SLEEP APNEA - ITS TRUE DIMENSION

Helen Martin, MD

DR. WILLIAM BAKER: We will start the afternoon program. One of the most pleasant aspects of planning this meeting that Ann and I have encountered is having a son on site here in Chicago. Our son Steve graduated from the University of Massachusetts, came out to the University of Chicago for his internal medicine residency and then spent seven years in private practice in the southern suburbs.

He’s in his third year of pulmonary critical care medicine fellowship at the present time at Northwestern University, just three blocks away. He’s been able to give me excellent advice and to be on site and to planning for the Chicago based speakers at this meeting. He’s going to moderate this afternoon’s session.

All four speakers are mentors of his in the pulmonary medicine department at Northwestern. So I’d like to introduce Dr. Steven Baker who will moderate the afternoon session.

DR. STEVEN BAKER: Thanks, dad. Our first speaker this afternoon is Dr. Helen Martin who will talk to us about sleep apnea. She’s the co-director of the Sleep Lab at Northwestern Memorial Hospital. She did her medical school training at the University of Illinois and her residency at Northwestern where she was chief resident and did her fellowship in pulmonary medicine at Northwestern. Her research interests are in cerebral vascular aspects in complications of sleep apnea. I’d like to introduce Dr. Martin.

DR. MARTIN: The title of my talk is sleep apnea. The title in your handouts is Its True Dimension. I wasn’t sure if that was supposed to reflect how many patients have sleep apnea or how large their airways are. Anyway I’ve divided it into four broad categories. I’d like to talk about the definition and pathophysiology of sleep apnea, touch on the scope of the problem which I think is particularly of interest to this group, how we diagnose sleep apnea and how we treat it.

Definitions

Sleep apnea is somewhat arbitrarily defined as consisting of apneas of greater than ten seconds in duration, at least in adults, more than five apneas per hour, more than 30 apneas per night and you may recognize this number as one that some insurance companies use to define someone as having a significant problem with sleep apnea. These should be repetitive events and at least some of the apneas should occur in non-REM sleep.

This reflects the idea that your sleep is most irregular when you’re in REM sleep. So a few apneas, especially in REM sleep, are not abnormal. Some people will add to the definition that you should be sleepy during the day, have a complaint of hypersomnolence but I prefer to stick to this definition of sleep apnea syndrome.

Pathophysiology

Now, apneas can be divided into central, obstructive and mixed. This is a measurement of air flow. Esophageal pressure is a measurement of what kind of a respiratory effort you’re making. And then this is oxygen saturation.

With central apneas, what you see is that air flow and esophageal pressure stop at the same time. You aren’t making any respiratory effort. It’s kind of like holding your breath while you’re asleep. Whereas in obstructive apneas you have no air flow but your respiratory efforts continue and may even increase as the apnea progresses.

This is because you’re having an obstruction in your pharynx to air flow. Both of these are associated with oxygen desaturation. A mixed apnea is one that starts out looking like a central apnea and ends up looking like an obstructive apnea. In general these are classified as subsets of obstructive apnea.

I’m going to concern myself just with obstructive apnea today. It’s the one that is by far the most common. More than 80 percent of patients we see have obstructive sleep apnea, and it’s the one that’s associated with more significant morbidity and mortality.

If we move on to the pathophysiology of obstructive sleep apnea, how does it occur? We start out with sleep onset. The patient goes to sleep and then for some reason they develop an apnea. Why do you develop an apnea?

Well, your posterior pharynx, unlike your trachea and your larger airways, is critically dependent on those muscles to keep it open. When you’re supine, tissues are kind of flopping together, and when you go to sleep and the muscles relax, you’re predisposed to obstructing your airway.

Now, not everybody has obstructive apnea. So almost always you have a subtle or a major predisposing factor. You may have lots of extra pharyngeal tissue and a very floppy pharynx. It may
be that you have a bad nasal problem, a severe nasal obstruction and you're trying to breathe through your nose and generating a lot of negative pressure in your pharynx and so that will help cause the tissues in the pharynx to collapse in that vacuum.

