't tell you how many times I've seen a report called obstructive sleep apnea, but when you dig through the numbers, it actually had half the events were central. Most ENTs would know not to operate on that person if I told you they're half centrals, but if it was covered up in a one liner at the end of the sleep report saying, OSA, unless we took the time to look through the raw data, we would miss that, potentially offer surgery on somebody that wasn't justified. So, I think that's sort of, John, in a nutshell, how I would approach it, is look at the hypnogram, look at the tables, look at those things that we mentioned that are important already, the AHI, the oxygen levels, get some idea of get a good stall for yourself of what this person's sleep apnea is like, because then you can advise them on treatment and then after treatment, you can figure out, was it better? And if it's from another sleep lab, for example, you'll now be able to somewhat compare apples to apples with your standard assessment.

John Marinelli:

And I know in clinical practice, oftentimes we will have sleep studies that come from numerous different centers. Is there anything that you want to watch out for or be mindful of when interpreting different centers sleep studies?

Dr. Raj Dedhia:

John, most centers are now ASM accredited. So, their governing body of sleep medicine has done a site visit so that they are okay, they're doing the right standard. So, generally speaking, it's more of an issue of understanding the layout of the document. That can be difficult because you're used to seeing things a certain way from your lab. And now you don't know where the hypopneas are, what they're reporting. Are they using Medicare or are they using ASM? So, I think you want to look at that in particular, is what is your definition of hypopnea? And that'll help you understand how to interpret it. But I think, otherwise, it's familiarizing yourself with taking a minute to look at the document, seeing where things are, looking at the definitions before you go ahead and launch into it is my advice.

John Marinelli:

All right Dr. Dedhia, is there anything else content-wise that you wanted to touch on before we transition to the summary?

Dr. Raj Dedhia:

No. I think, John, the reason for this podcast, I think, the relevance is that if an ENT is seeing patients with sleep apnea, this is like our audiogram, and I think most of my otology colleagues wouldn't operate on somebody without an audiogram, and taking time to understand the audiogram and repeating it if you need to at your own center so that you're comfortable with it. I think that's really, here in sleep apnea, sleep medicine, just as important.

John Marinelli:

All right. Well, I will now transition to the summary portion of our podcast. I started the episode off by talking a little bit about in-lab type 1 sleep studies, as well as type 3 sleep studies as defined by the ASM, which are home sleep studies. And really, the gold standard test to determine the presence and or severity of OSA is in type 1 in-lab sleep study. Some advantages of a home sleep study is it that may

have more accurately represent patients natural sleep. However, just be mindful that really these tests are reserved for patients with high pre-test probability, given their inherent less sensitivity to the diagnosis of OSA, just because they have tendency to underestimate AHI. And then talked about all the nuts and bolts of a sleep study. Patients have to get an EEG put on, chin EMG, EOG on their eyes, abdominal effort belts. They've got nasal airflow pressure monitors, body position sensor, EKG, and then as well as the two leg EMGs.

So, quite a bit of stuff that patients have to get. We're obviously collecting a lot of data. And then transitioning to interpreting the sleep study covered a whole bunch of different things, but a couple of highlights I'll just mention is an apnea. Apnea is defined by a decrease in airflow by greater than 90% for at least 10 seconds and an obstructive apnea necessitates that there's ongoing respiratory effort during this period. Whereas a central apnea, you won't see that ongoing respiratory effort. Hypopnea really has two definitions and the first more general definition is, a decrease in nasal airflow by greater than 30%, but not more than 90% for at least 10 seconds and an oxygen desaturation by at least 3% or an arousal determined on the EEG. Now, a more restrictive alternative definition that is used in patients on Medicare or Medicaid is that they have at least 30% decrease in nasal airflow, but not more than 90 for at least 10 seconds, but they don't include EEG arousals and the oxygen has to drop by at least four seconds.

So, in this way, these patients might have more severe AHI if using the ASM criteria, but on Medicare criteria appear to have less severe sleep apnea. And then AHI by definition is the number of apneas plus the number of hypopneas divided by the total sleep time. And kind of when you pull it all together in clinic before seeing a patient, it's important to get an idea of their AHI, assessing the severity of sleep apnea, pay attention to their sleep latency, how long it takes them to go to sleep as well as their sleep efficiency and see how representative the study is being cognizant of their AHI by sleep position and oxygen desaturation index, as well as time spent at various desaturations helps to appreciate the cardiovascular risk. And then lastly, looking at the EKG summary. Dr. Dedhia, anything in the summary you wanted to add?

Dr. Raj Dedhia:

You nailed it again, John.

John Marinelli:

All right. Well, thank you so much for being here today.

Dr. Raj Dedhia:

A lot of fun.

John Marinelli:

All right, well, I'll just wrap up our episode as we normally do with a few questions.

The first question for today is, what are some of the primary advantages and disadvantages of a home sleep study? The primary advantages of a home sleep study is that they're cheap, more comfortable for patients, in this way more accurately represent a typical night's sleep for patients, but there are some disadvantages. Number one, they will likely underestimate AHI, really for two reasons, primarily sleep efficiency is likely underestimated just by the fact that the patient has to denote when they have started sleeping and stopped sleeping. So, there's an inherent likelihood that they are probably overestimating the time spent sleeping. And recall, the AHI is the denominators divided by time spent sleeping. And then also just recognizing that hypopneas are typically defined also using an

EEG via arousals and that's not possible during a home sleep study. For these reasons you want to reserve a home sleep study for patients with high pre-test probability of having obstructive sleep apnea.

And transitioning to the next question, which stage of sleep do humans kind of across their whole lifetime, from one til you're no longer here, do you spend about 25% of your night in and why is this relevant to obstructive sleep apnea? So, we spend, over the course of our lifetimes, about a quarter of the night in REM sleep. And this is very significant for obstructive sleep apnea because the natural paralysis that accompanies this stage can exacerbate APNIC events. And similarly, if a patient has abnormally low percentage of time spent in REM, their AHI could be underestimated.

And lastly, define AHI and how it's calculated. So, AHI is apnea hypopnea index as calculated by the total number of apneas plus the total number of hypopneas divided by the total sleep time. And recall, for adults, a normal AHI is less than 5, mild OSA being 5 to 15, moderate, 15 to 30 and severe, over 30. But of course, this is a spectrum and the importance is understanding where patients fall on that spectrum. And also that this differentiates a little bit from children where kids, a normal AHI is less than 1, mild sleep apnea is 1 to 5, moderate, 5 to 10, and severe is only over 10.

That'll wrap things up for today's episode. Thanks so much for joining us and we'll catch you next time.