

## Kessler Institute for Rehabilitation 8th Annual Stroke Conference: Part 8 of 8 Sleep Apnea and Stroke

Recorded October 2019. [Listen to it here.](#)

- BRIAN BENOFF: 00:04 [music] You'll see dramatic drops in the sleep in oxygen level. The moment we get them at their right pressure, the sleep is consolidated, the oxygen levels remain stable, and the apnea events are gone.
- ANNOUNCER: 00:17 Welcome to the eighth annual stroke conference Breaking Barriers: There's More to Getting Home than Walking. In this lecture podcast, Dr. Brain Benoff of Holy Name Medical Center presented sleep apnea and stroke. For more information about Dr. Benoff, read his bio in the program notes. This conference was sponsored by Kessler Institute for Rehabilitation and was a one-day event that provided participants with an understanding of the multidisciplinary approach to rehabilitation that enables stroke survivors and their families and caregivers to rebuild their lives. Discussion will focus on communication, motivation, spatial neglect, sleep issues, bowel and bladder management, and community integration. [music]
- BENOFF: 01:08 So good afternoon everyone. My name is Brain Benoff. Our practice is pulmonary critical care and sleep disorders. And so we wear different hats in addition to this one, depending on the site of practice. So I'm going to wear the sleep medicine hat for today. Today's topic is sleep apnea and stroke. So sleep, it's important that we all speak the same language. Before we talk about sleep, I just want to give a general overview to the structure of this talk. We need to speak the same language. So first, we're going to define sleep as the medical community defines sleep. Thereafter, we will then-- once we know what normal sleep is, we can talk about pathological or pathology within obstructive sleep apnea. We can then talk about treatments for obstructive sleep apnea. And finally, we can finish off with how one deals with the post-stroke patient who has obstructive sleep apnea. So in terms of sleep, this is how we find out how the person's sleep is. Generally, the patients don't look this happy when we do this to them [laughter]. The standard sleep test, the standard montage, it's called a polysomnogram. We've been doing them for decades at this point already. The polysomnogram consists of brainwave activity measurement, so we know objectively when the patient is awake and asleep. So you see the electrodes over the scalp. We measure eye movement. So you'll see one on the lateral aspects of each eye. Those are to help us gauge whether or not the patient is in rapid eye movement sleep. We measure muscle activity, muscle tone, and so there is a chin EMG, which is very easy to see. And finally, we measure chest and abdominal motion as well during sleep. This is important and vital for diagnosing obstructive sleep apnea.
- BENOFF: 03:05 Now, most people I said are not that happy to have this done to them. This is not too far off from what reality can feel like for the person who's undergoing a polysomnogram. We don't expect the person to get their normal night's sleep. We do hope, though, that the person will drift off. We do hope that we get a snapshot of their sleep. The literature has been very clear. The original sleep studies were done on sequential nights, Monday through Friday, and with each sequential night the patient got more and more accustomed to wearing this stuff, and they got more sleep each night. What the study showed was, was that even though they slept more on night five than they did on night one, the diagnosis never changed. So if we saw two hours

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of sleep apnea on night one, we would see eight hours of sleep apnea on night five, but the diagnosis was sleep apnea. And so that's important. So we tell the patient ahead of time, you're not going to get your normal night's sleep. We just need a snapshot. This is how we define sleep. Sleep is not, well, I closed my eyes and I was unconscious and then, whatever happens, happens. So there are classic brainwave activity findings that help us define sleep. We call them stages. So there's stage wake. Stage wake, stage one and two are light or superficial sleep. They used to be-- stage three and four delta wave or slow-wave sleep used to be independent, now they've been grouped together. That's a very, very deep restorative sleep. And then finally, there is rapid eye movement sleep, which is classically when people have dream activity. And so we need all of these to define the stage of wakefulness or sleep. So the EOG, which is the eye movements, they'll be sort of rolling as the person falls asleep. They stop when the person's in deep sleep. And finally, when they're in REM, they're this rapid eye movements back and forth, very dramatic.