Whatever happens, breathing is cut off and the result is that the patient's oxygen saturation drops, CO₂ goes up and the PH drops.

Now, eventually the patient will arouse. Exactly what causes the arousal isn't well established but it may be how much of a respiratory effort you're making that awakens you. When you're aroused, all of those muscles in your pharynx are reactivated, you frequently shift position a little bit, your airway opens up and you start to breathe again.

Unfortunately what happens is you return to sleep and the whole cycle starts over and over and over again. Patients with really severe obstructive apnea can obstruct 40, 50, 60, 70 times per hour. And the number of respiratory events you have per hour is called your apnea index or your respiratory disturbance index.

Now, that sequence of events causes some secondary physiologic responses, and you can divide them up into two broad categories. Show you the cardiac and respiratory events that are associated with obstructive apneas.

As a result of the hypoxemia, hypercarbia acidemia, patients can develop arrhythmias. Arrhythmias are well established in patients with obstructive sleep apnea. The most common one is bradycardia during the episode followed by tachycardia when you start breathing again, but there is an increased incidence of ventricular arrhythmias as well.

And it's thought that this might contribute to the nocturnal death that may or may not be associated with obstructive sleep apnea.

During the apneic event you have both systemic vasal constriction and pulmonary vasal constriction. On the most part when the apnea is over, these resolve; however, 40 percent of patients with obstructive sleep apnea will have sustained hypertension during the day. And it's not clear if it's just that the majority of patients who have obstructive apnea are obese middle aged men or whether it's a causal effect.

In the same way, but to a lesser extent, patients can have sustained pulmonary hypertension during the day which is associated with obstructive sleep apnea at night. Then during the acute apneic event, you retain CO₂.

The normal response is that when you start breathing again you hyperventilate, blow your CO₂ back down to normal. There's a small group of patients who aren't able to normalize their CO₂ and actually develop CO₂ retention during the day.

This is an important group to know. These are the so called Pickwickian patients or the obesity hyperventilation patients and it's this group where I'm convinced that we have the strongest evidence that there's significantly increased mortality associated with obstructive apnea.

There are neuropsychiatric and behavioral events that are associated with obstructive sleep apnea. All of those arousals cause sleep fragmentation and it's postulated that they cause cerebral dysfunction, although it's not easy to measure these. What you see is that the patients complain of impaired memory and concentration and a very significant complaint is excessive daytime somnolence.

So they're sleepy during the day when they're being continuously aroused at night. In addition every time the patient arouses, they shift in their sleep a little bit and this accounts for the restless sleep that's associated with obstructive sleep apnea.

Epidermiolgy

How common is obstructive sleep apnea? There's a recent study that came from the University of Wisconsin and was published in the New England Journal of Medicine in 1993. In this they sent questionnaires to all of their employees that work for the government at three different agencies between the ages of 30 and 60. So you're taking a presumably relatively healthy working population. They sent questionnaires to these patients about how sleepy they were and whether they snored and then they selected a randomized group of them for overnight sleep study.

They screened about 4,500 employees and about 650 underwent sleep study, and from this data they came up with the numbers that the prevalence of sleep disordered breathing by which they meant that they stopped breathing more than five times per hour was 24 percent of men and nine percent of women.

So almost a quarter of these people who are working for the government in Madison have more than five apneas per hour. If you added in the caveat that they had to be sleepy during the day, it dropped to four percent of men and two percent of women. This is something of a landmark study.

There have been other studies in the past that have tried to estimate the incidence and prevalence of obstructive sleep apnea but they were limited to specific groups of people, they didn't include women.

This is a working employed population of men and women. They're not particularly elderly, they range in age between 30 and 60 and there are a lot of them out there who have significant sleep apnea. It's a significant health problem, one that we all have to deal with.

Mortality and morbidity

Just how dangerous is obstructive sleep apnea? There have been three main studies that have looked at the mortality associated with obstructive sleep apnea and I wanted to mention the data from the three of them relatively quickly. It's interesting, they
were all published in *Chest* in the last half of 1988, so they all came out within about six months of each other. They're all from well established sleep groups around the country.

