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Clinical Radiographic Predictors of Response to Hypoglossal Nerve Stimulation for Obstructive Sleep Apnea

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Abstract

Objective.—To determine if clinically acquired cephalometric measurements, specifically soft palate size, can predict hypoglossal nerve stimulation outcomes.

Study Design.—Combined prospective cohort study and retrospective review.

Setting.—US sleep otolaryngology training program.

Methods.—Adults with obstructive sleep apnea and apneahypopnea index greater than 15 events/h who underwent hypoglossal nerve stimulation. Eligible subjects had diagnostic preoperative sleep studies and full-night efficacy postoperative studies for analysis. Lateral neck x-rays were obtained as part of routine clinical care and measured for key cephalometric variables by trained head and neck radiologists. Continuous variables were compared using the Student *t* test, while χ^2 testing was used for categorical variables.

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Author Contributions

Clara H. Lee, design, conduct and analysis of the research; **Everett G. Seay**, conduct, analysis and presentation of the research; **James W. Reese**, conduct of the research; **Xin Wu**, conduct of the research; **Richard J. Schwab**, analysis of the research; **Brendan Keenan**, analysis of the research; **Raj C. Dedhia**, design, conduct, analysis and presentation of the research.

Competing interests: None.

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This work was presented as a poster abstract at the Associated Professional Sleep Societies (APSS) Meeting; June 9, 2019; San Antonio, Texas.

Results.—Fifty-one patients met all study criteria. On average, patients were white, middle aged, and overweight. Following hypoglossal nerve stimulation, the overall cohort achieved a significant apnea-hypopnea index reduction from 36.7 events/h to 20.6 events/h ($P < .01$) and a response rate of 47% (defined as apnea-hypopnea index reduction $>50\%$ and apnea-hypopnea index <20 events/h). On average, therapy responders had significantly thinner soft palates than nonresponders (13.4 ± 3.8 mm vs 16.0 ± 3.4 mm, $P = .045$).

Conclusions.—Patient-specific anatomic factors, specifically soft palate thickness, may help identify optimal candidates for hypoglossal nerve stimulation. A larger, prospective study including both anatomic and physiologic variables is required to validate these findings.

Keywords

obstructive sleep apnea; hypoglossal nerve stimulation; radiograph; outcomes

Obstructive sleep apnea (OSA) is characterized by upper airway collapse during sleep, leading to oxygen desaturations and recurrent arousals. OSA is estimated to affect 13% to 33% of men and 16% to 19% of women worldwide, representing a significant public health burden.¹ Patients with OSA are at increased risk for a variety of health risks, including cardiovascular disease and neurocognitive dysfunction.^{2,3} First-line therapy for OSA is positive airway pressure (PAP).⁴ With long-term use, PAP has been shown to partially reverse adverse health effects.^{5,6} However, a major barrier to effective therapy is compliance; 46% to 83% of patients report inconsistent PAP usage.⁷ Therefore, alternative therapies are essential.

Over recent years, hypoglossal nerve stimulation (HGNS) has presented a promising new treatment modality for patients unable to tolerate PAP.⁸ Five-year data from the Stimulation Therapy for Apnea Reduction (STAR) trial demonstrated long-term reductions of apnea-hypopnea index (AHI) and improvement of sleep symptoms. Yet, the overall response rate (defined as AHI <20 events/h and $>50\%$ reduction of AHI) remains around 66%, leaving room for optimization.^{9,10}

A growing body of literature has focused on identifying patient characteristics associated with HGNS therapy success. Initial feasibility studies reported the greatest response rates in patients with AHI <50 events/h, body mass index (BMI) <32 kg/m², and lack of complete circumferential palatal collapse on drug-induced sleep endoscopy (DISE). Recently, our group investigated therapeutic PAP level as a predictor for HGNS success, identifying a 92% success rate in patients with PAP values less than 8 cm H₂O, compared to 44% success in patients with higher PAP values.¹⁰

In addition, anatomical factors may play a role in determining which patients benefit most from HGNS. Schwab et al¹¹ used awake computed tomography (CT) to identify anatomical differences between HGNS patients. Therapy responders were found to have a smaller soft palate volume than nonresponders, as well as increased tongue displacement, greater increase in retroglossal airway size, and increased shortening of the mandible-hyoid distance with therapy activation. These results suggest the possibility of using patient-specific anatomy to stratify patients in regard to their likelihood of success with HGNS therapy.

