Update on Obstructive Sleep Apnea: Links to Diabetes and Home Sleep Testing

A Web-based Training

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Introduction

During this training on Obstructive Sleep Apnea and Diabetes you will be hearing from two very distinguished physicians. Dr. Kelly Acton, Director of the IHS Division of Diabetes, will provide an overview and the rationale for discussion of this topic as it relates to diabetes care in American Indians and Alaska Natives. She will also provide some thoughts about diagnosing and treating sleep apnea in the diabetic population in Indian country. Dr. Teresa Green, Sleep Specialist from the Nashville Area, will provide information on the clinical definitions, pathophysiology, prevalence and treatment options for Obstructive Sleep Apnea. The training ends with a discussion of several case studies.

Let's begin with Dr. Kelly Acton, Director of the IHS Division of Diabetes.

Overview of Sleep Apnea and Diabetes

For those of you who may not know, obstructive sleep apnea is a sleep related breathing disorder that is characterized by recurrent collapse of the upper airway, that results in drops in oxygen saturation and transient arousals from sleep. And this is what OSA looks like. You can see the ECG there going on above and you can ignore that. Here's what happens with the air flow. The patient inhales and exhales, inhales and exhales, and then the airway obstructs and when the airway obstructs, what happens? Well their thoracic effort starts to increase and there is this abdominal effort that's going on as well, and something called paradoxing starts where these two efforts are sort of working in tandem to try to open that airway and get that patient a supply of oxygen once again. But until that airway opens, you see that oxygen desaturates and then when the paradoxing ends and the airway opens that patient is free to, you'll see the oxygen start to go up again way out here, but basically the patient does this over and over and over again throughout the night.

Who is at risk for obstructive sleep apnea? Patients who are obese, particularly central obesity; as patients get older the risk goes up; mostly male gender, although it is seen in plenty of females as well. And its been shown in different ethnic groups to be at greater risk - African Americans, Mexican Americans, Pacific Islanders, and Asian. The one thing you don't see American Indians and Alaska Natives here, but I was approached by a pulmonologist here in Albuquerque who came up to tell me that the majority of his patients that he was seeing in the sleep lab were Native American and he had a great concern that there didn't appear to be an awareness of this on behalf of the Indian health system providers. So he was the first one to approach me and said we need to be talking about this and then over the next year it started being talked about over and over again, every diabetes meeting I went to. So I do believe American Indians and Alaska Natives should probably be added in here, but we don't have enough data to know that for sure.

Who else is at risk? People with craniofacial features, such as congenital anomalies, retrognathia, enlarged soft palate and tonsils, macroglossia; patients with increased neck circumference; people who have a positive family history are a 2-4 fold increase and then certain people with endocrine abnormalities. Obstructive Sleep Apnea is aggravated by alcohol, sedatives, sleep deprivation, people who sleep on their back, supine position, respiratory allergies, and nasal congestion.

So there are many sleep experts who are now saying well here's the picture of cardiometabolic risk for diabetes and cardiovascular disease and what we're used to talking about and we're wondering if maybe obstructive sleep apnea should also be added to this list

of cardiometabolic risks and they are starting to show in more and more studies that this may actually be something we should be paying serious attention to.

We know that from some work that's been done that there is less insulin sensitivity in obstructive sleep apnea patients, compared to normal patients. So those with mild moderate and severe sleep apnea showed 27, 37, and 48 % reductions in insulin sensitivity and this is separate from age, sex, or how overweight they are. A higher prevalence of prediabetes and incident diabetes in patients with obstructive sleep apnea was shown in this cross sectional analysis of over 2500 non-diabetic patients. There was a significantly higher prevalence of prediabetes and incident diabetes, when patients had obstructive sleep apnea and this was independent of the degree of obesity.

The prevalence of obstructive sleep apnea in type 2 diabetics from some studies that have been done suggests that maybe nearly 17 million of the estimated 24 million diabetic people in the US will have some level of obstructive sleep apnea. The severity is associated with poorer glucose control in patients with type 2 diabetes. And this is probably why we should be paying a little closer attention to it in our patients with diabetes, because if you look at this they've adjusted for age, gender, race, BMI, the number of their diabetes medications, level of exercise, years of diabetes, and total sleep time. And in spite of all that you still see that patients with mild obstructive sleep apnea have compared to no OSA, have worse control, moderate even more worse glycemic control and severe far more worse glycemic control of their diabetes. So this is really important I think.

This suggests that if we don't treat obstructive sleep apnea it may worsen glucose control and increase the need for more intensive pharmacotherapy in our patients. It also suggests that maybe treatment of sleep apnea could improve glucose control as much as some of our widely used pharmacologic agents. And that may be a way to prevent side effects from medications as well as reduce costs. I'm not sure we haven't really looked at it that closely yet.

