

SLEEP IS *NOT* A RESPIRATORY DISORDER: EEG MONITORING IN THE SLEEP LABORATORY

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When most patients are evaluated in a sleep laboratory, the standard monopolar electrode derivations are sufficient for accurate staging of sleep. Yet as more sleep labs are being asked to study increasingly complicated patients, consideration and attention to a more thorough evaluation of the central nervous system (CNS) during sleep becomes valuable and important. Recording additional electroencephalographic (EEG) channels is important not only for increased accuracy in sleep staging but also for the detection of CNS abnormalities that may contribute to, or be solely responsible for, the reason the patient is referred to the sleep laboratory. Recording minimal EEG data during PSG evaluation may cause the clinician to overlook important data that can influence breathing abnormalities, unusual nighttime behaviors or movements and common daytime symptoms such as excessive daytime sleepiness (EDS) attention deficit hyperactivity disorder (ADHD) in children or adults. Epileptiform activity, an interpretive term applied to abnormal EEG activity generally, can fragment or otherwise alter sleep in a number of ways and yield symptoms commonly associated with poor sleep. While the primary focus in sleep laboratories is nighttime breathing, those laboratories studying medically complicated patients, children or patients with unusual presentations should also be familiar with the reasons and methodologies for employing an extended EEG montage array, become familiar with common epileptiform activity and be able to accurately interpret these findings to avoid missing or reporting erroneous information in these populations.

EEG abnormalities in the adult patient are often known prior to PSG evaluation but there is intrinsic clinical value in identifying changes in this known abnormal EEG activity. However, in children, EEG abnormalities may remain undiagnosed because of the sometimes subtle nature of the clinical signs associated with epileptiform activity. A patient with abnormal EEG discharges may be hyperactive, inattentive or unresponsive and these symptoms can be attributed to other causes including poor sleep-related breathing or movement disorders. Also, while accidental findings are uncommon in children, they do occur and are felt to be sufficient justification for employing an extended EEG montage. Once previously unknown discharges are discovered, a formal evaluation including an EEG is recommended.

In addition to the standard EEG montage where electrodes are placed at A1, A2, O1, O2, C3, and C4 to obtain the monopolar derivations for sleep staging, a simple bipolar EEG montage can also be used to better detect abnormal CNS activity. This typically only requires adding a few more EEG leads. Organizing your montage into an array or chain can be accomplished in a longitudinal, anterior-posterior (front to back) direction or transverse (usually left to right) derivation. The American EEG Society's Guidelines in EEG-1980 refers to these arrays as longitudinal bipolar (LB) and transverse bipolar (TB). To cover both hemispheres of the brain and localize an electrical event during a sleep study with the minimal amount of lead application, a LB array also referred to as a parasagittal montage is often sufficient. This montage includes F3-C3, C3-P3 and P3-O1 on the left and F4-C4, C4-P4 and P4-O2 on the right as a standard montage. It is also helpful to record the left parasagittal array over the right parasagittal array to maintain organization and for accurate interpretation. If unfamiliar with these lead placements, there are many resources available as a reference to help determine placement. Since accuracy in lead placement is critical to the proper interpretation of the EEG, the placement of the EEG leads should always be based on the International Ten-Twenty Electrode Placement System. Most digital polygraph systems now have the capability to record multiple channels and most extended EEG montages can be easily monitored and viewed.

More commonly seen in the sleep laboratory are patients with known epileptiform activity, or sleep disorders possibly related to EEG abnormalities such as unusual movements during sleep or sleep terrors. For these patients, this activity can usually be adequately monitored with the addition of bilateral mid-temporal leads, T3-T5 and T4-T6 (again, left over right). While positive EEG findings on a polysomnogram necessitate full EEG evaluation, the most thorough EEG montage during a sleep study would include the addition of F7 and F8 leads. The reason for this is that discharges are most likely to be seen in the temporal regions and using the frontal leads in your montage array will better show where the reversal occurs since temporal leads would then be in the middle of the lateral chain. Using this more extensive montage the clinician is more likely to be able to rule out CNS involvement. While the application of the mid-temporal leads is sufficient in most cases, if a patient's previous EEG is available, that information can also help determine the best EEG montage

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pneumothorax were present. Again, to make a long story short, the patient coded and within a minute we had 20 people around the bed, but unable to get any air into the patient. Finally, a resident drove home a chest tube and we were able to get some air into the patient. Unfortunately, though, it was too late and we were unable, after over 75 minutes, to resuscitate the woman. The unit was stunned; more so than at a "routine" unsuccessful code. As people started drifting out, the team of 20 dwindled down to about 6 of us. The 3rd year resident on duty then said, "What the hell happened here. I just told this lady's husband that she was fine and he could talk to her in the morning. What happened?" No one answered. I didn't know *what* to say but suddenly, the nurse who had been doing the bagging, began to cry hysterically as she was ushered into the hallway by 5 of her nurse coworkers. As a result, just myself and the resident were left standing at the bedside. He then said to me "What do *you* think happened here?" I didn't know what to say. Again, I didn't want to "rat out" the nurse, but at the same time I didn't want to stay silent and just say that I didn't know when I felt I did. The resident, sensing that I was holding something back, asked me to come out into the hallway with him. When we did, we of course, ran right into the nurse and her 5 colleagues, all of whom were consoling her. Upon seeing me, the five of them all gave me a dirty look as if to say "You better not blame this on our friend".

