The Laryngoscope

Upper-Airway Stimulation Before, After, or Without Uvulopalatopharyngoplasty: A Two-Year Perspective

Armin Steffen, MD; Nils Abrams, MD; Maria V. Suurna, MD; Barbara Wollenberg, MD; Katrin Hasselbacher, MD

Objective: Upper airway stimulation (UAS) is an effective second-line treatment for obstructive sleep apnea (OSA). In certain patients, there is a considerable need for advanced programming, notably with inadequate palatal response to therapy. The aim of the study was to investigate the impact of uvulopalatopharyngoplasty and tonsillectomy (UPPP-TE) on UAS therapy outcomes from a 2-year perspective after implantation.

Methods: This study included all consecutive patients implanted with UAS in which a full set of 1- and 2-year follow-up assessments (M12 and M24) were obtained. Cases were analyzed in three groups: patients with UPPP-TE after (group 1) and before (group 2) UAS, and those without UPPP-TE (group 3).

Results: Therapy success could be achieved in about 80% of the entire cohort. Groups 2 and 3 did not differ significantly with regard to obesity, Apnea-Hypopnea Index, or Oxygen Desaturation Index. With regard to initial sleep endoscopy, there were fewer patients without any obstruction at the palatal and oropharyngeal levels and higher prevalence of lateral obstruction patterns at oropharynx in group 1 in contrast to groups 2 and 3. Groups 2 and 3 showed similar results, although group 2 patients underwent UPPP-TE before UAS implantation.

Conclusion: UPPP-TE should be considered in patients with persistent OSA after UAS implantation if the obstruction is identified at the level of velum and oropharynx. Although this approach has higher response rates and better outcomes can be achieved in patients with UAS, there is no indication for patients to routinely undergo UPPP-TE prior to UAS implantation.

Key Words: Hypoglossal nerve stimulation, upper airway stimulation, PAP failure, PAP intolerance, sleep apnea, UPPP.

Level of Evidence: 4

***Laryngoscope*, 129:514–518, 2019**

INTRODUCTION

Obstructive sleep apnea (OSA) is a widespread disease affecting from 9% to 38% of the adult population, depending on age, in today's society.[1] Daytime sleepiness is the most common symptom, and OSA represents an increased risk factor for cardiovascular disease.[2,3] In this context, positive airway pressure (PAP) is widely accepted as first-line therapy for patients suffering from OSA. Although PAP achieves good therapy results, intolerance caused by mask leak, high-pressure settings, or claustrophobia is a common problem. In recent years, selective upper airway stimulation (UAS) has become an accepted second-line therapy for select OSA patients with PAP intolerance. Breathing effort is detected by an intercostal sensor lead, which sends input to the infraclavicular implanted pulse generator, which then generates a smooth electric impulse via a stimulation cuff lead to the medial branches of the hypoglossal nerve that results in protrusion and stiffening of the tongue.[4] There is a need for better understanding of the submental anatomy combined with intraoperative neuromonitoring, which can guide correct surgical placements to improve surgical outcomes, specifically through complete exclusion of retractor branches innervating styloglossus and hyoglossus muscles.[5] The effectiveness and safety of UAS has been previously demonstrated in large cohorts[6] and in long-term follow-up.[7] Even in cases with higher body mass index (BMI) up to 35 kg/m^2 and expanded Apnea-Hypopnea Index (AHI) range from 15 to 65 per hour, success rates remained comparable to initial phase III trials.[8–10] This appears to be confirmed by preliminary results of a worldwide UAS registry study with no BMI limits.[6] In a cohort of OSA patients looking for PAP treatment alternatives, 20% to 25% had a complete concentric collapse pattern (CCC).[11,12] Findings of this study gained interest because they demonstrated that previous tonsillectomy (TE) reduced prevalence of CCC, which is the major exclusion criterion for UAS, by almost half in a cohort of 210 PAP-intolerant

From the the Department of Otorhinolaryngology, University of Lübeck (A.S., N.A., B.W., K.H.), Lübeck, Germany; and the the Department of Otolaryngology–Head and Neck Surgery, Weill Cornell Medicine (M.V.S.), New York, New York, U.S.A.

Editor's Note: This Manuscript was accepted for publication May 21, 2018.