In this novel study, we posed the following research question: can cephalometric measurements, acquired in the clinical setting, predict HGNS outcomes? Our primary hypothesis was that nonresponders would have larger soft palates than responders. Our secondary hypothesis was that nonresponders would have a longer mandibular plane-hyoid distance. Exploratory outcomes included mandible length and posterior nasal spine to vertebral body distance.

Materials and Methods

A prospective cohort study was approved by the Emory University Institutional Review Board (IRB00088402). Subjects were recruited from May 2016 to October 2018 at the sleep surgery clinic of the senior author (R.C.D.). A retrospective chart review was subsequently added to include patients from September 2015 to May 2016 to maximize sample size. Inclusion criteria were >18 years of age and HGNS for treatment of OSA. Indications for HGNS were AHI >15 events/h on the most recent diagnostic sleep study, PAP intolerance, and lack of complete circumferential palatal collapse on DISE. Patients were excluded if they were missing any of the following: preoperative diagnostic sleep study, full-night postoperative efficacy study, and lateral neck x-ray.

Medical record extraction was performed by C.H.L. and E.G.S. Data were manually entered into an Excel spreadsheet (Microsoft) and stored on a password-protected institutional server. Variables extracted from the medical record were age, sex, race, BMI, and medical/surgical history. Variables extracted from sleep studies were AHI and 4% oxygen-desaturation index (ODI4). Preoperative values were obtained by averaging values from the most recent diagnostic sleep study and all studies obtained within 3 years prior to HGNS. Postoperative values were extracted from efficacy studies, which were obtained between 3 and 12 months after implantation. Efficacy studies represent a full night of sleep at a single device setting. When possible, HGNS use was verified through Inspire Cloud (Inspire Medical Systems) software that allows remote monitoring of HGNS compliance.

Lateral neck x-rays were obtained in the postanesthesia care unit following HGNS implantation as part of routine clinical care. The following key cephalometric variables were retrospectively measured by trained head and neck radiologists (J.W.R. and X.W.): soft palate length (cm), soft palate area (mm²), soft palate thickness (mm), mandibular length (cm), posterior nasal spine to C1 vertebra distance (cm), and mandibular plane to hyoid distance (cm). The length of the palate was measured by drawing a multisegmented center line through the plane of the soft palate, from the interface of the hard and soft palates to the tip of the uvula. Soft palate thickness was measured at the point of maximal width. In images where the soft palate was positioned against the tongue or posterior pharyngeal wall and therefore indistinguishable, measurements were excluded. Mandibular plane to hyoid distance measurements were excluded in patients with mouths in the open position. Mandibular length was not measured if the mandible was not entirely in the field of view. In addition, the degree of patient rotation at the time of x-ray was graded as minimal, moderate, or severe based on the degree of centered overlap between the left and right mandibular rami, as this would limit accuracy of the above measurements.

Data analyses were performed with Stata/SE 14.2 (Stata-Corp LP). Therapy response was assessed according to Sher criteria (AHI reduction >50% and AHI <20 events/h).¹² Categorical data are presented using frequencies and percentages and continuous data summarized using means and standard deviations. Where applicable, changes or percent changes in measures were calculated as postoperative minus preoperative values. For unadjusted analyses, the Student *t* test was used to compare continuous values between responders and nonresponders, while χ^2 testing was used to compare categorical variables. Analyses of primary continuous anatomy measures were performed unadjusted and controlling for established clinical covariates of age, sex, and BMI,¹³ using linear regression models with responder status as a binary predictor. To understand the predictive ability of anatomy and clinical covariates, both alone and in combination, we calculated the area under the receiver operating characteristic curve (AUC) and 95% CI for each variable individually, for the 3 covariates combined, and for the combination of covariates and any anatomical variables that differed between responders and nonresponders. As a sensitivity analysis, assessments were repeated after excluding patients with prior palatal surgery (n = 7) using the same methods.