BENOFF: 05:08

The EMG, which is muscle tone, which is also important to understand sleep apnea, is high. There's muscle tone while the person is awake. And then as the person falls progressively into a deeper stage of sleep, the muscle tone drops until finally, it's at its lowest during dream sleep. Sleep is not one homogeneous entity. Sleep occurs in cycles. The cycles are generally established in young childhood, and they persist throughout adulthood into old age. Sleep cycles are generally 90 minutes long, and they change as the night progresses. Early in the evening, one will see a lot of slow-wave or a lot of delta, a lot of deep sleep, stages three and four. And then as the night progresses, one will get less and less, to the point where one may not see any at all. But one will see progressively increased amounts of REM sleep or our dream sleep, which is why when we wake up in the morning, we frequently will remember a dream that we're in the middle of because statistically speaking, you're having much more dream sleep at the end of the night than you're having at the beginning of the night or middle of the night. This is how we would like everyone to feel when they wake up in the morning [laughter]. I have never met this person, but that's the goal. So that's normal sleep, so now that we know what normal sleep is and how we define normal sleep and how we conceptualize it, now we can step back, and we can say, "Okay, so what is sleep apnea?" Apnea means not breathing. Hypopnea means breathing just a little. There are strict definitions to score an apnea event or a hypopnea event, and when we combine these together, we will generate something known as the apnea-hypopnea index. That's the amount of time, the average amount of time of partial or complete closures of the airway while the person sleeps.

BENOFF: 07:09

This is where all the activity occurs with respect to obstructive sleep apnea. Sleep apnea is primarily a disorder of excessive muscle relaxation that will occur in the back of the nose and the back of the throat. Most pronounced soft palate area, tonsils, throat, and tongue, and you'll see why here. When the person is awake, but laying down on their back-- if I told this person, "Please lay down, close your eyes, and just breathe," the airway remains patent. This area particularly, that's the area of particular concern. The reason that that airway remains open is because there is normal, wakeful resting muscle tone. If I lift up someone's arm when they're awake, even if they're not actively resisting me, there's tone to the musculature. This applies

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to the upper airway, as well. When people go to sleep, as you saw from that EMG tracing before, resting muscle tone drops. In patients with obstructive sleep apnea, it drops to the point where there is partial or complete closure of the airway, hypopnea or apnea. That is the pathology of sleep apnea. Now, obviously, gravity's going to play a very significant component because I'm just going to flip back and forth. The reason that this is falling backwards is because of gravity. That's it. Person's lying on their back. So if you work with gravity-- so if you take the person from the prone position and either put them on their belly-- kids with sleep apnea are very commonly seen-- they're belly sleepers. Adults tend to sleep more on their sides. But either way, they're trying to take advantage of gravity with respect to their upper airway patency.

BENOFF: 09:04

If you speak with anyone who shares a bed with someone with bad sleep apnea, what you hear is that he will roll over onto his back, he will snore to the point where he wakes me up, I can't take it anymore, and I roll him over, and then the snoring stops or gets significantly better. The reason it gets better is because gravity is now working with the patient instead of against the patient. In terms of diagrams, essentially what we have-- we'll start at 12 o'clock and work clockwise around, so the person falls asleep, muscle tone drops, airway begins to narrow and collapse, there's critical closure where the person has to choose between breathing and sleeping. They try to breathe even more, but again, this is a plumbing issue. It's the upper airway that's closed off. No matter how hard they move their chest and abdomen, there's not going to be any airflow because the upper airway is obstructed. At that point, the person says, "Look, I can't breathe," the brain then wakes up. Again, this is an electrical arousal. It's not an awakening from sleep. There's generally not a consciousness that is attained. It's too brief. But it is enough just to restore the resting muscle tone to the wakeful state. There's a large snort and gas after the muscles return, and then the cycle repeats itself again. That's classic obstructive sleep apnea. What we see as the sleep doctors, is we'll see-- this is moving along with time, so time is going away from me. The EEG will start off with the person asleep, then the airflow will stop, the apnea event occurs, the chest and the abdomen continue to try to breathe. In fact, you will even see the esophagus, the pressure there will get even more and more negative, suggesting that there are exaggerated breathing movements. Some people will actually get acid reflux during sleep apnea because they will suck the acid up from their belly all the way up into their chest. Oxygen level drops, and then the person wakes up electrically, where it says arousal. And then airflow is restored.

BENOFF: 11:06

The reason I brought this slide is very important because we're talking about post-stroke patients. There are two different kind of apneas that occur during sleep. There is obstructive, which is what this talk is about. And then there are centrals, which is what this talk is not about. Central apneas are generally seen either in the setting of severe cardiomyopathy, congestive heart failure, otherwise known as Cheyne-Stokes respiration. And it can also be seen in the setting of acute stroke. I'm going to talk about treating obstructive sleep apnea where the literature says that people will benefit from it. To be very clear, with central sleep apnea, the literature says is that I will do the patient a disservice. There's actually an increased mortality in treating central sleep apnea in the setting of heart failure. No one has studied it in respect to

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stroke. But again, we're talking only about obstructive sleep apnea. This is the last time you'll see one of these. Not to worry, but this is what we see on our screen. This is about two minutes worth of data. And essentially, what we're looking at is sleep electrically, the chin EMG is down so the muscles are nice and relaxed, and the airflow goes down flat, no airflow. The chest and the abdomen are still moving, and now the oxygen level begins to go 98, 97, 96. It's bottoming out. And then all of a sudden, the chin EMG jumps up. That is the patient snorting and gasping for air. Patient wakes up, and all of a sudden, the [airstow?] is restored, and then the cycle begins anew.