But there is if we don't recognize it here is the cycle that people are saying, if you don't treat obstructive sleep apnea in type 2 diabetes, you end up with patients who have increased their sympathetic nervous activity and this worsens their glucose control (see the slides I just showed you). This may increase their need for more intensive pharmacotherapy, may increase their risk of cardiovascular disease, then increase their risk of weight gain, which worsens their obstructive sleep apnea. And you can see how this cycle could just keep spiraling down and down. So we want to intervene at some point.

We know that A1C levels do decrease after treatment of sleep apnea with CPAP, continuous positive airway pressure, CPAP therapy. That's the way that obstructive sleep apnea is treated. This study looked at all patients with diabetes and then patients whose A1cs were above 7 to begin with and what they showed was that there was a significant decrease in A1c levels before CPAP therapy versus after.

So even when you look at healthy adults, what happens if you lose sleep? Well they've shown that people who don't get adequate sleep or don't get enough sleep have altered glucose metabolisms, which increases insulin resistance. They have an up-regulation of their appetite. Maybe they have more time to eat or for some reason they seem to make poorer food choices, which can lead to weight gain. And there also appears to be a decrease in energy expenditure, which may also increase weight gain. All of these contribute to diabetes risk – down here on the left. So that may be what's going on.

So in summary obstructive sleep apnea is associated with insulin resistance, glucose intolerance, and risk of diabetes, and that's independent of being overweight. Obstructive sleep apnea is highly prevalent and we don't recognize in many of our patients with type 2 diabetes. Untreated OSA may worsen glucose control and increase the need for more therapy. Robust clinical trials are really needed to assess the effects of this on glucose control. And sleep duration and quality are potentially modifiable risk factors for this, so we may start to look at those in terms of some of our patients.

What do I think for our Indian health system? I think we first of all need to consider it. Is it something that we are seeing in our patient population? Are you looking for it? We don't know what the prevalence is. We need to ask patients with type 2 diabetes about their sleep quality and how long they are sleeping. And not only ask the patients, but also ask their loved ones, the people who sleep with them or sleep in the next room. Is there a lot of loud snoring going on? Do they notice their family members or loved ones falling asleep during the day? Do they fall asleep at the wheel? All those questions about sleep apnea need to be considered.

We need to consider obstructive sleep apnea in patients with unexplained worsening glucose control. You know they say doc I'm not eating anything different than I ever have before. Let's consider OSA. And then we need to look at what are our local resources for diagnosis and treatment of this. I know that there aren't a lot of sleep labs out in many of our facilities, so how can we get this treated? How can we get it diagnosed and treated? And we are working with a number of folks to try to figure out some of the more innovative ways so that we don't have to send patients to these overnight sleep labs that are going to break the bank on us. There are some newer methods. In fact this week is the Advances in Indian health course and a pulmonologist is going to come talk and I intend to ask her just that question. How can we diagnose this in a more cost effective and easier way for our more rural remote sites?

Clinical Definitions and Pathophysiology

Thank you all for being here today, it's actually tonight where I am. I am Dr. Teresa Green and I am a Sleep Medicine Specialist with Western Carolina Pulmonary and Sleep Consultants in Western North Carolina. And I am actually in Portugal right now because I have taken a little year sabbatical to do a year abroad with my children and so that's why we coordinated this from here

I am going to review some simple definitions to kind get us all on the same page about what we are talking about when we talk about sleep apnea and sleep apnea severity, and I'm going to try to review this little bit about the pathophysiology prevalence and clinical implications, and then touch on some of the new information regarding links to diabetes. And then I wanted to include a little bit about testing, mainly because of the recent addition of home testing in the field of sleep medicine, and the fact that I think probably a lot of primary care practices are being inundated by offers from durable medical equipment companies to do home testing. And so I think it might be helpful to kind of know what setting that would be useful in and how to go about doing it.

So, if everybody is ready I'll go ahead and start with some definitions. And the first definition that I want a cover is, what is an apnea, what are we talking about, when we talk about apneas? Basically, apnea is a cessation of airflow for at least ten seconds in the presence of respiratory effort, and so this is actually an example of what we see on the sleep study, and you have two -- the two top tracings are airflow measured this one at the nose and this one at

the mouth, and then following two tracings are effort, chest effort and abdominal effort, and then oxygen saturation.

And so you can see here that you have airflow initially and then the line goes flat, which means there is no airflow, but there is some effort, so this person is trying to breathe. And in fact, the effort, the amplitude of the waves increases reflecting an increased effort until finally you have resumption of airflow, and that's the end of the apnea. So this repeats itself intermittently and these are all apneas. And one other thing that apneas are associated with that is not shown in this image is that in addition to the oxygen desaturation usually these apneas are also associated at the end with an arousal from sleep and that has a lot of importance later on when we talk about the clinical consequences of sleep apnea.

