Use of overnight pulse oximetry and a type 3 sleep study to titrate hypoglossal nerve stimulation therapy

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This is a case series of 3 patients with moderate-severe OSA who were PAP-intolerant and underwent implantation of the hypoglossal nerve stimulator. All patients recorded baseline overnight pulse oximetry without the hypoglossal nerve stimulator and at least 1 night at each hypoglossal nerve stimulator setting as they up-titrated the device at home. Because of the impact of the novel coronavirus on sleep laboratories, all patients proceeded directly to type 3 sleep studies performed at a single setting determined by a combination of self-reported improvement and pulse oximetry data.

Keywords: obstructive sleep apnea, hypoglossal nerve stimulation, overnight pulse oximetry

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INTRODUCTION

OSA is a common disorder with short-term quality-of-life impairment and long-term cardiovascular and neurocognitive health risks.1 Patients often struggle to tolerate the first-line therapy of PAP, and a subset of these patients undergo implantation of the hypoglossal nerve stimulator (HNS; Inspire Medical Systems, Golden Valley, MN), which was approved by the U.S. Food & Drug Administration in 2014 for patients who are CPAP-intolerant and have moderate-to-severe OSA.2 After implantation and activation of the HNS, the device is slowly up-titrated until patient- and bed partner–reported symptom improvement (eg, daytime energy level, sleep quality, snoring). Given that moderate-severe OSA commonly causes nocturnal hypoxemia, overnight pulse oximetry represents another strategy to assess therapy efficacy in the home that is inexpensive, easy to perform, and an objective measure. Properly titrating HNS is of importance because patients who are not at or close to their therapeutic level typically have a suboptimal in-lab titration polysomnogram (tPSG). In stark contrast to pulse oximetry, type 1 polysomnography is an expensive and labor-intensive test. Therefore, optimizing HNS with this additional objective measure before in-lab titration maximized the utility of this study and minimized the likelihood of repeat testing because of an inadequate in-lab titration. In a patient with good self-reported improvement and resolution of nocturnal hypoxemia, one can consider proceeding directly with a type 3 sleep study using an effectiveness home sleep apnea test (eHSAT), which measures airflow, pulse oximetry, and thoracic effort, at a single setting.3

REPORT OF CASES

Case 1
A male patient aged 62 years with a history of severe residual OSA after septoplasty and uvulopalatopharyngoplasty (Table 1; AHI = 95.2 events/h; oxygen [O2] nadir = 72%; time spent below an O2 saturation level of 88% [T88] = 71.7 minutes). underwent drug-induced sleep endoscopy that revealed predominantly tongue-base-related obstruction and partial residual anteroposterior collapse of the soft palate (VOTE score V1apT2)4 and a subsequent implantation of an HNS.

After the implant surgery but before device activation, the patient started measuring overnight oximetry levels with the EMay wireless pulse oximeter (Emay Limited, Wanchai, Hong Kong). This commercially available product was selected because it has a sampling rate of 1 second and can run continuously for 14 hours, storing 40 hours of data at a time. The device transmits data to a proprietary smartphone application that allows for O2 measurements such as O2 nadir, O2 maximum, and an adjustable O2 threshold level (eg, T88). After recording several nights to serve as baseline oximetry data points in addition to his PSG, we began to systematically record his O2 level every night while he received therapy. As shown in Table 1, he had an immediate improvement in O2 parameters even at the lowest settings such that we began tracking his time spent below an O2 saturation level of 93 rather than his T88. His self-reported response was outstanding as well. We had initially planned to proceed to a tPSG as informed by his self-reported response and oximetry data, but because of the COVID-19 pandemic we proceeded directly to an eHSAT (Alice NightOne, Philips Respironics, Murrysville, PA). As seen in Table 1, the patient had a robust objective response on the eHSAT, with most of the events and hypoxemia confined to two 15-minute periods of time corresponding to device pauses. We are therefore planning to delay his tPSG indefinitely for now.

Case 2
A female patient aged 70 years with asthma and severe OSA (AHI = 36.5 events/h; O2 nadir = 78%; T88 = 13.2 minutes) underwent a drug-induced sleep endoscopy revealing a predominantly...
tongue-based obstruction (V1apT2) and subsequently received an HNS implantation. Based on the experience of Patient 1, the patient opted to purchase the same pulse oximeter and record nocturnal oximetry as she began HNS therapy. She had a robust self-reported and oximetry response (O2 nadir = 89%; T88 = 0 minutes at final setting). We decided to proceed directly to an eHSAT, which showed excellent objective response despite the study being performed at a lower setting because the patient had difficulty sleeping with the eHSAT equipment in place and had 80 minutes of sleep time without HNS because the therapy timed out after 8 hours with the eHSAT still recording (AHI = 14.1 events/h; O2 nadir = 87%; T88 = 2.4 minutes; oxygen desaturation index [ODI] = 14.7). Similar to the results with the first patient, we will delay the tPSG for this patient because the HNS was clearly effective when it was on.