Presented at the 88th Annual Meeting of the German Society of Otorhinolaryngology, Head and Neck Surgery in Lübeck, Germany, May 11, 2018.

Armin Steffen is a study investigator and received honoraria and travel expenses for invited talks on behalf of Inspire Medical, Inc., outside the submitted work. Maria V. Suurna is an investigator for Inspire Medical, Inc. Katrin Hasselbacher received travel expenses from Inspire Medical, Inc. The authors have no other funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Armin Steffen, MD, Klinik für HNO-Heilkunde, Ratzeburger Allee 160, 23538 Lübeck, Germany. E-mail: armin.steffen@uksh.de.

DOI: 10.1002/lary.27357

patients.[12,13] This was underscored by studies that showed CCC could be changed to non-CCC by expansion uvulopalatopharyngoplasty with tonsillectomy (UPPP-TE), albeit in short-term trials.[14] In contrast to previous publications demonstrating UPPP-TE as a positive outcome predictor,[9] there is a new single-center experience demonstrating that prior airway surgery had no statistically significant effect on postoperative AHI or nadir oxygen desaturation in patients treated with UAS.[15]

Despite careful preoperative patient screening in a small number of cases, UAS therapy success cannot be achieved. One of the possible reasons might be insufficient palatoglossal coupling.[16,17] Other conditions such as the sleep position[18] might negatively impact therapy results despite repeated therapy adjustments, settings optimization during polysomnography, or drug-induced sleep endoscopy (DISE) with activated upper airway stimulation. In cases for which collapse of the soft palate is caused by insufficient palatoglossal coupling, thus hindering success of UAS therapy, UPPP-TE or its variants can improve outcomes. The aim of this study is to investigate whether UPPP in cases with poor response to UAS can improve outcomes based on daytime sleepiness and polysomnographic parameters.

MATERIALS AND METHODS

All consecutive patients who were implanted with a selective UAS system (Inspire Medical Systems, Maple Grove, MN) from November 2012 to September 2015 at the Department for Otorhinolaryngology of the University of Lübeck, Germany, were included in this trial. The patient inclusion criteria for implantation were AHI between 15 and 65 events per hour, central apnea index less than 25% of total AHI, and absence of complete concentric collapse at velopharynx during DISE. Home sleep study (HST) was used to assess OSA severity using the definition of apneas as a complete breathing cessation for at least 10 seconds and hypopneas as breathing restriction by 30%, accompanied by a decrease in oxygen saturation of at least 4%. At month 2 (M2) postimplantation, patients underwent polysomnography (PSG) with UAS system optimization in a sleep laboratory. In case of insufficient treatment response after therapy optimization PSG, advanced titration was performed that involved testing different electrode configurations, impulse setting, prolonged therapy start delays, respiratory sensing optimization, and so forth, followed by HST. If these adjustments did not improve therapy response, the decision was made to proceed with DISE evaluating response to UAS at various settings and voltages. Initial DISE and repeat DISE with and without stimulation were scored using the VOTE classification.[19] In cases with soft palate obstruction that did not resolve sufficiently during UAS stimulation or that required intolerable voltages, UPPP with TE was performed. After 12 and 24 months (M12 and M24), effectiveness of the therapy was evaluated by HST. Outcomes for all groups are reported for subjects treated with UAS for at least 24 months. Cases without M12 or M24 follow-up were excluded. For better comparison, we divided our cohort into three groups:

Group 1: patients who underwent UPPP-TE after implantation of UAS
Group 2: patients who underwent UPPP-TE before implantation of UAS
Group 3: patients without UPPP-TE before or after implantation of UAS

Outcome measurements and differentiation between responders and nonresponders were evaluated using the following criteria:

M12 AHI reduction by at least 50% from baseline and AHI less than 20 per hour
M12 AHI below 15 per hour
M12 AHI below 5 per hour

Informed consent was obtained for each patient. The study was approved as a retrospective chart review by the local ethics committee (AZ 17-300A; Ethikkommission, Universität zu Lübeck, Germany).

Statistical Analysis

Version 22.0 of SPSS (IBM Corp., Armonk, NY) was used. For outcome measures HST, patient-reported outcome at 12 months and 24 months as well as stimulation thresholds were compared with the baseline measurements using a two-sided Wilcoxon test. Descriptive statistics were calculated for demographic variables. Results were reported as mean and standard deviation.