Results

Sample Characteristics

Fifty-one patients met all study criteria. Their demographics and baseline polysomnographic measurements are described in Table 1. On average, patients were middle aged (62.3 ± 14.5 years) and overweight (28.7 ± 4.1 kg/m²), and most were male (59%) and white (82%). The most common non-OSA sleep disorders were insomnia (n = 17 [33%]) and restless leg syndrome (n = 6 [12%]). Twenty-seven patients (53%) had prior upper airway surgery, including 12 tonsillectomies, 11 nasal surgeries, 7 palatal surgeries, and 1 maxillomandibular advancement. Of the 7 patients with neurologic disease (14%), 3 had trisomy 21, 2 had prior stroke without residual deficits, and 2 had mild dementia. Nineteen patients (37%) had moderate OSA (AHI 15–29.9 events/h) and 32 (63%) had severe OSA (AHI ≥ 30 events/h).

Following HGNS, the overall cohort achieved a significant AHI reduction from 36.7 events/h to 20.6 events/h ($P < .01$). The mean AHI reduction was 16.1 events/h. The overall response rate (defined as AHI reduction >50% and AHI <20 events/h) was 47%. There were no statistically significant differences between responders (n = 24) and nonresponders (n = 27) in regard to age (65.4 ± 15.5 vs 59.6 ± 13.2 years; $P = .16$), BMI (27.7 ± 4.5 vs 29.6 ± 3.6 kg/m²; $P = .11$), or baseline AHI (37.3 ± 14.8 vs 36.2 ± 20.1 events/h; $P = .83$).

Associations With Primary Outcome Variable (>50% Reduction in Overall AHI With AHI <20)

Representative cephalometric measurements are depicted in Figure 1. Measurements were compared between therapy responders and nonresponders in Table 2. Upon analysis of soft palate thickness (available in 36 patients), therapy responders (n = 16) had significantly thinner soft palates than nonresponders (n = 20) (13.4 ± 3.8 mm vs 16.0 ± 3.4 mm; $P = .045$). When adjusting for differences in clinical factors (age, sex, BMI), differences in soft

palate thickness remained similar to unadjusted analyses (-2.6 mm [$P = .042$] vs -2.1 mm [$P = .061$]), although the P value increased to slightly above .05. There were no differences in soft palate length or area. Second, therapy responders and nonresponders had similar mean mandibular plane to hyoid distances (3.8 ± 1.5 vs 3.5 ± 1.3 cm; $P = .46$). Exploratory analyses of mandibular length and posterior nasal spine to C1 vertebra distance revealed no significant differences between groups. After excluding 7 patients with previous palatal surgery, soft palate thickness remained significantly different between therapy responders ($n = 13$) and nonresponders ($n = 18$) (12.7 ± 3.5 mm vs 15.6 ± 3.3 mm; $P = .027$).

In order to evaluate the predictive ability of soft palate thickness and clinical factors (age, sex, BMI, and AHI), we evaluated the AUC shown in Table 3. Soft palate thickness resulted in an AUC (95% CI) of 0.712 (0.531–0.984), which was higher than any individual covariates alone. Combined age, sex, and BMI resulted in an AUC (95% CI) of 0.668 (0.509–0.827), which improved to 0.737 (0.566–0.909) with the addition of soft palate thickness to the predictive model (Figure 2).

In regard to image quality, 75% of patients ($n = 38$) had none to minimal body position rotation, 22% ($n = 11$) had moderate rotation, and 4% ($n = 2$) had severe rotation.