### Case 3

A male patient aged 58 years with moderate OSA without hypoxemia (AHI = 25.2 events/h; O2 nadir = 90%; time spent below an O2 saturation level of 90 = 0 minutes) underwent a drug-induced sleep endoscopy that showed multilevel collapse (V1apO2T2). After discussing a wide variety of treatment options, he opted to proceed with the HNS despite the presence of complete oropharyngeal wall collapse, which is a negative predictor of response to therapy (P. Huyett, MD, et al., unpublished data, 2020).5 Again, after learning of Patient 1’s experience with overnight oximetry, the patient began recording nocturnal oximetry as he began HNS treatment. His self-reported response was excellent (no longer falling asleep while driving, able to sleep through the night [which he never could untreated or with PAP]), but his oximetry data were mixed. The patient was unable to proceed beyond his fourth setting because of discomfort but was satisfied with his improvement in excessive daytime sleepiness in particular. We proceeded to an eHSAT, which showed improvement to mild OSA (AHI = 13.9 events/h; O2 nadir = 86%; T88 = 0.3 minutes; 3% ODI = 19.5/h; 4% ODI = 4.6/h). Given his inability to progress beyond setting 4 and a strong suspicion of a low arousal threshold, we opted to use his oral appliance therapy device and the HNS concurrently to reduce the therapeutic setting on each.6,7 His wife, who had noted persistent snoring with the HNS alone, noted complete resolution of snoring with the oral appliance therapy plus HNS. We will plan to perform a tPSG with the oral appliance therapy in place.

### DISCUSSION

This case series is the first to report that overnight oximetry has the potential to enhance postactivation HNS titration in several ways. First, it provides immediate feedback of efficacy to patients through smartphone-based applications. This is perhaps most useful in patients who are struggling to accommodate to the stimulation and/or the relatively asymptomatic patients who require positive reinforcement.

Second, it provides objective data points to inform when patients are at or near their optimal therapeutic setting. The traditional data points are all self-reported in nature and are not always reliable, particularly in relatively asymptomatic patients. The initial postactivation protocol was to perform the tPSG at 1 month. Given that patients often required longer to find this therapeutic “sweet spot,” the tPSG was recently pushed back to 3 months according to the official company protocol. Rather than following a set timeframe, we can consider referring patients for their in-lab titration study as informed by the pulse oximetry data.

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**Table 1—Baseline PSG, eHSAT, and oximetry data during home titration of HNS for Patient 1.**

<table>
<thead>
<tr>
<th>Study Type</th>
<th>AHI (events/h)</th>
<th>Average O2 Saturation (%)</th>
<th>O2 nadir (%)</th>
<th>T88 (min)</th>
<th>T93 (min)</th>
<th>ODI 3% (h)</th>
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</thead>
<tbody>
<tr>
<td>Baseline PSG</td>
<td>95.2</td>
<td>92</td>
<td>72</td>
<td>71.7</td>
<td>—</td>
<td>79.6</td>
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<tr>
<td>Baseline HNS off pulse oximetry</td>
<td>—</td>
<td>—</td>
<td>80</td>
<td>74.5</td>
<td>74.5</td>
<td>—</td>
</tr>
<tr>
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<td>—</td>
<td>—</td>
<td>87</td>
<td>3.5</td>
<td>97.5</td>
<td>—</td>
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<tr>
<td>HNS setting 1.6 pulse oximetry</td>
<td>—</td>
<td>—</td>
<td>84</td>
<td>0.75</td>
<td>70</td>
<td>—</td>
</tr>
<tr>
<td>HNS setting 1.7 pulse oximetry</td>
<td>—</td>
<td>—</td>
<td>86</td>
<td>0.77</td>
<td>41</td>
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<tr>
<td>HNS setting 1.8 pulse oximetry</td>
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<td>—</td>
<td>85</td>
<td>0.82</td>
<td>45.5</td>
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<td>—</td>
<td>87</td>
<td>0.2</td>
<td>23.4</td>
<td>—</td>
</tr>
<tr>
<td>HNS setting 2.0 pulse oximetry</td>
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<td>—</td>
<td>89</td>
<td>0</td>
<td>43.7</td>
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<td>88</td>
<td>0</td>
<td>139.7</td>
<td>—</td>
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<tr>
<td>HNS setting 2.2 pulse oximetry</td>
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<td>88</td>
<td>0</td>
<td>29.2</td>
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<tr>
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<td>87</td>
<td>0</td>
<td>34.7</td>
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<tr>
<td>HNS setting 2.4 pulse oximetry</td>
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<td>—</td>
<td>89</td>
<td>0</td>
<td>73.6</td>
<td>—</td>
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<tr>
<td>HNS setting 2.5 pulse oximetry</td>
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<td>89</td>
<td>0</td>
<td>45.2</td>
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</tr>
<tr>
<td>eHSAT, HNS setting 2.8</td>
<td>12.3</td>
<td>93</td>
<td>84</td>
<td>5</td>
<td>—</td>
<td>14.8</td>
</tr>
</tbody>
</table>