BENOFF: 12:46

So how do we-- we have to categorize it. That's what doctors do. So obstructive sleep apnea is categorized as either mild, moderate, or severe, depending on how many apnea events occur per hour of sleep. You can stop breathing, rather, 4 times an hour, and we don't care. That's perfectly normal. 5 to 15 is considered mild. 5 to 15 events per hour of sleep. 15 to 30 is moderate. Greater than 30 is considered severe. It's very common. Sleep apnea, depending on who you read, is usually about 3 to 4 percent of the adult male population. It usually runs about 2% of the premenopausal female adult population. Women tend to catch up postmenopause. Female hormones make you breathe more. If you do blood gases on someone who is pregnant in their first trimester of pregnancy, they will have a respiratory alkalosis. Their carbon dioxide levels will be low, and their pH will actually be high, and that's because the baby's only microscopic. So it's not a mechanical issue, but they have more circulating female hormones, and they breathe more. That's normal. But postmenopausally, women and men probably have about the same incidents. So what do they experience? If someone said, "I think I have sleep apnea. What would I expect?" And so there are lots of different things. Number one, anyone will tell you if you spend the night with someone with sleep apnea, the snoring is a big part of your night. Patient with sleep apnea, again, what you're looking at is regular, frequent sleep fragmentation and because of that-- even though they clock the right amount of time in the bed at night asleep, they're not getting the full restorative properties from it. And so they will be functionally sleep deprived. The observer will see gasping or choking during sleep. My kid genuinely enjoyed that picture [laughter].

BENOFF: 14:45

Sleep apnea patients are mouth breathers, and so when they wake up, mouth is very, very-- they are parched. In fact, many of them will wake up in the middle-- they will sleep with a glass of water at the bedside, and they will drink during the night, which also makes them get up to urinate and makes matters even worse. And they will genuinely be foggy. They're slower. An untreated sleep apnea patient is not as smart as he or she would be were he treated. It's just the reality. It's been demonstrated by studies from the University of Pennsylvania. They took people with severe sleep apnea. They give them neurological batteries. And then they repeated them eight weeks into therapy, and they did much better, and it was not a learning effect, they were genuinely faster mentally. Let's see what else we have here. I don't know what this machine does, but it picked up a lot of stuff that I wanted to point out to everyone here. So in terms of who's likely to have sleep apnea, odds are if your mom and dad had sleep apnea, you're going to have it too. The anatomy that you have is probably very similar to your parents. So it does run in families. And the anatomy being large neck, large tonsils, long, soft palate, elongated uvula, those things are all

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transmitted genetically in terms of how you look internally. Obesity is clearly associated with obstructive sleep apnea. It's a weight-sensitive disorder. The bigger you get, the worse it gets. The smaller you get, the better it gets. No one knows why. So anatomy is something we talked about. I think it's hilarious that drinking alcohol is upside down [laughter]. So, yes. So alcohol is a muscle relaxant. If you talk with any-- people will say, "Well, he snores a little bit, but if he has a nightcap, then he really, really snores." That's because alcohol is a muscle relaxant. Anything with muscle relaxant properties benzodiazepines, Valium, Xanax, all of these things will decrease your muscle tone even more and will worsen when superimposed on preexisting obstructive sleep apnea.

BENOFF: 16:53

And finally said neck size and gender we touched upon. Again, I don't want to read the slide to you. But again, the importance here is actually the common signs and symptoms. What you will see is that a lot of this stuff is not necessarily you would say, "Well, this guy is not sleeping well." So when people complain of GERD, when they complain of impotence, when they complain of memory loss, irritability, these are genuinely signs of obstructive sleep apnea. And so yes, it affects everyone. So why do we care? So let's say, "Okay, someone has obstructive sleep apnea. Why do I need to treat this?" And so there are different categories of reasons to want to treat obstructive sleep apnea. The first is people with obstructive sleep apnea genuinely tend to underperform. They're sleepy during the day. They're cognitively slower during the day. They have a time maintaining wakefulness during the day. Quality of life is reduced in someone with obstructive sleep apnea. Also, quality of life for those around the person with obstructive sleep apnea is also reduced as well. So quality of life is number one, but that's not the only reason, and that's not why we're here. We're here because of this slide. Obstructive sleep apnea is associated with a whole host of medical disorders. Some of which are reversible. Some of which are not reversible. And we're going focus here on stroke, high blood pressure. Now, those are the big ones for everyone here, but they're very wide and very, very varied. If you're a numbers person, enjoy.