Discussion

This is the first study to use lateral neck x-rays to examine the predictive value of cephalometric variables in the evaluation of patients for candidacy for HGNS. In accordance with prior studies, therapeutic responders had significantly thinner soft palates than nonresponders. The remainder of measurements, including soft palate length and area, did not have a predictive value for HGNS response. Overall, our results suggest that lateral neck x-rays may play a role in the preoperative evaluation for HGNS candidates; patients with thinner soft palates may be more likely to succeed with HGNS.

Plain film radiographs in the way of cephalometry have been used extensively to screen for OSA, as well as predict response to therapy. Neelapu et al¹⁴ performed a systematic review and meta-analysis of cephalometric studies between OSA and control patients. Three measurements were found to be significantly different for OSA patients compared to controls: increased mandibular plane to hyoid distance, decreased pharyngeal airspace, and increased anterior facial height. More important, the mandibular plane–hyoid distance measurement has shown to have implications on treatment response. Millman et al¹⁵ studied the outcomes of 46 patients undergoing uvulopalatopharyngoplasty. A mandibular plane–hyoid distance of <20 mm was associated with surgical success. A systematic review of mandibular advancement devices also showed that reduced mandibular plane–hyoid distance was 1 of 2 predictive cephalometric measures of success.¹⁶ Given the relevance of plain film measures for both OSA screening and treatment success, the present study sought to explore the measurements of lateral plain films on HGNS results. While baseline mandibular plane–hyoid distance was not significant in the current study, previous work has shown that the amount of reduction of this distance was greater in responders than in nonresponders.¹¹

Our findings are in agreement with a recent study, which used CT to examine anatomic differences between HGNS responders and nonresponders. In the study by Schwab et al,¹¹ 13 patients with OSA who were implanted with HGNS underwent CT imaging (mean age, 53 years; BMI, 27.8 kg/m²; baseline AHI, 33.5 events/h). Under awake conditions, responders (n = 7) and nonresponders (n = 6) were imaged with and without HGNS stimulation to assess for differences in anatomical structures. Responders were found to have a smaller soft palate volume when compared to nonresponders (8789.3 ± 1811.4 vs 11,394.3 ± 2217.1 mm³, *P* = .03). Similar to the present study, mandibular plane to hyoid distances were not different between groups. Schwab et al¹¹ measured the retropalatal airway, analogous to the posterior nasal spine to C1 distance in this study, and did not find this variable to demonstrate significant differences between groups. Previous work by our group has shown that patients with lower therapeutic PAP levels have improved outcomes compared to those with higher levels.¹⁰ It stands to follow that tissue bulk (ie, thickness) as measured by volume (3-dimensional studies) or thickness (plain films) may better capture airway distensibility than measures of length (eg, soft palate length, mandibular plane–hyoid distance).

We acknowledge several limitations of our study. First, radiographs were obtained by the radiology technicians in a clinical setting, leaving room for inconsistency across patients. For example, we were unable to obtain soft palate thickness measurements in 29% of patients. In addition, obtaining radiographs in the immediate postoperative period may capture expected edema. While we deliberately avoided measures that may be directly affected by postintubation edema (eg, tongue base, epiglottis), we recognize that preoperative imaging is superior. We have changed our practice to include noncontrast computerized tomography on all patients undergoing HGNS. In evaluating our post hoc power calculations (Table 2), our study was underpowered (less than 0.8) for all variables, introducing the possibility of a type II error. Thresholds for power calculations were created using clinical experience as there are no normative data available to our knowledge. An additional methodological limitation exists in the absence of a prespecified soft palate measurement; instead, our assessment included 3 soft palate parameters (length, thickness, and area). In terms of racial diversity, our study included 82% whites. Existing literature demonstrates that ethnic phenotypes influence anatomic determinants of OSA risk and, by extension, response to non-PAP therapies. Our lack of racial diversity potentially limits the generalizability of our findings.¹⁷

Our study has several strengths. The use of clinically available radiographs offers readily generalizable and applicable results. Our image measurements were performed by trained neuroradiologists. Our study includes full-night efficacy studies at a single HGNS setting to obtain our primary outcome variable of AHI, providing accurate HGNS results.¹⁸ Not surprisingly, our AHI response rate is lower compared to studies using titration polysomnograms in which vulnerable sleep states (stage REM) and positional data are limited at each HGNS setting. When home sleep studies were performed, Inspire Cloud was used to verify HGNS use during these studies. In addition, our study encompasses the practice of a single surgeon, ensuring consistency in surgical decision making, surgical technique, and perioperative care.