RESULTS

Five patients were lost to follow-up for either the M12, M24, or both. Twenty-five consecutive patients who met the above described criteria were included (Table I). All seven patients in group 1 underwent UPPP-TE after UAS implantation, with an average time between UAS and UPPP-TE of 188 days ranging from 86 to 290 days. In group 2, six patients underwent TE, two underwent UPPP, and two underwent UPPP-TE before UAS. There were eight patients included in group 3 who never had palatal surgery. Apnea-Hypopnea Index in group 1 was higher than in group 2 ($P = 0.02$) and group 3 ($P = 0.01$), whereas the Oxygen Desaturation Index (ODI) was higher in group 1 than group 2 ($P = 0.02$) but similar to group 3 ($P = 0.13$). Body mass index (group 1 vs. 2, $P = 0.15$; 1 vs. 3, $P = 0.13$) and ESS scores were similar (group 1 vs. 2, $P = 0.64$; 1 vs. 3, $P = 0.32$).

Entire cohort and subgroups demonstrated improved OSA burden based on AHI (initial to M12 and to M24, both $P < 0.01$), AI (initial to M12, $P = 0.03$; initial to M24, $P = 0.01$), ODI (initial to M12 and to M24, both $P < 0.01$), and ESS (initial to M12 and to M24, both $P < 0.01$) (Table I) with no significant BMI change. Between implantation and M24, five patients presented with relevant weight loss, two from group 1 and one case each from group 2 and from group 3. One patient in group 2 had relevant weight gain during this period.

The evaluation of DISE data (Table II) revealed several differences between the three groups. In contrast to groups 2 and 3, group 1 had fewer patients without any palatal collapse and more patients with lateral oropharyngeal collapse. Unexpectedly and of particular interest, groups 2 and 3 showed nearly identical results

TABLE I.
Outcome of the Entire UAS Cohort and Subgroups With Regard to UPPP-TE Showing Mean Values and Standard Deviation

		Initial	Re-DISE	Month 12	Month 24
Entire cohort	BMI kg/m^2	29.4 ± 4.9	NA	29.3 ± 4.6	29.0 ± 4.6
n = 25	AHI per hour	32.8 ± 15.4	NA	13.6 ± 17.5	11.2 ± 10.0
mean age = 52.6 years	AI per hour	16.6 ± 17.5	NA	10.6 ± 17.3	6.4 ± 7.9
	ODI per hour	20.1 ± 18.5	NA	10.3 ± 17.3	10.7 ± 13.1
	ESS in points	12.5 ± 4.9	NA	6.6 ± 4.3	5.4 ± 3.9
Group 1	BMI kg/m^2	33.3 ± 3.5	34.1 ± 3.1	32.6 ± 3.5	31.4 ± 3.4
UPPP-TE	AHI per hour	49.4 ± 13.4	35.3 ± 19.6	27.4 ± 29.3	13.1 ± 10.2
after UAS	AI per hour	29.5 ± 23.4	25.7 ± 20.6	22.8 ± 29.3	7.1 ± 3.5
n = 7	ODI per hour	38.3 ± 23.0	30.8 ± 15.4	23.1 ± 29.3	12.3 ± 16.3
mean age = 55.4 years	ESS in points	12.0 ± 4.5	NA	7.1 ± 4.9	6.1 ± 4.1
Group 2	BMI kg/m^2	28.4 ± 5.0	NA	28.6 ± 4.8	28.4 ± 5.0
UPPP-TE	AHI per hour	25.3 ± 10.1	NA	7.6 ± 6.7	10.0 ± 12.3
before UAS	AI per hour	10.2 ± 12.5	NA	4.8 ± 6.1	4.2 ± 9.6
n = 10	ODI per hour	12.4 ± 12.3	NA	4.3 ± 3.6	14.2 ± 15.0
mean age = 48.7 years	ESS in points	10.4 ± 4.4	NA	5.7 ± 4.7	5.4 ± 4.1
Group 3	BMI kg/m^2	27.2 ± 4.1	NA	27.3 ± 4.0	27.4 ± 4.5
UAS without	AHI per hour	27.6 ± 11.8	NA	9.1 ± 5.4	11.0 ± 7.4
UPPP-TE	AI per hour	11.9 ± 9.2	NA	7.3 ± 5.2	8.2 ± 8.5
n = 8	ODI per hour	13.3 ± 7.7	NA	6.6 ± 6.1	4.9 ± 2.9
mean age = 54.9 years	ESS in points	15.5 ± 4.9	NA	7.3 ± 3.5	4.7 ± 3.7