The first one came from Henry Ford Hospital. It was a retrospective questionnaire study of their patients with obstructive sleep apnea who had sleep studies done in the late '70s and early '80s. And they sent these questionnaires to 385 men.

They didn't have a control group in this study. What they did is compare sub-groups of their patients to one another and compared with their life table survival analysis. What they came out with, their conclusion was that there was an increased mortality with an apnea index greater than 20, especially if you were less than 50 years of age.

They postulated that above 50 there are many other causes of cardiovascular deaths that tends to obscure the findings associated with obstructive sleep apnea. I think this is an important number to remember and I use it as kind of a warning sign.

The second study came out of the University of Florida. Again it was a retrospective questionnaire study of patients with sleep studies but in this they had not only patients with obstructive sleep apnea, but controls as well. Their control patients were patients who had been sent to the sleep lab for complaints of being sleepy during the day but who did not have sleep apnea on their sleep studies. The results of their study showed that there was no difference in mortality between sleep apnea syndrome patients and control.

They did point out that the patients with sleep apnea syndrome who died were the Pickwickian patients, the daytime CO2 retainers. So there's one study kind of for increased mortality and one against.

And then finally the last study came out of Stanford. About 200 patients, a retrospective questionnaire study of patients with sleep apnea. They had two groups. They had patients who were treated conservatively and conservative treatment in the late '70s and early '80s meant telling the patient to lose weight. They didn't document whether they lost weight, didn't follow them, just told them to lose weight. And they compared those with patients who were treated with tracheostomy. And their conclusions were that there was an increased mortality in patients who were conservatively treated, 14 of 127 patients died. Whereas in their group of patients who underwent tracheostomy, none of them died, none of the 71 patients died.

What I thought was interesting is the United States age adjusted controls for this study, it's hard to understand how these obese, hypertensive, middle aged men would survive to a much greater extent than their US aged matched control groups, simply by virtue of having a tracheostomy placed.

So I think that these are important studies in that they're the best we have, but that there are frequent problems with them.

In all of these studies the patients had a similar severity of problems, but a critical point is that there was no information on the oxygen saturation or desaturation associated with their respiratory events because oximeters were not commonly available in the late '70s and early '80s.

Who has the worst prognosis? When is it more severe? These kinds of questions are difficult to answer. What I can tell you is that saturations below 60 percent are associated with increased incidence of ventricular arrhythmias. So that's another warning point that I use, saturations of 60 percent.

The conclusions that I draw from these studies and some others, are that there is some increased mortality with obstructive sleep apnea. I think that it's especially true for your daytime CO2 retainers. That's a group that needs treatment and there's no question about that, even if the treatment is severe and more than most patients would like to have happen to them. I think that the apnea index of greater than 20 is another warning sign.

If somebody has more than 20 apneas per hour, particularly if they're associated with significant oxygen desaturation, that's another group that's at risk. I think that the relationship between oxygen desaturation and mortality has not been adequately explored. I don't think mortality is as marked as was previously believed. I don't think it's as marked as it's made out to be in the public press, and on "20/20."

Every time there is a television special on obstructive sleep apnea, we're flooded with calls from patients convinced that they're going to die in their sleep. I don't think that's happening. It would be nice to have some prospective studies but we really don't. I don't think that in this day and age it's likely to be done.

**Diagnosis**

How do you diagnose obstructive sleep apnea? Patients come to you with two sets of complaints generally. They have their nocturnal symptoms and their daytime symptoms.

By far the most common complaint I hear in the office are patients coming with a complaint of snoring. It most commonly doesn't disturb them but it disturbs everybody else around them. They also complain of restless sleep. Their nocturnal sleep is frequently disrupted, although somebody's whose apneas are occurring 40 times per hour will frequently tell you that they're awakened one to two times per night, and all of the other arousals are so brief that they don't remember them.

Then there's an interesting association with nocturia. It's possible that this continuous sleep disruption interferes with the ability to concentrate urine and so it's not just because these patients are awake and decide to go to the bathroom. It's because they actually have an increased urine output.

The daytime symptoms, the most common, is excessive daytime somnolence. If they have severe enough apnea and sleep dis-
ruption, they will complain of difficulty concentrating and personality changes. I think that these sexual problems and complaints of headaches are highly overrated and it's not a real common presenting complaint.