In summary, our findings support that patient-specific anatomic factors, specifically soft palate thickness, may help identify patients more likely to achieve success with HGNS. Lateral neck x-rays represent an adjunct to the preoperative evaluation of HGNS candidates. A larger, prospective sample with a singular radiographic measure is required to validate these findings, with the potential to change future clinical practice for patients undergoing HGNS.

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References

1. Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev.* 2017;34:70–81. [PubMed: 27568340]
2. Kendzerska T, Mollayeva T, Gershon AS, Leung RS, Hawker G, Tomlinson G. Untreated obstructive sleep apnea and the risk for serious long-term adverse outcomes: a systematic review. *Sleep Med Rev.* 2014;18(1):49–59. [PubMed: 23642349]
3. Beebe DW, Groesz L, Wells C, Nichols A, McGee K. The neuropsychological effects of obstructive sleep apnea: a meta-analysis of norm-referenced and case-controlled data. *Sleep.* 2003;26(3):298–307. [PubMed: 12749549]
4. Epstein LJ, Kristo D, Strollo PJ, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med.* 2009;5(3):263–276. [PubMed: 19960649]
5. Cao MT, Sternbach JM, Guilleminault C. Continuous positive airway pressure therapy in obstructive sleep apnea: benefits and alternatives. *Expert Rev Respir Med.* 2017;11(4):259–272. [PubMed: 28287009]
6. Marin JM, Carrizo SJ, Vicente E, Agusti AGN. Long-term cardiovascular outcomes in men with obstructive sleep apnoeahypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet Lond Engl.* 2005;365(9464):1046–1053.
7. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. *Proc Am Thorac Soc.* 2008;5(2):173–178. [PubMed: 18250209]
8. Strollo PJ, Soose RJ, Maurer JT, et al. Upper-airway stimulation for obstructive sleep apnea. *N Engl J Med.* 2014;370(2):139–149. [PubMed: 24401051]
9. Woodson BT, Strohl KP, Soose RJ, et al. Upper airway stimulation for obstructive sleep apnea: 5-year outcomes. *Otolaryngol Head Neck Surg.* 2018;159(1):194–202. [PubMed: 29582703]
10. Lee CH, Seay EG, Walters BK, Scalzitti NJ, Dedhia RC. Therapeutic positive airway pressure level predicts response to hypoglossal nerve stimulation for obstructive sleep apnea. *J Clin Sleep Med.* 2019;15(8):1165–1172. [PubMed: 31482839]
11. Schwab RJ, Wang SH, Verbraecken J, et al. Anatomic predictors of response and mechanism of action of upper airway stimulation therapy in patients with obstructive sleep apnea. *Sleep.* 2018;41(4).
12. Sher AE, Schechtman KB, Piccirillo JF. The efficacy of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. *Sleep.* 1996;19(2):156–177. [PubMed: 8855039]
13. Deng X, Gu W, Li Y, Liu M, Li Y, Gao X. Age-group-specific associations between the severity of obstructive sleep apnea and relevant risk factors in male and female patients. *PLoS One.* 2014;9(9):e107380. [PubMed: 25211035]
14. Neelapu BC, Kharbanda OP, Sardana HK, et al. Craniofacial and upper airway morphology in adult obstructive sleep apnea patients: a systematic review and meta-analysis of cephalometric studies. *Sleep Med Rev.* 2017;31:79–90. [PubMed: 27039222]