Why does this happen? Mainly because during sleep, we have relaxation of the muscles and we have sort of a reduction in the tone of the pharyngeal musculature, and so while awake you have an open airway with sleep and the muscle relaxation you have falling back of the tongue and some of the other pharyngeal structures and you have obstruction of the airway with interruption of airflow.

A central apnea in contrast within obstructive apnea is a situation where you have absence of airflow, but in the absence of effort. So this is not a problem with the airway becoming obstructed but rather with the respiratory centers of the brain not initiating breathing at the proper times. And in this situation what you see is you have periods of no flow, but you also have no effort in that corresponding time, and so this tells you that there is no signal to breathe and that's what results in the apnea.

Finally a hypopnea, you can think of as a partial airway obstruction where you have narrowing of the airway causing a reduction in airflow associated with either significant oxygen desaturation - has to be 3 to 4% or an arousal, and over here I put the formal definitions and the top definition is the more commonly used. The second definition we use in people who sometimes do not tend to desaturate as much, but still have a significant amount of sleep disruption because of the arousals that I mentioned. And with a hypopnea what you see is that you don't have complete disappearance of flow but you do have a significant decrease in the amplitude of the flow waves reflecting a drop in the flow associated with continuation of effort and so that's a hypopnea.

So when we talk about Apnea-Hypopnea Index, we are talking about the number of apneas and hypopneas per hour of sleep, and basically that's calculated by totaling the number of events and dividing by the actual time that that patient spends asleep. Obstructive Sleep Apnea is considered to be present when the AHI is greater than 5, but that does not equal Obstructive Sleep Apnea Syndrome. An Obstructive Sleep Apnea Syndrome where you consider that this person actually has the disease, it's usually an AHI between 5 and 15, if it's associated with symptoms such as sleepiness, sleep disruption, etcetera or significant comorbidities like high blood pressure, diabetes, cardiovascular disease, or an AHI greater than 15 regardless of symptoms or associated comorbidities.

And so really it's a spectrum of disease where you have people who snore but don't actually experience airway obstruction, and generally that's not considered to cause much in the way of problems. Upper airway resistance syndrome exists in people who have some increased airway resistance but not enough to cause desaturation and doesn't meet the criteria for hypopneas, but it does cause some sleep disruption and so sometimes these individuals are

symptomatic. Finally, more severe than that is the obstructive sleep apnea and you then have mild, moderate, and severe, and the other end of the spectrum is the very severe cases of obesity hypoventilation where in an addition to nocturnal problems with breathing they also develop impairments in breathing while awake.

This here kind of gives you a picture of moderate sleep apnea, this first graph is sort of a summary graph that we use during sleep studies and it sort of combines all of the information that we gather. The top line is body position because that effects obstruction of the airway which occurs predominately in the supine position. The second graph represents sleep staging with stages 1, 2, and 3, REM and awake, arousals and awakenings. And here are the respiratory events with the red lines representing hypopneas and purple lines usually represent apneas in this particular software, and over here oxygen saturation.

And then when we look at the actual tracing what you see here in pink are the hypopneas. This patient has mainly hypopneas associated with some desaturations here and this also shows the EEG portion of the sleep study indicating up here in the little blue marks, these are the arousals. And so what you see is this person has a hypopnea and an arousal followed by another hypopnea and another arousal, followed by another hypopnea and arousal. So you can see that this would cause a pretty significant disruption in your sleep if every 30 seconds or so you are arousing and kind of coming very briefly to an awake stage of sleep and then going back into sleep

This is an example of a more severe case, and over here in this case, there is actually half of the study that's diagnostic, and over here where you see these lines, this is where we started CPAP, and this patient you can see has more purple lines, which means more apneas, and this is an example of her tracing and you can see here that she has just a large number of apneas, sometimes separated by only 2 to 3 breaths. So she has an apnea, 3 breaths, apnea, arousal, apnea, 3 breaths, arousal, apnea, arousal, 3 breaths, apnea, and so this person had an AHI of I believe if I remember correctly about 168, which was one of the more severe ones that I have seen.

And another interesting thing that I am going to point out is that during the diagnostic part of the study when she was having all these events, she never went below stage 2 sleep, and that's typical because the deeper stage of the sleep particularly in REM the worse the obstructive sleep apnea tends to get, and then once we started CPAP even though she never stopped having even hypopneas, but what happened is she improved significantly and she actually had prolonged periods of REM sleep, this red line here is REM sleep and she had what we call REM rebound, which basically was her brain realizing that it could sleep and breathe at the same time and survive, and so she kind of made up for lost time there, and this is a case of very severe sleep apnea.