The gold standard for the diagnosis of obstructive sleep apnea is an overnight polysomnogram. This consists of a number of measures that you're making. You need some recordings to define sleep stages, so those are EEG leads, eye movements, the chin EMG to define REM sleep.

You need some way of determining sleep apnea, a measure of air flow, a thermistor or CO2 monitor at the mouth and nose. You need some way to determine respiratory effort. The way are esophageal balloons to reflect intrapleural pressure, but we tend to go with less invasive methods, belts around the chest and the abdomen. No sleep study today is complete without a measurement of oxygen saturation and you need an EKG lead as well. In our lab we also want to monitor patients' leg movements which can disturb their sleep and cause daytime sleepiness as well.

The problem is that a night study in our laboratory costs about $1,700. In the Wisconsin study they estimated that 45 percent of that working population of men were habitual snorers and the economics of sending half of the men, aged between 30 and 60, for a $1,700 test adds up. So we need better ways to screen for sleep apnea, and there are a couple of ways of doing this.

The convention sleep study is your gold standard. You can either work on improving your clinical diagnosis of obstructive sleep apnea or you can work on having cheaper tests to actually screen for sleep apnea. I'd like to talk about these clinical scoring systems first.

There have been a number of clinical scoring systems developed for just this reason. People are looking for ways to talk to the patient in the office and determine who is a likely candidate to have sleep apnea so that not everyone needs to be tested.

Our history and physical examination, a good screening test for sleep apnea, was published in the Annals of Internal Medicine and came from the University of Toronto, published in 1991. In this the authors attempted to determine if history, physical and the overall subjective impression of a very experienced clinician could serve as a sensitive screening test for obstructive apnea. They developed one of those very complicated predictive models based on clinical impressions, signs and symptoms.

Their conclusions were that the predictive models were not sensitive or specific enough to allow confident prediction of disease. Of the clinical features that they looked at, the ones that they thought were the most predictive for obstructive sleep apnea were older age, greater body mass index, so the fatter you were the more likely you were to have obstructive sleep apnea, male sex and snoring. All of these were significantly more common in patients with sleep apnea, but it wasn't a good enough way of screening them.

In this next study, they combined screening for sleep apnea using a clinical score with overnight pulse oximetry in the patient's home which I think is kind of a neat idea. This study was published in Chest in 1991 and came out of UCLA. Basically they questioned their patients and then they sent them home and did overnight oximetry in the homes and then brought them back into the lab for overnight sleep studies.

From their clinical data, what they found was that five features were used to arrive at a clinical score; the presence of snoring, the presence of witnessed apneas, the presence of hypersomnia during the day, obesity and hypertension. What they found is that if your patient had four or five of these present, all of these patients had significant apnea, more than 20 apneas per hour. Unfortunately if they had one, two or three of these they may or may not have significant apnea.

So at the lower end it wasn’t very helpful. But if you had someone who came in and complained of all of these, you could justify sending them for a sleep test.

Unfortunately the oximetry didn’t work as well as I would have hoped. About a third of the tracings had to be tossed out just because they were technically inadequate. When we’re looking at cost effective ways of screening for sleep apnea, it costs several hundred dollars to have one of the home health care companies go out to your patient’s home, tape an oximeter probe to their hand and turn on the oximeter and the strip chart recorder and then come back in the morning and collect it.

And if a third of those are going to be technically inadequate, you’re running into the problem of whether it’s worth using this for a screening test.

I tried to put all of these together from these and other articles in a general statement of clinical predictors and what I’ve come up with is that clinical predictors of sleep apnea are generally not sensitive or specific enough for diagnosis. The clinical features which are the most predictive are snoring, obesity, witnessed apnea and hypertension, and perhaps male sex and daytime somnolence as well.

Finally, if we can’t sort the patients out in the office in terms of who should be sent for sleep study and who shouldn’t, should we send all snorers if we have a cheap enough screening test? That’s kind of another way to get around the exorbitant cost of screening patients for obstructive sleep apnea.