15. Millman RP, Carlisle CC, Rosenberg C, Kahn D, McRae R, Kramer NR. Simple predictors of uvulopalatopharyngoplasty outcome in the treatment of obstructive sleep apnea. *Chest*. 2000;118(4):1025–1030. [PubMed: 11035673]
16. Guarda-Nardini L, Manfredini D, Mion M, Heir G, Marchese-Ragona R. Anatomically based outcome predictors of treatment for obstructive sleep apnea with intraoral splint devices: a systematic review of cephalometric studies. *J Clin Sleep Med*. 2015;11(11):1327–1334. [PubMed: 25979102]
17. Lee RWW, Vasudavan S, Hui DS, et al. Differences in craniofacial structures and obesity in Caucasian and Chinese patients with obstructive sleep apnea. *Sleep*. 2010;33(8):1075–1080. [PubMed: 20815189]
18. Dedhia RC, Woodson BT. Standardized reporting for hypoglossal nerve stimulation outcomes. *J Clin Sleep Med*. 2018;14(11):1835–1836. [PubMed: 30373702]

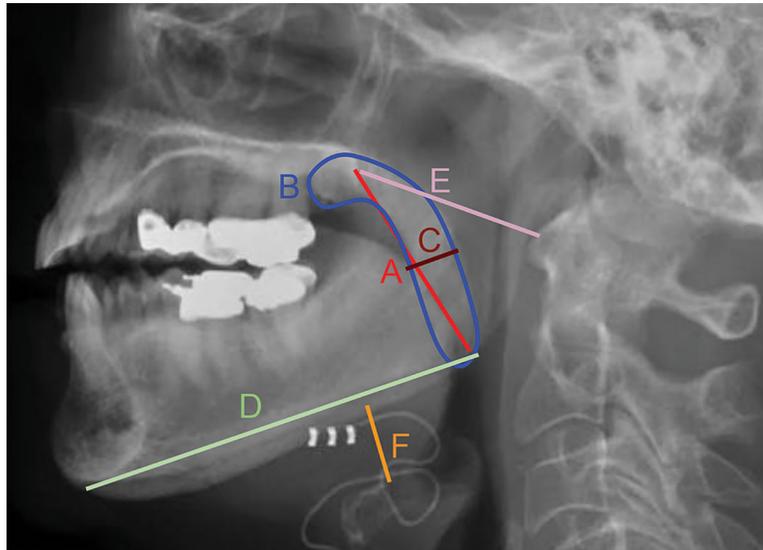


Figure 1. Lateral x-ray of implanted hypoglossal nerve stimulation. Colored lines represent cephalometric measurements. (A-C) Soft palate length, area, and thickness, respectively. (D) Mandibular length. (E) Posterior nasal spine to C1 vertebra distance. (F) Mandibular plane to hyoid distance.

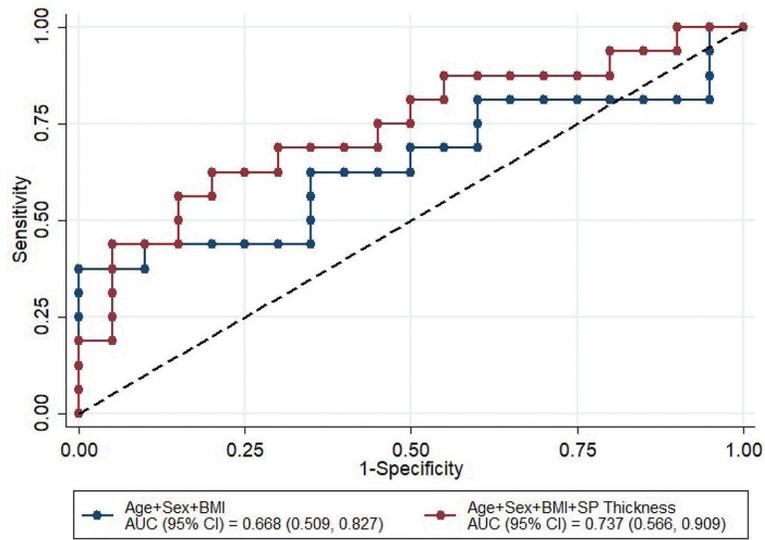


Figure 2. Receiver operating characteristic curve for overall efficacy apnea-hypopnea index based on prediction models using age, sex, and body mass index (BMI) only (blue line) and the 3 covariates plus soft palate thickness (red line). AUC, area under the curve; SP, soft palate.

