

PSGs: More Than Just the AHI

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The in-laboratory attended polysomnogram (PSG) has been the driver of the growth in Sleep Medicine for the last 25 years. Reimbursements drove construction of more laboratory space and the training of more technicians. However, within the last two years, the in-lab PSG has been increasingly replaced by home sleep testing (HST). This rapid shift has had a disastrous impact on practices built around an in-laboratory model. Reasons behind the shift have been discussed both in the lay press and in academic journals. First, HST devices are marvels of biomedical engineering, which offer patients the option of accurate testing in the comfort of their own home. Second, home studies are much cheaper than in-lab studies and thus offer value to both patients and their insurers. Implicit in these arguments is the idea that all we need from a sleep study of any type is the apnea-hypopnea index (AHI), and that continuous positive airway pressure (CPAP) is the only viable treatment for obstructive sleep apnea (OSA). We believe that this singular focus on the AHI and CPAP is detrimental to both patients and to the field. We argue here that physiology is the key to revitalizing Sleep Medicine. That is, the PSG can re-vitalize Sleep Medicine if we are able to use the valuable information that each study contains.

Using a PSG for nothing more than the AHI is as absurd as using an electrocardiogram (ECG) for nothing more than the heart rate. Instead, the ECG is interpreted to provide anatomic (e.g., ventricular hypertrophy) or functional information (localizing coronary ischemia), as well as to diagnose other conditions (e.g., pericarditis, atrial fibrillation). From this simple test, there is a wealth of information gained. In contrast, an in-laboratory PSG requires more than 20 electrodes and other sensors, approximately 45 minutes of setup time, hours of sleep time, a specialized laboratory, and the continuous presence of a trained and certified technician. At the end of the study, however, for the overwhelming majority of studies, the only “take-home” value is the AHI, or perhaps also the oxygen saturation nadir. Certainly the PSG has a role in a subset of patients, but in the vast majority of cases—that is, patients with OSA—it is clear that most of the recorded data are never used. We should be able to do better.

So what could a PSG tell us (that HST cannot)? First, we and others believe that careful study of the PSG might reveal the underlying cause of OSA in individual patients. That is, two OSA patients might have the same AHI for very different reasons. Although the majority of cases will be due to poor airway anatomy, other factors such as ventilatory control, arousal threshold, and upper airway muscle responsiveness

might be important in some.¹ We recently described a method to determine these traits, but we hypothesize that much of the same information could also be determined from a careful study of the PSG.² Similarly, careful study of inspiratory flow patterns (not just peak amplitude of flow, as respiratory events are currently scored) might tell us about timing (inspiratory vs. expiratory) or location (e.g., palate, tongue base, lateral walls) of upper airway collapse. These observations could lead to targeted OSA therapy. Second, EEG parameters may also yield useful information about the effect of sleep in a variety of biological processes. For example, EEG and cardiorespiratory coupling parameters signal glucose-insulin homeostasis.³ Focus on the AHI has limited recognition that different PSG-derived parameters may be more relevant for other clinical, patient-centered outcomes of interest.⁴ Thus, the PSG could predict individual OSA-related morbidity and mortality. Overall, sleep apnea research suggests such a personalized approach is possible in the near future, but it will require more careful diagnostic testing, not less.

In terms of treatment for patients, the era of all-night attended CPAP titration is also over, and likely unnecessary if these studies are only used to determine an effective CPAP level. Instead, we propose that an in-laboratory, technician attended study is an ideal opportunity to try the effectiveness of both CPAP and non-CPAP therapies. After a period of acclimatization to CPAP and rapid rough titration (e.g., 1 hour of sleep), other therapies such as position therapy, oral appliance or suction device, nasal insufflation, oxygen therapy, nasal valves, etc. could be tried for ~1 hour each, with the choice of attempted therapies informed by the baseline PSG. In-lab studies will only make sense if we move beyond the AHI, and are able to provide three key pieces of information to patients: (1) why they have OSA, (2) what OSA-related complications they are at risk of, and (3) what non-CPAP treatment options are likely to provide benefit.

While ambitious, we believe that this goal is attainable. Indeed, it might be essential for the success of Sleep Medicine. Our dependence on the AHI has reduced the care of OSA patients to treatment algorithms that minimize the role of the Sleep Medicine physician. Interesting physiology and multiple options for treatment could restore the physician's role, and draw in the best and brightest residents who are interested in practicing the art of medicine in an exciting and dynamic field. Again, using the ECG analogy above, witness the growth in cardiac electrophysiology over the last 25 years. The emphasis on CPAP has also kept some patients away from the clinic;

understanding which patients might be treated without CPAP could improve their quality of life and prevent morbidity. If we can justify bringing back the in-lab attended PSG, it will be because we have better understood OSA pathophysiology, put the PSG information to good use, and are better serving our patients.

CITATION

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DISCLOSURE STATEMENT

Drs. Owens and Wellman were previously employed by Sleep HealthCenters, LLC. Dr. Wellman is a consultant for Respronics, Sova, and Apnicure. Dr. Owens is a consultant for Apnicure. Dr. Edwards has no conflict of interest to disclose.