A variety of systems have been developed. We’ve talked about one study that used nocturnal oximetry. I do occasionally use oximetry. I’ll oximetry if somebody comes in and tells me they snore but they don’t have any apneas and they’re not tired at all but I’m still a little bit worried that they might have apneas. In that case then sometimes I feel comfortable enough to use a nocturnal oximetry, and if they don’t desaturate then I’ll classify them as simple snorers as opposed to patients with obstructive apnea.
There are a number of home screening devices which go a step farther. They don't do all the measurements that we do in our sleep lab, but they pick and choose among several. It depends on which device you choose. Some of them will look at air flow and respiratory effort and oxygen saturation. Others will monitor eye movements as kind of a way to make sure that you’re getting into REM sleep so that you’re not missing the point where a lot of saturations occur.

The home screening devices have not been officially accepted by the ASDA, the kind of home group of sleep physicians, but they’re the trend of the future. There’s been at least one study where a home screening device was validated and compared nicely to in-lab screening. I think we’re going to see more of this to come. It makes me a little uncomfortable. As a sleep physician I like to see the tracing, I like to see how the patient is sleeping, I like to see the raw data for how much they desaturate, what their respiratory events are.

The other thing that you lose with these home screening devices are you can’t pick up a lot of other things that can cause the patients to be sleepy. You won’t be picking up their leg movements, you won’t be picking up abnormal brain waves, such as an alpha-delta pattern which is something that can make them sleepy during the day and is potentially treatable. So these devices I think will be coming into their own but you have to realize their limitations.

The other idea is that it depends on the home health company which is doing the screening. There’s a company in Chicago that is doing home screening for $800. For $800, it’s half the cost of our overnight study in our laboratory and you don’t get anywhere near as much data. I’m not sure that that’s a cost effective choice. I think there are other companies that do data at a much lesser rate in which case your cost benefit ratio changes.

Finally there are those people who recommend that you do a half night study, that you study someone as a base line for only half the night and then if they need CPAP, you start that in the second half of the night. I think if you get a representative sample of REM sleep in that first half of the study before the patient is treated, that there is some merit to doing studies this way.

We’ve done it in our lab simply because occasionally we have such a long waiting list to get into our laboratory. What you have to realize is that frequently this doesn’t end up saving you any money because you don’t have time to titrate their CPAP.

So you have to bring them back for a second night as well. So this may save us a little money, but it’s certainly not going to cut the number of sleep studies we do, in half.

Treatment

Then finally, I’d like to move on to the treatment of obstructive sleep apnea. I’ve divided it broadly into conservative measures, pharmacologic therapy, surgical therapy and mechanical therapy.

This is the talk I have with all my patients who have obstructive apnea. I advise them to avoid sedatives and alcohol. People who are snorers can attest to the fact that if they have a few drinks shortly before they go to sleep, their snoring is markedly increased. The same thing happens with apnea.

In terms of sedatives, as little as 30 milligrams of dalmane has been shown to increase the number of apneas and so I try to get patients to stay away from sedating drugs and I use things like non-sedating antihistamines if they’re necessary. It’s not shown that they’re better, but I kind of assume that they are.

In terms of weight loss, I’ve summarized two studies. In one study, before jejunial by-pass patients started out weighing 231 kilos. So we’re close to 500 pounds. They went down to 123 kilos, so they lost more than a whole person’s worth of weight. And their apnea index went from 78 to 0.4 and the lowest their oxygen dropped improved significantly.

Most of our patients are not this extreme. So I’ve added the data from another study in which patients went from 117 kilos to 93, so they lost 50 pounds. They’re still not skinny, but they have a substantial weight loss.

You can see that the apnea index, the number of times they stopped breathing per hour, dropped from 57 to 14, and the lowest their oxygen went improved from 54 to 80. I try and make a big point of telling patients who are obese that 30, 40, 50 pounds can have a significant impact. If you’re telling someone who weighs 350 pounds that they have to get back down to 170, I think that they stop listening to you right away.

If you tell them that if they got below the 300 mark or the 275 mark that they might be significantly better, I think that they are at least a little bit more open to that idea.