This is a case of central sleep apnea and what you see here again is the periods of apnea without any effort, and this is a person who has Cheyne-Stokes for respirations, which we see in heart failure patients and sometimes in stroke patients, but I am not going to get into central sleep apnea very much.

And this finally is another recent definition in sleep apnea called Complex Sleep Apnea, and this talks about a group of patients that have primarily obstructive disease on the diagnostic part of their study so if you look at this part of the study, this person had mainly purple lines, which are obstructive apneas, but then when you start them on CPAP interestingly these light

blue lines show up, which are central apneas, and we are not 100% sure why some people have this and others don't, it's thought to be related to sort of a hypersensitivity to the CPAP and that these patients are very sensitive and start developing some sleep disruption because of the discomfort, the CPAP and then this causes some ventilatory instability. But just to point out that this does happen in some patients and is actually fairly difficult at times to treat and oftentimes if you just give them a little time on some low pressure CPAP to allow them to adjust and then tends to get better and actually disappear.

Prevalence and Clinical Implications

So how big a problem is sleep apnea? Well, if you consider the pure definition of sleep apnea being an AHI greater than 5, then about 24% of men and 9% of women, although these are fairly old numbers. So this may represent an under-estimation of the true prevalence. The sleep apnea syndrome meaning with symptoms or the more severe cases is thought to be about 4% of men and 2% of females.

However, when you look at the high-risk populations, which really is what we all see in our clinics then the prevalence is much, much higher. And I am just going to point out some of the more common ones: coronary artery disease about 30% of patients, all hypertensive's about again 30-35%, but if you look at drug-resistance hypertension, your patients that require 3 and 4 drugs for control, then 80% of those patients have sleep apnea. Congestive heart failure, fairly high incidence also, 40%- although, in some of these there is a combination of both obstructive sleep apnea and central sleep apnea. End-stage renal disease, 50%, AFib 50%, CVA 50%. And this you know kind of since the topic here is diabetes, this actually should say obese Type 2 diabetics, 86% of your obese diabetics have been found to have sleep apnea, and this is pretty incredible when you think that these are the folks that you see everyday.

And so we've developed screening tests for some other conditions that really don't even have the same prevalence, and here you know you have sleep apnea in a quarter of your hypertensive's, a-third of your coronary disease patients, half or your stroke patients and four-fifth of your diabetics. So maybe we ought to be doing a little screening too for sleep apnea.

I am going to spend just a little bit more time in the prevalence in obese diabetics just because this was sort of a landmark study where this came out in Diabetes Care in June 2009, and they basically just looked at all comers into a diabetic clinic and they looked at 306 patients and tested them all and then also looked at symptoms etcetera, but the testing actually was just in the whole population, and they were surprised to find that 86% had sleep apnea by the definition of an AHI greater than 5. But what was also surprising was that a great majority actually had moderate disease or worse. Only about 30% had mild disease and over 50% had moderate to severe disease. And of course the larger waist circumference and the higher the BMI, the more severe the sleep apnea tended to be.

So, this kind of brings us to, so what is the relationship between sleep apnea and diabetes, sleep apnea and insulin resistance? And this kind of summarizes what's thought to mediate this pathophysiology and one is the intermittent hypoxia that as I mentioned, it's not just the hypoxia because like I mentioned before, some people don't seem to have as much of the oxygen desaturation, but they have sleep fragmentation, and the sleep fragmentation, sleep disruption itself seems to also contribute to these consequences.

So it leads to sympathetic activation, neuroendocrine changes and increased inflammatory processes which then affect insulin sensitivity, glucose utilization, etcetera, and the common result of these pathways is an increase in insulin resistance.

Interestingly, the thought is then, will treating sleep apnea improves glucose control, and there is some evidence that suggests that that it does improve glucose control, although a recent study looking at insulin sensitivity did not show significant change with the institution of CPAP. So I think we are just in the early days of research in this area. So when you look at sleep apnea and the concept of metabolic syndrome it almost seems like that ought to be a part of the disease group that is included under metabolic syndrome because they all are interrelated.

So, we can all kind of in our practices probably easily think of sleep apnea in the first patient here, your typical obese patients, although not all obese patients have sleep apnea. Now, the second gentleman over here is very fit obviously, but is a pretty good candidate for sleep apnea as well. If for nothing else the fact that his neck size is probably 22 inches or so and anything above 17 inches seems to increase the incidence of sleep apnea.

And now this young man over here we may not think of as being at risk for sleep apnea. But in fact about 20 to 30% of patients with sleep apnea are not obese, and there are some other anatomic features that predispose for sleep apnea, and one of them is retrognathia or micrognathia, so recessed or very small chin , the things like nasal obstruction, enlarged tonsils of course and those kinds of things. So don't if you have patient who come in and complain of being sleepy and having sleep disruption and some other symptoms that might make you think of sleep apnea, don't dismiss them because they are not obese.

