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## **Polysomnography**

This grand rounds covers the following topics:

- stages of sleep
- indications for polysomnography
- what happens to the patient
- what is measured
- how it is interpreted
- what it all means
- treatment options

The emphasis will be on the sleep study itself as the surgical options for treatment of sleep apnea were covered in a lecture by our chairman that same day.

Sleeping and arousal are governed by a complex interaction of central nervous system components. Arousal mechanisms are governed by the reticular formation in the brainstem, referred to as the reticular activating system (RAS). It communicates with the cerebral cortex through the thalamus: sensory input from the cerebral cortex can activate the RAS and cause arousal. During sleep, fewer stimuli arise from the cortex because of a feedback loop to the cortex from the RAS. However, the cortex can be stimulated, but the stimulus intensity must be much higher than needed to cause a cortical response during consciousness.

Any talk about polysomnography must include the stages of sleep. By definition, awake is one necessary stage of sleep. Awake is characterized by alpha waves on EEG and reactivity to external stimuli is maintained. The other stages of sleep are broken into

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Non-Rapid Eye Movement (NREM) and Rapid Eye Movement (REM). NREM has three stages and REM is its own stage.

**Stage 1 NREM sleep** is characterized by drowsiness. EEG rhythms slow to mixed delta and theta waves. There is reduced muscle activity, decreased minute ventilation and increased PaCO<sub>2</sub>. Heart rate and cardiac output decrease from increase in parasympathetic tone.

**Stage 2 NREM sleep** is the next stage, a deeper sleep than stage 1. It comprises 45-55% of total sleep time. The EEG shows sigma waves and K complexes. This stage of sleep requires a higher intensity stimulus for arousal. Reduced muscle activity, decreased minute ventilation and increased PaCO<sub>2</sub> continue. Heart rate and cardiac output decreased more than Stage 1 NREM sleep from continued increase in parasympathetic tone.

**Stage 3-4 NREM sleep** was recently combined into one stage. It comprises 13-23% of total sleep time in adults. It has an EEG with characteristic slow delta waves, and this phase requires highest intensity stimulus for arousal. The respiratory rate is most regular in this phase, and this is the phase most associated with restful, restorative sleep.

The REM stage of sleep is characterized by rapid eye movements and dreaming. EEG shows mixed frequencies with alpha waves, similar in character to the awake EEG. However, skeletal muscle tone is decreased, most importantly the upper airway tone is decreased so that the airway has the least amount of protection at this stage of sleep. During REM sleep, surges in autonomic activity, suspected to be related to dreaming, can destabilize heart rate and cause fatal arrhythmias. Respiratory rate can increase because of these same surges, but actual airflow is decreased because of muscular atony. Also, hypoxic ventilatory response is depressed as well.

Now that we have covered the basics of sleep, we turn to the basics of the sleep study, formally known as polysomnography. The indications for ordering a sleep study include:

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- Diagnosis of sleep related breathing disorders
- Continuous positive airway pressure (CPAP) titration
- Assessment of treatment results (i.e. post surgical)
- With a multiple sleep latency test for assessment of narcolepsy
- For evaluating possibly injurious sleep related behaviors

How does one go about diagnosing sleep related disorders? Sleep history is key to diagnosis. Sleep disordered breathing symptoms include snoring, witnessed apneas, nocturnal choking or gasping, restlessness, and excessive daytime sleepiness are key symptoms that point towards a diagnosis of sleep disordered breathing. Epworth Sleepiness Scale can be helpful in distinguishing the severity. Evaluation of daytime sleepiness should include evidence of sleep deprivation, use of alarm clock, shift work, snoring, recent weight gain, family history, morning headache, or a sore throat or dry mouth. One should also ask about alcohol consumption, nasal congestion, hypothyroidism, and menopause as these factors can affect the restfulness and restorative

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function of sleep. A sleep log can be helpful to diagnose daytime sleepiness as being from lack of sleep time vs. pathologic sleep patterns.

Children are not just small adults. Their symptoms and treatments can differ greatly. Behavioral problems, learning problems, lack of attentiveness, and hyperactivity can characterize sleep disordered breathing in children more than classic signs of fatigue that adults suffer.

The next step towards diagnosing a sleep disorder is doing a physical exam. Findings that support diagnosis of sleep disordered breathing in adults are obesity, hypertension, and cardiopulmonary disease. In children, adenotonsillar hypertrophy and obesity point towards sleep disordered breathing.