In terms of pharmacologic therapy, drugs are not the answer for obstructive sleep apnea, except in rare cases. Tricylic antidepressants, medroxyprogesterone and oxygen have all been used in obstructive sleep apnea.

The idea behind tricylic antidepressants is that they decrease REM sleep. Since you frequently have more severe apnea during REM, if you decrease the amount of time you spend in REM sleep, you may improve your apnea index. I use it rarely, only in patients who have mainly REM associated apneas, mild oxygen desaturation and who will not use nasal CPAP.

Medroxyprogesterone is a respiratory stimulant and that’s been used in patients with obstructive sleep apnea. It’s only been shown to be effective if they are the patients who are the daytime hyperventilators. So if they’re nighttime CO2 retainers, then this drug may have a role as a respiratory stimulant and possibly decrease apneas independently as well. It’s rarely effectively enough on its own but it may be used as an adjunct for other therapy.
Oxygen may improve oxygen saturation. Actually the idea of using oxygen to treat obstructive apnea is very appealing. People desaturate and you want to make it so they don’t desaturate so you give them oxygen. You can clean up their oximetry trace and you can make it so they don’t desaturate so much.

Unfortunately what can happen is that you can actually prolong their apneas so that the hypercarbia and acidemia worsen. Some people have used oxygen to treat mild cases of apnea. If you do that I think that it’s necessary for you to repeat a sleep study while the patient is on oxygen to make sure that you haven’t lengthened the average duration of their apneas.

Let’s move on to surgical treatment of obstructive sleep apnea. I divided it into nasal surgery, uvulopalatopharyngoplasty, maxillofacial surgery and tracheostomy. Nasal septoplasty will be helpful if patients have a severe nasal problem. Most of us are preferential nasal breathers at night, so even if you have a partial nasal obstruction, you’ll attempt to breathe through your nose and you’ll generate more negative pressure in your pharynx and tend to collapse your pharynx. Therefore sometimes straightening up a nose can help.

It’s rarely sufficient treatment for sleep apnea on its own. The most common surgery done for obstructive sleep apnea is the uvulopalatopharyngoplasty. It involves the resection of the uvula, the distal margin of the soft palate, the palatine tonsils and any excessive lateral tissue.

So you basically clean up all the loose tissue around here. The idea is to get all the soft tissue and leave the musculature below intact. Uvulopalatopharyngoplasty is generally successful about 50 percent of the time.

There are a number of studies in which patients with obstructive apnea were treated with the uvulopalatopharyngoplasty and these are the number of patients in each study and this is the success rate over here. It averages out to be 50 percent. The trick is that surgeons defined a surgical success as a 50 percent reduction in apnea index.

So if you stop breathing 50 times an hour and you go down to 20 times an hour, you’re a surgical cure. Now, you may not be any better, you may not feel any better, you may be at increased mortality still, but you’re a surgical cure. So you have to be a little bit careful about this. The failure of uvulopalatopharyngoplasty to successfully treat obstructive apnea is when you’re obstructing below the area where the uvulopalatopharyngoplasty works, in the hyperpharynx and you can’t affect this area with your uvulopalatopharyngoplasty.

There are a lot of ways of trying to determine who is an optimal candidate uvulopalatopharyngoplasty. This came from the Mayo Clinic and it’s something that I think I found practically works; if patients have an enlarged uvula, enlarged tonsils, prominent tonsillar pillars or narrow palate to posterior pharyngeal wall dimension. After looking at hundreds of pharynxes, you look at one and you say that’s a tight crowded pharynx and that patient is likely to do better with surgery than not.

An occasional patient is absolutely cured. Many patients get no relief at all. But it’s something that I think we have to continue to reserve for patients who can’t be treated any other way. A more recent concern is how long do the effects of uvulopalatopharyngoplasty last? It seems to drop off a bit with time. This may be related to the fact that patients continue to gain weight.

So it’s by no means an overall cure, but it’s a procedure that’s available for patients who can’t be treated in other ways. There are significant complications, a small but present mortality. In the hands of a good surgeon, in general patients do very well.