BENOFF: 18:37

How does it occur? What's the connection between sleep apnea and stroke? How do we make that leap? And so that's what this slide is all about. When the person has their apnea event, oxygen levels drop. So there are free radical formation. There is sympathetic activation. If you look at serum catecholamine levels in individuals with obstructive sleep apnea, they remain elevated. Their sleep is not restful. They're in a constant state of wakefulness and vigilance, and that takes its toll over time on the rest of the body with respect to the cardiovascular system. Another reason to treat sleep apnea, it is a public health issue. It's not that the woman is sleepy, she's sleepy behind the wheel. And so this is dangerous not only to her but to everyone else around her who's on the road. There is a fifteenfold increase of traffic accidents with untreated severe obstructive sleep apnea. It begins to rival that of driving drunk in the severe state. This pretty much sums up everything and the important things, like I said, car accidents, quality of life, and medical disorders. There is a subset of patients known as Pickwickian patients, otherwise known as obesity hypoventilation patients. These patients are generally morbidly obese. This should probably read 40, not 30. You're looking at BMIs in the morbidly obese range. Morbidly obese is generally

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defined as 40 or above. The difference between a Pickwickian patient and obstructive sleep apnea is that these people have elevated levels of carbon dioxide even while they're awake. Things have gotten so bad that their midbrain has reset and now tolerates a higher level of carbon dioxide before the drive to breathe actually kicks in.

BENOFF: 20:30

We care about this because a large-- a group of these patients have obstructive sleep apnea, and they absolutely need to be addressed, and they're probably the most severe of the group. Why do they call it Pickwickian? No one ever asks, but the reason is it's actually The Pickwick Papers written by Dickens. No one under here under 40 probably has read this. But bottom line is that in The Pickwick Papers, there was one student his name was Joe, the fat boy. Joe, the fat boy, would fall asleep during Pickwick's lectures, and he would need to be beaten with a stick in order to maintain consciousness to get the lesson. So, in summary, sleep apnea and brain function, not a good mix. So how do we treat? There are different modalities of treatments of obstructive sleep apnea. The gold standard of therapy is something ending in the letters P-A-P. There is CPAP. There is BiPAP. There is APAP. CPAP is continuous positive airway pressure. BiPAP is bilevel positive airway pressure. APAP is auto-titrating positive airway pressure. They all work essentially with the same premise, which is we're going to take a box, which is a pressure generator, room air, no oxygen involved here, and we're going to take a mask that will form an airtight seal over the patient's airway and will provide room air under backpressure. So when that floppy airway wants to close off, the backpressure will just hold it up. The analogy I give is if someone has a balloon with a really long neck, so if you just put it out on the table, the neck is going to be collapsed. If you put in just a little bit of air pressure, not to inflate the balloon, but you will then see the shape of that neck. We're doing that with the people's necks. That's our patient's neck.

BENOFF: 22:23

This is what someone looks like. They generally don't look this happy to be wearing it, and he's this happy because it's actually not put on correctly. His straps are loose. Only to be outdone by this person who wore full makeup for the picture [laughter]. I give her a lot of credit. This is what we see in someone before and after with respect to sleep apnea treatment. This is the oxygen level as the person sleeps. Each one of these lines is an apnea or a hypopnea event. You'll see dramatic drops in the oxygen level. The moment we get them at their right pressure, the sleep is consolidated, the oxygen levels remain stable, and the apnea events are gone. There are other ways to treat obstructive sleep apnea. So, in addition to positive airway pressure, which is the gold standard modality of treatment, there is also something called a mandibular advancement device or an oral appliance. And this is essentially a bite plate that the patient wears and what it does is it projects the base of the jaw and tongue forward while the patient sleeps to pull the airway forward to open up the airway. That is one option. All right, we're getting there. And finally, there are surgical options that are available. There are two surgical options that are presently available. One is the classic uvulopalatopharyngoplasty, otherwise known as the UPPP. The UPPP is a very, very dramatic surgery. The surgeon will essentially take out the tonsils. It will shave off the soft palate and the uvula and, if need be, actually reduce the size of the tongue to refit the tongue to the patient's mouth, the idea of surgically creating a large, full airway that's patent.