So some of the symptoms of sleep apnea, probably the most common one is excessive daytime sleepiness, fatigue, and we all know that sometimes patients have difficulty distinguishing these two. Fragmented Sleep and or what we call Sleep Maintenance Insomnia is actually a very important thing because oftentimes people will come in and complain of, 'Oh! I can't sleep. I wake up all the time and our inclination is to say, okay, well I will give you a sleeping pill and actually that can make the sleep apnea worse while if there are awakenings caused by apnea then we may not be improving their situation. We may actually be worsening the situation.

Morning headaches, irritability, mood disorders, particularly depression, difficulty with concentration, memory problems and interestingly nocturia. So in your elderly men who we kind of assume that nocturia equals prostate problems, that's not always the case and I always like to tell a story of my elderly gentleman who had sleep apnea and also had in his multiple medical problems, BPH, and we treated his sleep apnea and he came back and was very pleased with the treatment because he felt better but also added, Doc, it's a miracle! My prostate problems, they disappeared. And so I was very happy for him but the reality of it was that his nocturia was probably at least in part related to his sleep apnea.

Some morbidity from sleep apnea, first of all neuro-cognitive as I mentioned the impaired vigilance that is the sleepiness, but not just sleepiness you actually have detectable psychomotor impairment. Impairments in attention and concentration, impairments in memory and one of the main things that seems to be affected is executive function. So the higher level cognitive function, the problem solving, the prioritization, those kinds of cognitive functions seems to be affected preferentially. And these often lead to increased risk of accidents. So

obviously motor vehicle accidents, but not just that but also work accidents, issues with psychosocial stress, with the mood disorders, marital discord and impaired job performance.

And then the cardiovascular problems that we've probably all heard more about the relationship with hypertension, increased risk of myocardial infarction, CHF, cardiac arrhythmias, stroke, and contribution also possibly, especially in patients who also have underlying lung disease to pulmonary hypertension.

So, of course, it's all a spectrum and so the more severe, the disease, the more you are going to be worried about these complications. And then from a metabolic standpoint as I mentioned, the insulin resistance and ultimately diabetes and then obesity in itself because of changes in leptin and gherlin that were mentioned in this slide above and it also seem to contribute to weight gain.

Diagnosis and Sleep Tests

I am going to switch gears a little bit and talk about diagnosis of sleep apnea and they have now divided testing devices into 4 levels.

Level 1 is the typical in lab polysomnogram where we monitor EEG, breathing, oxygen saturation, and movements, cardiac activity etcetera, and it's the gold standard, but of course it's costly. It has limited accessibility, and sometimes some labs have long waits. And the bottom-line is, it's still not a perfect standard because a lot of times when somebody goes to a sleep lab to sleep, they don't sleep as well, and so we call it first night effect and sometimes we don't get a real picture of their normal sleep because of that and that's one of the reasons that the thought was also looking into home testing devices.

The level 2 test is basically the full polysomnogram, the full sleep study like we do in the lab, but done portably at home, and really that's been shown to not be a very feasible option. It's still very expensive, it's technically more difficult because you don't have a technician there to problem shoot, and so it's not used very much.

Level 3 tests, are the main tests that you'll probably be seeing used for home testing, and they have a minimum of four channels and typically will monitor airflow, oxygen saturation, cardiac activity and body position, and in some cases also a respiratory effort and they are quite simple to use and the patients can oftentimes just put them on themselves. They are fairly inexpensive and they are fairly feasible from a technical standpoint.

There is also level 4 device. It's actually with less than three channels, and so these things are things like overnight oximeters that monitor just oxygen saturation and pulse, or some new devices such as the ones shown in this picture that monitor airflow with or without oxygen saturation. And these really are not adequate, and I am going to show in the clinical case with some examples of the potential problems with these, just because they don't give you enough information.

Guidelines and these were put out by the American Academy of Sleep Medicine. The decision of whether to use a portable monitoring device that is a home sleep study should always be used in conjunction with a clinical sleep evaluation and even that also goes towards the decision even of testing for a sleep apnea. So there should be a clinical sleep evaluation that then determines how that patient needs to be worked up. The device should include like I said

a minimum of four channels and whoever interprets it should have access to the actual tracing similar to what I showed you in those images with the definitions where you can actually look at the wave forms and really see what's going on. The AASM says the study must be interpreted by a board-certified sleep medicine specialist, since there are not many of us out there yet this may or may not be feasible in your area that it should be somebody who is at least experienced with these devices and their tracing.