The resident, again sensing that I was hesitating due *now* to the peer pressure of six nurses, then told me to take a walk with him down the hall. I told him I wanted my supervisor present which at that moment was the closest thing I had to asking for an attorney. My supervisor came and the three of us went into a room. I told the doctor that although I was no expert and certain-

ly couldn't say for sure what had happened, that it sure looked to me that the "pneumo" had been caused by the nurse's poor bagging technique. I told them both what had transpired and after awhile we all went back to the unit. When we walked in, the doctor asked to see the nurse (with her supervisor) and of course from that moment on, my name was mud. What bothered me much more however, was that inevitably the resident had to call the husband to tell him the news. I was standing right there when he told the husband that they had no idea what had happened but that his wife's heart had simply stopped and that they had done all they could. I shook my head with disgust but didn't say anything. The husband arrived 45 minutes later crying. Again, they reiterated to him that it was just one of those "rare things". A half-hour later I actually ran into the husband, a chaplain, and the resident who were walking the poor guy to the exit. I wanted to blurt out to him that it was "our fault" and that I would be his star witness at the malpractice trial...but I didn't. I don't know why I didn't, and I wonder to this day whether I should have. I really don't know what happened to the nurse either, but I heard that *nothing* happened to her, not even a write-up (even though *my* supervisor told *her* supervisor *my* version of the story). All I *do* know is that later, on the day shift, the head nurse of the PACU told the Director of RC that she didn't want *me* working in the PACU ever again. I never did.

Read the articles by John Salyer and Paul Mathews in this issue. After doing so you may want to discuss "bagging" not only with your own staff but with anyone who might be in the position to manually ventilate a patient via a BVM.

Regards,
Bob Miglino RRT, MPS

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array to use. Since one of the most effective methods for activating epileptiform activity is sleep, employing a thorough EEG montage for sleep studies on children and adults with known CNS abnormality is necessary in order to detect any EEG changes and a relationship, if any, to disordered sleep.

Data used for the staging of sleep include the combined measurement of the EEG, the electrooculogram (EOG) to record bilateral eye movements, and the electromyogram (EMG) to record facial and intercostal muscle tone. However, because of the special criteria used to define sleep states in infants younger than 6 months old and the unique EEG features for this population, an extended EEG montage is critical and should be standard. Any infant PSG montage should also include bilateral mid-temporal EEG electrodes. EEG features specific to infants, such as tracé alternant and "brushes," as well as certain epileptiform activity can provide useful information regarding the maturity of the brain and alert clinicians to potential problems in CNS activity. Additionally, certain normal features of the infant EEG, such as rudimentary sleep spindles, are better seen using an extended EEG montage that includes frontal leads.

Epileptiform activity can also make staging the sleep record difficult and accurate and conscientious clinical descriptions from technologist performing the study are critical to alert the person scoring and interpreting the study to perceived state changes. While the descriptive language used for describing EEG activity can seem confusing, there are some basic guidelines that may be useful. EEG characteristics are described in terms of frequencies, voltages and locations. Abnormal frequencies can be faster or slower

than healthy individuals of the same age and state. Voltages can be higher or lower and can further be described as continuous, intermittent or paroxysmal (activity that emerges from background with rapid onset, reaching (usually) quite high voltage and ending abruptly). Locations of epileptiform activity can be either generalized (i.e., seen in all areas) or lateralized (i.e., asymmetrical and seen on only one side) or focal (i.e., seen in a restricted area). While there are several EEG abnormalities, main findings can usually be classified as spikes, sharp waves, spike-and-wave complexes and rhythmic hypersynchronous activity. One common sleep stage-related finding is that when many patients with discharges enter REM sleep the discharges are diminished in frequency and or amplitude or are eliminated altogether. For a more thorough explanation and description of abnormal EEG activity, the reference below is suggested.

When performing sleep studies, knowledge of the reasons and methodology for appropriately monitoring CNS activity is an important aspect for serving those patients who have certain unusual presentations or known EEG abnormalities. Even sleep studies on adult patients with a known seizure disorder should include a montage that will allow the clinician to more accurately visualize any epileptiform activity that may be present. Since epileptiform activity can yield symptoms associated with sleepiness, obtaining the "full picture" by employing a more thorough EEG montage to detect this activity is of vital importance. A thorough EEG montage is particularly important in the pediatric population where CNS problems may remain undiagnosed but early detection and treatment can have life-long consequences.