When you send a patient to a sleep lab, what actually happens? In our sleep lab at UTMB, the patients arrive around 9 p.m. to be set up. In the pediatric population, families are allowed to sleep in the same room (but not in the bed with the patient as this can confound the readings). I decided to follow one of our patients through his setup in the sleep lab. Introducing “Tony” an adolescent patient of Dr. Harold Pine, here for follow up sleep study after his tonsillectomy and adenoidectomy performed for obstructive sleep apnea/hypopnea syndrome (OSAHS). Please refer to the “Polysomnography Demystified” powerpoint presentation for pictures and graphics.

When “Tony” is set up in our sleep lab, one of the two techs on duty that evening place the electrodes:

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- 2 occipital, 3 cranial, 3 frontal for EEG
- 2 eye movement electrodes
- 1 chin movement sensor
- 2 sensors on each leg for leg movement
- 2 EKG sensors
- one combination sensor that measures air flow in the nose or the mouth (nasal cannula pressure transducer)
- one pulse oximeter
- an abdominal movement sensor on an elastic band
- a chest movement sensor on an elastic band

The room is then darkened and the tech goes through a series of tests of the sensors to make sure they are recording properly. After that, the door is closed and the video monitor starts recording the patient sleeping. Pictures of the monitor screen and the patient in the darkened room are available on the powerpoint.

Before venturing over to our sleep lab, I searched the internet for sleep study patient pictures to use in this presentation, and came across the fascinating case of Haven. I was given permission to use her pictures by her mother if I would take a moment to talk about her syndrome.

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Haven is a 6 year old girl with a rare syndrome that has been termed ROHHAD: Rapid-Onset Obesity With Hypothalamic Dysfunction, Hypoventilation, and Autonomic Dysregulation. This syndrome is characterized most often by rapid onset obesity occurring around the age of 3. Other frequent hypothalamic dysfunction symptoms are hyperphagia, polydipsia, polyuria, and hypernatremia.

Autonomic dysregulation often follows around the age of 4, most frequently manifested in ophthalmic problems (e.g. strabismus), GI dysmotility, altered sweating, thermal dysregulation, and tumors of neural crest origin. Haven has ganglioneuroblastoma that was diagnosed in 2007 at the same time as her ROHHAD syndrome. Present in all patients is alveolar hypoventilation that begins to manifest at the average age of 6. Obstructive sleep apnea is common, and death can occur suddenly from cardiorespiratory arrest from central apneas. Seizures are also seen with this syndrome.

Haven sleeps with a pulse oximeter at night that alarms so her mom can go in and arouse her if her oxygen saturations dip too low. Haven undergoes sleep studies every 6 months to try and catch central apnea before it is symptomatic. She has confirmed obstructive sleep apnea and her mother and doctors are planning to have her tonsils and adenoids removed in the near future. Her arterial CO<sub>2</sub> used to run about 50 but she has undergone chemotherapy with cyclophosphamide, rituximab, IVIG, and prednisone that has her weight steady and her arterial CO<sub>2</sub> down to 45.

Back to the topic of polysomnography. Many things are measured during a sleep study. The EEG measures the brain's neuronal activity. Brain waves have characteristic frequencies, amplitudes, and morphologies and each stage of sleep has characteristic brain waves. Drawings of the waves are available on the powerpoint presentation.

Alpha waves are the first brain waves discovered. They are predominant in relaxed wakefulness and have a characteristic frequency of 8-13 Hz. Theta waves are predominant in Stage 1 NREM sleep and have a characteristic frequency of 12-14 Hz. Spindles and K complexes are characteristic of Stage 2 NREM sleep and they occur in setting of variable low frequency waves. Delta waves are predominant in Stage 3-4 NREM sleep and have a frequency between 0.5 and 2 Hz. The EEG of REM sleep looks very similar to Stage 1 NREM but it is characterized by rapid oscillating eye movement and skeletal muscle atony.

The EEG tracings, the muscle electrodes, and the eye movement sensors are used to break the tracings down into the stages of sleep. The American Academy of Sleep Medicine uses the following guidelines to interpret the polysomnogram:

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| Stage         | EEG  | EOG  | EMG   |
|---------------|--|--|---|
| Wakefulness   | Eyes closed; alpha prominent in the occipital region. Alpha attenuates with concentration<br>Eyes open; low voltage mixed frequency, beta activity | Voluntary control; blinks, REMs, SEMs if drowsy                      | Tonic activity, relatively high, voluntary movement |
| NREM Stage I  | Low voltage mixed frequency, theta activity, vertex sharp waves  | SEMs   | Tonic activity, slight decrease from wakefulness    |
| Stage II      | Relatively low voltage mixed frequency background. Sleep spindles and/or K complexes   | Occasionally SEMs near sleep onset; otherwise, reflects EEG activity | Tonic activity                                      |
| Stage III-IV  | >20% delta waves, 0.5–2 Hz; greater than 75 uV in amplitude  | Reflects EEG activity  | Tonic activity                                      |
| REM           | Relatively low voltage mixed frequency, possible sawtooth waves, theta activity  | Phasic REMs  | Tonic suppression, phasic twitches                  |
| Movement time | Obscured   | Obscured   | Very high activity                                  |