So who should we use portable monitoring on? Mainly, for the diagnosis of sleep apnea in patients who clinically you have a high pretest probability and really that's with any testing and medicine of course. But these are the patients who you see and you often see and you say, you know, I am fairly convinced this person has sleep apnea. I just need to confirm it and see really how bad it is and they shouldn't have any comorbid cardiopulmonary disease or neuromuscular disease.

So, if they have hypertension fine, you are 40-year-old obese, hypertensive patient with diabetes. There are great candidates for home study. If they have congestive heart failure, if they have end-stage renal disease, if they've had a stroke, if they have some sort of muscular dystrophy. No, those are patients that you really need to study in the lab, because they often have complex sleep apnea or combinations of obstructive and central sleep apnea that are not very easily identified on home testing. We also use them in patients who can't travel to the lab, and that really is regardless of what other comorbidities they have. If they can't go to the lab then I think it's better to test them at home than to not test them. And of course then we also use them sometimes to monitor response to treatments. So, in somebody for example who has undergone bariatric surgery and lost a lot of weight, then we can do a home test to document improvement or resolution of their sleep apnea. Contraindications really, it's not a screening tool.

You should not be using these for screening in asymptomatic populations because then you are going to have problems with false-negatives and false-positives, and you shouldn't use them in patients as I said with significant comorbid medical conditions, and again, I kind of reiterated those CHFs, severe COPD, neuromuscular disease, and the other one that I didn't mention is chronic use of narcotic pain medications because in these patients you get enough respiratory suppression where oftentimes what they have is central sleep apnea.

And then also should not be done in patients in whom you worry about other sleep disorders. So if you are worried that they might have central sleep apnea. If you are worried that they have acting out of dreams or sleep walking, or narcolepsy or any other sleep disorder then you really get more information from the in lab study.

Some of the limitations of portable monitoring, a high false-negative rate, the false-positive rate depends on the likelihood ratio. We have a fairly high data loss and equipment failure rates and I can tell you from personal experience because I did two studies on myself and one of them didn't work, so I had a 50% failure rate. And then you also tend to get an under-estimation of the AHI. Mainly because you are counting events, but you can't tell on those studies when the patient was asleep and when they were awake, so you are actually averaging the number of events into the total recording time, which may be if the patient was in bed for ten hours and was reading three of those hours you are going to dilute that number quite a bit. And then many devices as I mentioned don't do a very good job of distinguishing between obstructive and central sleep apneas so you may end up with problems with accuracy of diagnosis.

Treatment Options

So this kind of gives you a little bit of a decision tree on where to go with your individual patients and I'll let you kind of look through, but if you have a high pretest probability and no significant comorbid medical disorders, no comorbid sleep disorders, then you go to portable monitoring.

If it's positive, you treat, if it's negative because your pretest probability was still high, then you worry that it was a false-negative, and so you go forth with a lab study. If you have any of these other conditions that weren't in lab study, you move onto that. If you have somebody who has symptoms of a sleep disorder, but you feel like clinically there the suspicion for sleep apnea is low, then you really should go directly to an in lab study. This is a multiple sleep latency test that looks for other sleep disorders like narcolepsy and idiopathic hypersomnia, other causes of excessive daytime sleepiness.

So, then if you do a home study and it's positive, what do you do with that patient well? One thing is of course you can then decide, okay, he has sleep apnea, I am going to send him to the lab and get a CPAP titration, and that bypasses all the issues of educating the patient on CPAP, figuring out what mask they need, what pressure they need, etcetera, but the more cost-effective route really is using a home titration with an auto-adjusting CPAP device. If somebody has mild disease, then you can also consider just discussing alternative treatments, use of oral appliances which work pretty well in mild sleep apnea, ENT surgery, if somebody has clearly abnormal anatomic features that contribute to their disease or sometimes just conservative treatment like weight loss and avoiding the supine position or treating their nasal congestion and their reflux.

If you decide for a home CPAP titration, then there are few steps and the first is Mask Fitting, and this is where you would need to have somebody, a respiratory therapist or a durable medical equipment company with good respiratory therapist who will do this for the patient, and really assess the mask fit adequately because that's the key to success, and then instructing the patient on CPAP use and really telling them what to expect and how to adjust because it's far from easy and people need to know that they are not expected to slap this on their face and sleep like a baby the first night and feel like a million bucks the next day. So, you really need to kind of give them a road-map of what to expect and how we can problem-shoot that the issues that may come up and offer them help and somebody to kind of turn to when they hit the wall so to speak.