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BENOFF: 24:23

There are new treatments as well. One is essentially a hypoglossal muscle nerve stimulator. This serves to actually stimulate the upper air while the patient sleeps at night, again, to keep the upper airway patent. So there are different ways of treating it. With respect to primary prevention-- well, there I will say that most people will believe that primary prevention-- if you have someone with sleep apnea and if you treat them, they are less likely to have a stroke, they are less likely to have cardiovascular disease. And so that is a good idea. The question is, how do we approach secondary prevention, which is what this is all about? Secondary prevention really says the person has had their stroke, and now they're in the acute care setting, they're in the rehab setting, and so you know what? In retrospect, this person probably has sleep apnea that's untreated. Is there any benefit to treating the sleep apnea? And there the data is much more sparse and much less convincing and robust. There were two groups of studies that were looked at. One where it was started very close to the neurologic event in the acute care setting. Person was brought in to the emergency department, facial droop, hemiparesis, and they said, "Okay, let's treat this person with CPAP right away in addition to all the forms of supportive care." Again, the numbers were just too small. There were improvements in a number of things. Functionality was improved. Cognitive function really was not improved with respect to benefiting, and that is at the site at the moments of the acute event in the acute care center.

BENOFF: 26:26

That's not what you're seeing here. Here people have been treated for their acute event. They've been in-house for several days. Remember, if you want the brain-- you wanted to salvage as much brain as possible, you want to get as much oxygen to the brain as possible. You want to provide a good blood pressure with respect to that disautomated-- autoregulated area of stroke. And so for that reason, by the time they get here, they're out of that window. So the utility here, I believe, is primarily for patients, number one, who can tolerate the mask. Again, if you can't tolerate the mask, there's really not much to talk about. The other modalities are not available outside of the community. With respect to what we're trying to accomplish, if you have someone who's sleep is fragmented, if you have someone who is hypersomnolent, they're not going to be able to participate in what you want them to participate in, in addition to recovering from the sequelae of the stroke. Then at that point, they're also going to have to try to overcome the excessive sleepiness associated with their untreated sleep apnea. So this was the sleep apnea device, woman in makeup, and I think this is where we left off. And, yeah. And, yeah, no one's this happy [laughter]. It doesn't work that way. This is the oral appliance. And so this is essentially what it does, it pulls the lower airway forward, it pulls the jaw forward. It does require that you have intact teeth because really you're pulling the lower jaw against the upper jaw. In terms of surgery, those are always an option. Again, patients generally don't glow. We usually light the patients from externally [laughter]. I don't know what was happening here.

BENOFF: 28:16

See, this is a lot funny when I haven't already said what the uvulopalatopharyngoplasty is, which is exactly what this is. And so here this is the patient before, and this is after. And so the soft palate, the tonsils, the uvula, they're all excised or trimmed back. And these are real patients. This is what they looked like

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before and after. That having been said, you'd think that they would do very, very well, not necessarily so. There's only about a 50% success rate with respect to surgery. And a success rate being defined as not having apnea when they're done. And so this is not first-line therapy. It's really not. With the exception of certain individuals who have obscenely large tonsils and you'll say, "Wow. It's got to be that." And this was the hypoglossal nerve stimulator. And essentially what you have is you have a sensing lead, so the person tries to breathe, there's a box that generates the charge, and then the lead is tunneled-- so everything is subcutaneous, and this is-- and so it stimulates the hypoglossal muscle and the upper airway is stimulated and patent. So again, primary prevention, yes, we do believe it makes a difference. In terms of post-stroke-- so I skipped over this slide, and I apologize. And so in the setting-- so sleep apnea in the setting of an acute stroke makes life a lot worse and that's the point of this. We're trying to undo a lot of what we're seeing here. If we can help prevent some of the delirium, depressed mood, increase somnolence.

BENOFF: 29:57

We touched upon that one already. And so I said, acutely screening and treating because what we're really talking about is PAP masks. So right now there's not a large enough body of literature to suggest treating everyone with sleep apnea, however-- we did that. We did that. And so in terms of who I would recommend treating in the rehab setting, again, what I would say is if someone has an apnea index greater than 20, they're feeling it during the day. Whether they recognize it or not, they're genuinely feeling it, and so they would benefit. Anyone who has a lesser apnea index but still has excessive daytime sleepiness, absolutely. I would like it done, ideally, as close to the neurologic event as possible in the acute care setting because there you can really try to salvage brain post-stroke. And finally, - not on this slide - is the Pickwickian patients. So if you have someone with true obesity hypoventilation, there's a very good chance that you will improve their cognitive status and their level of awakesness and alertness with the application of positive airway pressure. I thank you for your time. [applause] [music]

ANNOUNCER: 31:14

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