**Please refer to the Powerpoint presentation for typical examples of tracings.**

The other leads (chin movement, leg movement, chest wall movement, abdominal movement, air flow, both nasal and oral, and pulse oximetry) are used to characterize breathing patterns during the phases of sleep. Apnea is the cessation or near cessation of airflow for a minimum of 10 seconds. It is usually associated with desaturation and an EEG arousal at terminus. Hypopnea is a 50% decrease in airflow for at least 10 seconds followed by an arousal and/or 4% oxygen desaturation. Respiratory Event Related Arousals (RERA) are periods of increased breathing effort during increased airway resistance, with subsequent arousals, but in the absence of hypopneas, apneas, or O<sub>2</sub> desaturations.

After the polysomnogram, the study is then scored according to AASM guidelines and a report is generated. Measurements include total recording time, total sleep time, sleep latency (amount of time from lights out to sleep stage 1), number of REM periods, number of stage shifts, number of arousals, number of apneas and type, number of hypopneas, pulse oximetry, and leg movements.

The key equation that summarizes all the data and provides guidelines for treatment is the Apnea Hypopnea Index (AHI).

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An AHI greater than 5 is diagnostic for Obstructive Sleep Apnea/Hypopnea Syndrome (OSAHS) in adults. An AHI greater than 1 is OSAHS in children. An AHI of 5-30 is mild, 30-60 is moderate, and greater than 60 is severe OSAHS.

Another important equation is the **Respiratory Disturbance index**. This equation is similar to the AHI but counts the number of RERAs as well.

**Upper Airway Resistance Syndrome (UARS)** is defined as daytime sleepiness associated with a sleep breathing disorder with RERAs but not enough apneas/hypopneas to diagnose OSAHS. No consistent RDI number has been defined to make a diagnosis.

Now that we can diagnose OSAHS, what can we do about it? Treatment options include, nothing, weight loss, oral appliances, positive pressure ventilation, and a variety of surgeries. Since they are not just little adults, children's options are a bit different.

It has been noted that some patients with OSA only have it in the prone position. The obstruction disappears when the patient is lying on their side, so sleep positioning can offer a cure. These are usually mild cases with AHI <30.

The Sleep AHEAD study showed that changes in weight, waist circumference, and neck circumference were strongly associated with reduction in AHI. The best improvement in AHI was found in the group that lost more than 10 kg.

Oral appliances are designed to advance the mandible. They require a personalized dental appliance, and they're only effective in mild cases of OSAHS.

Positive pressure ventilation is a well known treatment for OSAHS. It creates a "pneumatic splint" in the upper airway to prevent collapse, and there are several variants: nasal CPAP, autotitrating CPAP, BiPAP. This treatment is only as effective as the compliance to it, reported as 46-89%. Interestingly, the least compliant patients are the ones who present for evaluation only at the urging of their spouses and not because of their own symptoms.

Surgical options abound, but have mixed efficacy. Tracheostomy is the only surgical procedure consistently effective in the treatment of OSAHS, but it is indicated only for life threatening disease such as cor pulmonale, arrhythmias, or severe hypoxemia. The surgery most frequently performed on adults for OSAHS is **uvulopalatopharyngoplasty (UPPP)**. This is generally considered only 50% effective. Some research states that this is more effective in patients with lower body mass index and lower AHI.

Another technique available surgical correction of OSAHS is **maxillomandibular advancement (MMA)**. The maxilla and the mandible are both advanced, with the mandible advanced slightly more than the maxilla. In combination with UPPP or other procedures, the success rates are reported between 66.7% and 97.8% as long as the advancement is 10 mm or more relative to the original bony position.

In children, the treatment of choice for OSAHS is adenotonsillectomy. Complications after the procedure occurs more often in children younger than 3, those with severe OSA, and those with other medical problems. In children with obesity, this

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may not completely resolve OSAHS, so in those cases, CPAP and weight loss can help if adenotonsillectomy gives an incomplete cure.

**Please refer to the Powerpoint presentation for diagrams and pictures.**

## **Discussant: Harold Pine, MD**

That was very good, Dr. Smith, and I'm sorry I wasn't able to join you at the sleep lab at nine o'clock but I have done that in the past and I think you can now appreciate what a hurdle it is for us to get one of these started. It's not the simplest thing in the world and yet it's really amazing to me that the overwhelming majority of kids are able to get hooked up, fall asleep and give us some really useful data. My hat goes off to the terrific sleep techs we have here at UTMB. I have the occasional child where they just do not tolerate being hooked up to all this stuff and they have to call it a wash.