If you do do a home titration then you have a couple of options, the auto-adjusting CPAP devices, start out at a low whatever pressure is set as the lowest pressure, and then they adjust upward and downward depending on whether the machine detects airway obstruction or not, and so they kind of function somewhat independently and you can just put someone on an auto-adjusting machine and let the machine do its thing, and a lot of people are fine with that. Some people need further adjustment in the pressure range especially people with severe disease that need higher pressures, and so sometimes you do have to adjust that pressure range or some people use the auto-adjusting CPAP as a means to extrapolate an optimal pressure by using what we call the 90 or 95th percentile pressure.

But then ultimately you need to look at the patient's clinical improvements and use the compliance data that you now get from all these machines and then adjust your therapy depending on how things are going. If the data that you are getting from the devices is

equivocal or the patient is not feeling better or they are having a lot of problems then you always have the option of going back to the sleep lab and doing an attended titration.

So post-clinical follow-up is important because oftentimes you need to change masks, adjust pressures and then also nowadays with Medicare you need to document clinical improvement and adequate compliance in order for the DME companies to get payment and that's a pretty big motivator for them.

Since with the Indian Health Service you guys probably don't deal as much with Medicare. I am going to adjust very briefly and because of time constraints, these are the requirements for initial coverage by Medicare. They now approve home diagnosis and in lab diagnosis, so you can diagnose somebody with whatever device you feel is appropriate and they'll initially cover the first 12 weeks.

By the end of those 12 weeks that patient needs to have a visit with the practitioner again and this demonstrates that they are compliant and that they are getting clinical benefit and the compliance is defined by at least four hours of use per night on 70% of nights for 30 consecutive days during that treatment period.

So it's not always easy to do considering that again patients have a pretty significant period of adjustment, and so you really need to work with them during that initial 12 weeks. "If they failed CPAP in those first 12 weeks" usually Medicare will still cover the CPAP that they required a face-to-face reevaluation, and of course that really is what we need to do clinically because we need to figure out why are they having problems, is it the pressure too high, is it the mask that's a problem, do they need a device like BiPAP which uses bi-level pressure and oftentimes is more comfortable in patients who have difficulty exhaling against the pressure or are there other things like nasal congestion or claustrophobia. So you really need to kind of do a little medical investigation and then discuss the options for treatment either continue with CPAP with adjustments or discussion of alternative treatments if CPAP really is out.

The keys for success with home testing and home titration with CPAP is first of all patient selection. So, you need to have that patient that is again going to be a high probability that you think just have sleep apnea, that doesn't have other sleep issues.

You need to use adequate equipment, review the raw data, educate the patient, educate, educate, educate, and I know with diabetes you are all very familiar with that, because that's a huge part of treating diabetes as well, and then having close clinical follow-up and good support from either respiratory therapist or a durable medical equipment company who will support you in helping that patient out.

Case Studies and Summary

Now, if I may I'll take just five minutes to go over some cases that kind of illustrates some of the issues that we struggle with some of this home testing and some of the simpler devices that we use, and this is one that shows problems with overnight oximetry and we all use overnight oximetry, a fair amount, I hope mainly as a screening.

Case Study 1

But this has been example of a 50-year-old woman who complained of fragmented sleep, excessive day-time sleepiness, snoring, and witnessed apneas and paroxysmal nocturnal dyspnea. She had hypertension, had had a stroke, had COPD and had a chronic pain as well,

and was on some narcotic pain medications. Her family history is positive for sleep apnea in her father and two siblings. She had a BMI of 31 and neck circumference of 15 inches which is not significantly enlarged and had a Mallampati score of 3 to 4, which means that when she open her mouth and you looked in her throat, you could only see the very top of her soft palate and her hard palate and she had slightly enlarged tongue.

So, her primary care doctor did an overnight oximetry, and if you look here, you may not be able to see it but the medical equipment company who did the test start this here which meant that she really shows that she only spent about four minutes below 88% which didn't qualify her for oxygen, which was the main thing that at the time I think they were looking for, but nobody looked at this -- which is that she had what's called the Desaturation Index of 36, and these devices actually measure 4% drops in oxygen saturation of ten seconds or more.

So that's felt to sort of reflect possible apneas or hypopneas because it's a sudden drop followed by a return to a more normal level. And so when I saw that I thought this isn't normal obviously and she had symptoms, and so this is what the tracing looks like and what you see in somebody with sleep apnea is this what we call a saw tooth pattern where you have these dips and come back up, dip and come back up.

And so she had some of this saw tooth pattern, and sure enough when we did her sleep study, she had an AHI of 21.8, which is moderate sleep apnea and that was without REM sleep. So, I suspect that she probably had even slightly more severe disease and she was treated with CPAP and her symptoms got significantly better.

Case Study 2
This is a lady in whom one of the newer devices that just measure airflow was used and she was 31 and was referred for shortness of breath, hypoxia, abnormal chest x-ray, and her review of systems included morning headaches, sleepiness, and sleep maintenance insomnia. She has been told that she had apneas and she snored, she had orthopnea and PND, she had diabetes, hypertension, and she was morbidly obese with a BMI of 75. You can see the neck circumference and the Mallampati IV, it means that you just see the hard palate, so she has a very crowded oropharynx.