Note that during the HSAT the patient paused therapy twice, accounting for the hypoxemia and virtually all respiratory events. Settings 2.6 and 2.7 are not shown because when the remote is programmed for greater than 10 settings, it is not possible to know the device setting with certainty except for the maximum setting, eHSAT = effectiveness home sleep apnea test, HNS = hypoglossal nerve stimulation, O2 = oxygen, ODI = oxygen desaturation index, PSG = polysomnogram, T88 = time spent below O2 saturation of 88%, T93 = time spent below O2 saturation of 93%.
Finally, it is possible that patients who have good self-reported improvement and normalized oximetry can proceed directly to a type 3 eHSAT at a single setting rather than using the tPSG. This would potentially obviate the need for the tPSG in all patients, which obviously has cost and convenience implications.

The major limitation of type 3 sleep study testing is the inherent underestimate of OSA severity because of the lack of hypopneas qualified by arousals and because study run time rather than total sleep time is used in the denominator of the AHI. Therefore, when one is comparing a preoperative PSG to a postoperative HSAT, the results may seem slightly better than when comparing a PSG to a PSG. Rule 1b (4%) AHI or ODI may represent a better outcome measure to report when comparing PSG data to HSAT data. Another limitation is that the interpreting physician cannot know for sure when therapy is paused or has timed out (returning patients to their baseline OSA severity), as was the case with Patients 1 and 2. On the tPSG, one can see that the HNS is off in the chin electromyography or as tagged by the technician, allowing for exclusion of these data from the titration table. In addition, during an eHSAT there is no opportunity for device adjustments. However, the tPSG for HNS can be extremely misleading—especially when a setting is reported that is tested during favorable sleep conditions (eg, stage N3, lateral position). Despite these limitations, the full-night single-setting eHSAT (efficacy × usage) is increasingly being accepted as the definitive assessment of HNS therapy because it most accurately represents how the patient is using HNS therapy on a nightly basis.

The utility of overnight pulse oximetry is limited in those patients in whom hypoxemia is not a prominent feature of their OSA, such as in the low-arousal-threshold endotype. Patient 3 highlights exactly this situation: his baseline hypoxemia was minimal as defined by the O₂ nadir and T88, which are the current data points reported by the EMay pulse oximeter. To address this issue, we worked with the EMay to develop an algorithm to capture the 3% and 4% ODI. With a plethora of pulse oximeters on the market, this product was initially selected because of its 1-second sampling rate, ability to run continuously for 14 hours, low cost, data storage and manipulation capabilities, and user-friendly smartphone application. In patients with or without significant hypoxemia, pulse oximetry also offers the opportunity to track improvements in heart rate variability although it is most helpful to interpret the data in the context of therapy adherence through the use of Inspire cloud (Inspire Medical Systems, Golden Valley, MN) or otherwise.

CONCLUSIONS

In summary, the addition of pulse oximetry to self-reported improvements during HNS home titration seems to be of value, although future larger studies are needed. The implications include positive patient reinforcement, identification of readiness for tPSG, and possibly proceeding directly to eHSAT.

ABBREVIATIONS

eHSAT, effective home sleep apnea test
HNS, hypoglossal nerve stimulator
ODI, oxygen desaturation index
T88, time spent with oxygen saturation at or below 88%

REFERENCES


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DISCLOSURE STATEMENT

Both authors have reviewed and approved the manuscript. Work for this study was performed at the Division of Sleep Medicine and Surgery at the Massachusetts Eye and Ear Infirmary. The authors report no conflicts of interest.