Table 1.Cohort Characteristics (n = 51).^a

Demographics	Value
Male sex, %	59
White race, %	82
Age, y	62.3 ± 14.5
Body mass index, kg/m ²	28.7 ± 4.1
Non-OSA sleep disorder, %	39
Prior upper airway surgery, %	53
Neurologic disease, %	14
Polysomnogram	
Apnea-hypopnea index, events/h	36.7 ± 17.6
4% oxygen desaturation index, ^b events/h	30.0 ± 19.4

Abbreviation: OSA, obstructive sleep apnea.

^aValues represent mean ± standard deviation, unless otherwise stated.^bn = 45, not reported on all sleep tests.

Table 2.

Comparison of Key Cephalometric Measurements Between HGNS Therapy Responders^a and Nonresponders.

Characteristic	Responders (n = 24), mean ± SD	Nonresponders (n = 27), mean ± SD	P value	Power (difference) ^b
Soft palate thickness, mm (n = 36)	13.4 ± 3.8	16.0 ± 3.4	.045	0.37 (2)
Soft palate length, cm (n = 35)	5.3 ± 1.0	5.2 ± 0.9	.84	0.42 (0.5)
Soft palate area, mm ² (n = 34)	649.4 ± 236.8	720.8 ± 205.8	.37	0.24 (100)
Mandibular plane to hyoid distance, cm, (n = 46)	3.8 ± 1.5	3.5 ± 1.3	.46	0.64 (1)
Mandibular length, cm (n = 39)	9.6 ± 1.2	9.8 ± 1.5	.66	0.61 (1)
Posterior nasal spine to CI vertebra distance, cm (n = 45)	4.2 ± 0.7	4.4 ± 0.7	.47	0.64 (0.5)

Abbreviation: HGNS, hypoglossal nerve stimulation.

^aTherapy response defined as a >50% reduction in apnea-hypopnea index (AHI) with an overall AHI less than 20 events/h.

^bPower to detect a specified difference in the outcome variable.

Table 3.

Area Under the Receiver Operating Characteristic Curve for Clinical Factors and Soft Palate Thickness in Prediction of Response^a to HGNS.

Model	AUC (95% CI)	
	All patients (n = 51)	Prior palatal surgery excluded
Individual measures		
Age	0.641 (0.484–0.799)	0.630 (0.460–0.801)
Male sex	0.630 (0.460–0.801)	0.525 (0.374–0.675)
BMI, kg/m ²	0.638 (0.480–0.796)	0.633 (0.461–0.805)
AHI, events/h	0.562 (0.401–0.724)	0.550 (0.375–0.725)
Soft palate thickness, mm ^b	0.712 (0.531–0.894)	0.754 (0.567–0.941)
Combined measures		
Age, male sex, and BMI	0.668 (0.509–0.827)	0.658 (0.485–0.832)
Age, male sex, BMI, and AHI	0.688 (0.532–0.845)	0.665 (0.492–0.837)
Age, male sex, BMI, and soft palate thickness	0.737 (0.566–0.909)	0.778 (0.592–0.964)

Abbreviations: AHI, apnea-hypopnea index; AUC, area under the curve; BMI, body mass index; HGNS, hypoglossal nerve stimulation.

^aTherapy response defined as a >50% reduction in AHI with an overall AHI less than 20 events/h.

^bSoft palate thickness was measured in 16 therapy responders and 20 nonresponders. In the subgroup excluding patients with prior palatal surgery, soft palate thickness was measured in 13 therapy responders and 18 nonresponders.