And this is her RU Sleeping Test, which is Respironics airflow monitoring test and it gives you a calculated AHI which in here was 2.4. However, they also did an overnight oximetry on her and that oxygen Desaturation Index on the oximetry was 41, and this lady's lowest saturation was 43% and she pretty much spent her entire night with oxygen saturations below 90,

And this is her tracing again demonstrating these periods of sawtooth pattern and just grossly, grossly abnormal. And so, we went ahead and did a sleep study, and of course her AHI was 98 on the formal sleep study, and why did the home study not pick it up?

Well, it's only measured flow in the nose, may be she was a mouth breather, may be part of the time it was off her nose, so a lot of possible confoundants, that the issue is that this is somebody who had very high pretest probability for sleep apnea, and therefore if you get a negative home study, the thought needs to be there that wow, this is probably a false-negative and let me do something else.

Case Study 3
This shows issues with use of auto-adjusting machines. There are not full proof and not a 100%, and this is a gentleman who had sever sleep apnea, and in the lab, he actually had an attended titration that his optimal CPAP was 13, but he never slept on his back, which always makes me worried that when they get home and they sleep on their back they are going to need a little bit more pressure because their airway is going to be more prone to collapse in that supine position.

So I put him on an auto-adjusting device with a pressure range of 13, which I knew was pretty good on his side allowing the machine to go up to 20 if he rolled over on his back and needed higher pressure. And so, then I did an overnight oximetry to see how he was doing. He was using his machine seven hours a night, was feeling better, but this is his overnight oximetry, and it still shows periods of pretty significant oxygen desaturations. And interestingly, the machine, the compliance download to the statistics from the machine and it shows that he still having about 15 events per hour, but yet the machine was not titrating up all the way to 20, and over here I had actually already increased his minimum pressure to 16. And so for some reason the machine was not responding adequately to his event, and so -- in him we ended up putting him on a set pressure that was higher and that was able to control his residual events.

And finally, the importance of proper patient selection going back to those patients in home you don't want to do a home study because they've either CHF or neuromuscular disease or are on narcotic pain medications because of the issues with coexisting central sleep apnea.

Case Study 4
And this was a 50-year-old lady who came to me referred already with a diagnosis of sleep apnea by a home study and she was referred for CPAP failure. In other words she had gone, she was prescribed CPAP and diagnosed by her pain specialist to whom she had complained of daytime sleepiness and witnessed apneas. Her medical history was significant for chronic pain and she was on multiple narcotic pain medications.

Her BMI was 20, her airway was wide-open, not at a high risk from that standpoint, and her home sleep study showed an AHI of 27 with desaturations to 84%. But then in very small print it said possible Cheyne-Stokes pattern, but nobody saw the small print and so she was given a diagnosis of sleep apnea and she was started on auto-adjusting CPAP, but she didn't do well, she had persistent symptoms. Her machine kept detecting residual events, even though it was titrating the pressure way, way up, and so she wasn't doing well. And so when I saw her, I obviously was concerned about the possibility of central sleep apnea with her narcotic pain medications, and so we did an in lab study and in fact she had no obstructive sleep apnea, but she had moderate central sleep apnea.

And in these cases rather than CPAP you often need to end up using what we use now is adaptive servo ventilation where you have a more adjustable machine that actually gives you a backup rate, so if the patient doesn't breathe spontaneously then it's actually non-invasive form of ventilation where the machine breathes for them. And so we switched her to that and she did very, very well and felt quite a bit better.

Summary

So my suggestions for everyone in the primary care setting is to, to take a few minutes and screen your patients with the basic sleep questionnaire, use home testing if you feel comfortable with it, but use it in the proper patients with careful clinical correlation and then

establish a relationship with a sleep practice so that you can then refer the more complex patients or the ones that don't do well, and you can have some backup so to speak if things don't go well.

So I included here just a quick quiz mainly geared towards sleep apnea, so feel free to use this and yes answers to two or more these questions suggests increased risk of sleep apnea and this would take two minutes for someone to fill out and even though this talk was mainly focused on sleep apnea. I wanted to just make a point that sleep apnea is just a fraction of sleep disorders and that it's important to think about sleep as an important part of health maintenance, because it really plays a roll in that.

And so just ask your patients about sleep, they may not have sleep apnea, may be they have insomnia, may be they don't sleep enough, may be they think they can get by on six hours of sleep, and try to educate them some on the importance of making sleep a priority and making sure that they have good sleep habits and eventually they'll thank you, and that's all I have and I will be glad to answer any questions.