Something new on the horizon is more significant surgery in which both maxible and mandible are advanced and the hyodery suspended. This has had very good results, in a very small select group at Stanford. I think the most important thing to do is if you recognize someone who has an obvious jaw deformity to realize that they may be treated by this kind of surgery.

Last but not least, tracheostomy. It’s the ultimate solution for obstructive sleep apnea. You’re by-passing the site of airway obstruction, therefore by definition it should be curative. The problem is that not only do the patients not want a tracheostomy, they also don’t tolerate them terribly well. They have short fat thick sweaty necks and they get peristomal infections and they don’t tolerate the tubes and they can have bleeding. When you have nothing else left to do, that’s what you do.

Then finally, I’ve saved the best for last. Nasal CPAP or continuous positive airway pressure became available in the early 1980s and it has revolutionized the therapy of obstructive sleep apnea. It consists of an air compressor which blows air into a very tight fitting mask that fits over the patient’s nose and blows air at a very rapid rate into the posterior pharynx.

There are a number of theories on why nasal CPAP works. The most common one is that you just kind of pneumatically kind of splint the airway open with a positive airway pressure. The machines are designed to generate variable pressures from between two and 20 centimeters of water pressure. Your patient has to come into the lab to be titrated to the appropriate level.

Nasal CPAP works. It’s effective in more than 90 percent of patients with obstructive sleep apnea. The side effects are relatively minor; dry nose, dry throat, nasal congestion or rhinorrhea, sore eyes or mass discomfort. The problem is that patients get tired of using it. The initial compliance with nasal CPAP is about 80 percent. So 80 percent of the patients you sent to your lab who are able to be treated with nasal CPAP will agree to take it home and use it. If you check them after one year, it’s dropped down to 63 percent.

The others have just gotten tired of the inconveniences of using it or just don’t adjust well to the idea that they’ll have to use the
machine for the rest of their lives. I think something that’s even more disturbing is that at a recent American Thoracic Society meeting a number of groups presented the same findings, that even patients who were consistently using their CPAP, are using it four or five hours a night, not the seven or eight hours that they’re asleep. Is four or five hours enough? Does it help their desaturation enough?

I don’t think it’s clear. Nasal BiPAP allows you to independently control the inspiratory and expiratory pressures and theoretically make the device more comfortable for patients. I haven’t found that it’s made a lot of difference to patient compliance. I don’t think it’s going to be a great improvement in our current therapies. It also costs three times as much. So unless you have a really good reason for using it, I don’t think that it’s worthwhile.

So we do have therapies for obstructive sleep apnea, many more therapies than were available in the 1970s when it was trach or lose weight. But we’re far from finished with this problem.

(Applause.)

DR. STEVEN BAKER: We have time for one or two questions.

AUDIENCE MEMBER: I’ll ask one. The sleep labs can be expensive and perhaps lucrative and proprietary. I’ve also seen psychiatrists doing sleep lab studies. Are their results different from a pulmonary specialist’s sleep lab?

DR. MARTIN: I don’t think you need to have a pulmonary background to do a reasonable sleep study. What you need is somebody who is familiar with sleep disorder and preferably boarded in sleep medicine. There is a separate Board for that. If you’ve got somebody whose got those Boards I think that you can be relatively assured that they should be generating good data.

AUDIENCE MEMBER: Are there any advantages to laser therapy as opposed to the conventional surgical approach?

DR. MARTIN: Laser therapy is a new way of doing uvulopalatopharyngoplasty in which rather than putting the patient under general anesthesia, they have a series of three to four treatments in the office using a laser to resect the soft tissue from the palate. I think that there is a definite advantage to the laser therapy in that it avoids general anesthesia.

The results for the laser surgery for snoring and uvulopalatopharyngoplasty is that it is equivalent to the traditional therapy. Unfortunately the numbers are not yet out for obstructive sleep apnea. I think it’s probably worth taking the risk until it’s shown that it is effective, just in order to avoid the general anesthesia and the prolonged recovery. It takes a week to ten days to recover from a uvulopalatopharyngoplasty.

DR. STEVEN BAKER: Thanks, Dr. Martin, for that nice overview of obstructive sleep apnea.

References

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