A couple of comments about things you said or didn't say: I wanted to put in a plug for the twenty two question sleep questionnaire that I use routinely in my clinic. Dr. Chervin and his team developed this and while not meant for clinical use I find it very helpful in pointing out to parents some of the relevant issues involved. [Sleep Med.](#) 2000 Feb 1;1(1):21-32.

Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems.

[Chervin RD](#), [Hedger K](#), [Dillon JE](#), [Pituch KJ](#).

Department of Neurology, University of Michigan, Ann, Arbor, USA

A positive sleep questionnaire along with a good history helps raise your suspicions and have a meaningful discussion with parents and pediatricians alike. This is a validated questionnaire with a sensitivity around 80% and a specificity around 87% so it's certainly something easy to do and it can help you discuss with the family the likelihood of there being sleep disordered breathing.

We are all sent children for potential sleep disordered breathing and it is a very reasonable question to ask the parents, "Well, what do you see when the kid sleeps?" Some of the parents aren't terribly concerned but I think that Dr. Smith elegantly pointed out how easy it is for this issue to be missed. As we know, REM sleep happens later on in the night and apnea is worse during REM sleep. So, while the mom might

go check on her baby at eleven o'clock before she goes to bed, the child may be doing just fine. But later on in the evening when the REM sleep becomes more common, that's when the child really starts to obstruct. Again, parental reports have been proven not to be a terribly effective way to gauge obstructive sleep apnea but that certainly does not stop them from bringing in video clips on their iPhones for me to watch.

For the sleep studies themselves, a couple of interesting comments: For people who do this in the pediatric realm completely, they suggest that it's also very, very important to have a CO2 monitor because lots of kids while not having frank obstructive sleep apnea have hypoventilation and hypercarbia and that's not something we routinely measure. Could we be missing some children with obstructive hypoventilation? Also, which I find interesting, depending on where you send your patients for a sleep lab will depend on whether you get an AHI or an RDI. Now for our purposes, we're looking for the appropriate indication to do surgery. For me, it's better to get the RDI, because what might otherwise have been a negative study, the RERAs can push the final score over that magical number of RDI >1 and gives you a good indication for surgery. At Harborview, which is part of our system and at American Sleep Labs which is not part of our system, they routinely report just the AHI. When reviewing these studies done in children, if the RDI is greater than ten, most people consider that significant or severe OSA. The other thing to recognize and point out to our anesthesia colleagues is if the O2 saturation has dropped below eighty percent on the sleep study. That puts the child at higher risk of having respiratory compromise after surgery. So, I routinely point that out to our pediatric anesthesiologists. In my non-syndromic patients with a RDI < 5 who are not obese and do not have other medical issues, I feel comfortable doing their tonsillectomy and adenoidectomy surgery as an outpatient. Exact guidelines are really not out there so it pays to have a good discussion with your anesthesia colleague beforehand. If in doubt, I recommend doing the surgery in a place that has capabilities for 23 hour observation.

And then one final comment. In some of the papers I have read they suggest that when trying to make a diagnosis of obstructive sleep apnea in children, they should pull central apneas out of the equation, because, as you know, central apneas are more common in children than in adults. \* So if a kid has a few central apneas, that may push their overall RDI past the limit, while if you took those out, they still may be negative for a total RDI. As far as I can tell it all gets thrown into the pot together when calculating the RDI here.

Sleep studies are a valuable tool in helping to sort out a host of sleep issues and not just OSA. They are expensive and certainly not every child needs a sleep study but they are becoming easier to obtain and can provide very useful information. Does anyone know if there are any pure pediatric sleep labs out there? I bet it would be a lucrative venture.

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\*[Anesth Analg](#). 2009 Jul;109(1):60-75.

Perioperative management of children with obstructive sleep apnea.

[Schwengel DA](#), [Sterni LM](#), [Tunkel DE](#), [Heitmiller ES](#)

**Dr. Underbrink's remarks:**

It was very informative and I would like to encourage all of you that all of those tracings are on there and they make you mix and match which ones are sleep arousals and which ones are RERA's and it's quite involved so maybe we should all review the nuts and bolts of sleep studies because those are required for our main certification as though it would seem, though I think you could avoid it if you wanted to.

Secondly, I don't know if you know the answer to this but do you use the RDI as though it were and AHI because when I get some sleep studies that only report an RDI I don't know if there's any literature to prove that we can or can't.

**Dr. Pine:**

I use them interchangeably and I've only had a couple kids so for the most part the kids I'm seeing don't have that ...it was no apneas but there were a hundred RERA's.

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