AHI = Apnea-Hypopnea Index; AI = Apnea Index; BMI = body mass index; ESS = Epworth Sleepiness Scale; ODI = Oxygen Desaturation Index; UAS = upper airway stimulation, UPPP-TE = uvulopalatopharyngoplasty-tonsillectomy.

despite the fact that group 2 cases already had tonsillectomy earlier in life. Between all the groups, only small differences could be identified at the tongue base and epiglottis level. Comparing obstruction patterns between DISE and repeated DISE in group 1, there were slight differences. One case showed CCC of the velopharynx in contrast to previously noted complete anteroposterior collapse.

Therapy success was achieved in about 80% of the entire group using Sher criteria. There were obvious differences between the three subgroups, especially group 1 (Table III). In group 1, therapy success improved to 71% and 86% after UPPP-TE at M12 and M24, respectively. Groups 2 and 3 presented both with approximately 90% success rate at M12. At M24, success rates came down to 75% in group 3 and down to 80% in group 2.

DISCUSSION

This study represents the largest cohort of patients treated with UAS for OSA who also underwent palatal surgery, performed after implantation based on therapy response and DISE findings. Beside the long-term follow-up of the previous phase III study (i.e., the STAR trial) with patient-reported results[7,20] or with PSG re-evaluation,[21] data on UAS results with longer than a 12-month follow-up are scarce.

UAS expanded therapy options for OSA patients with PAP intolerance, even with higher AHI up to 50 per hour[8] and 65 per hour,[9,10] the latter even in cases with high BMI up to 35 kg/m^2. Nonetheless, the early report of Vanderveken et al.[13] identified the soft palate area as a critical factor for therapy success. They postulated CCC as an exclusion criterion for UAS. This is generally accepted among UAS surgeons at established implanting centers when selecting patients for UAS therapy. Several studies on DISE cohorts of patients with PAP intolerance found CCC in about 20% to 25%.[11,12,22] The fact that hypoglossal nerve activation enlarges not only the lower pharynx but also the velum region is explained by the palatoglossal coupling.[17] Coupling is thought to be more effective in opening a velum obstruction in a stiffened soft palate resulting from soft palate surgery intervention. In a large prospective multicenter study, Steffen et al.[9] identified a previous UPPP-TE having an effect on therapy response, which is different to other retrospective cohort studies[15] that found no effect of prior airway surgery on AHI reduction or minimal oxygen saturation values. This is highlighted by a large DISE cohort study, which included 210 CPAP-intolerant patients.[12] No difference was seen between the patients with or without prior tonsillectomy with regard to AHI and BMI. Tonsillectomy in the past made a substantial impact on the prevalence of CCC (30%–18%). This phenomenon is supported by a change from CCC to non-CCC collapse pattern after UPPP-TE surgery in PAP-intolerant patients.[14]

Our results add valuable data regarding the role of palatal obstruction in OSA treatment. We analyzed scenarios of soft palate surgery before or after UAS implantation, and without any intervention other than UAS. Together with the DISE data, we could show that cases with obstruction at the velum, and especially in cases

TABLE II.
Distribution of Collapse Patterns Following VOTE Classification, In Percent

		Initial						Re-DISE					
			AP		LL				AP		LL		
		N	C	P	C	P	CC	N	C	P	C	P	CC
Entire	V	40.0	32.0	24.0	0	4.0	0	NA					
cohort	O	68.0	NA	NA	0	32.0	NA						
n = 25	T	0	84.0	16.0	NA	NA	NA						
	E	64.0	20.0	16.0	0	0	NA						
Group 1	V	14.3	71.4	14.3	0	0	0	0	85.7	0	0	0	14.3
UPPP-TE	O	14.3	NA	NA	0	85.7	NA	14.3	NA	NA	14.3	71.4	NA
after UAS	T	0	85.7	14.3	NA	NA	NA	28.6	71.4	0	NA	NA	NA
n = 7	E	28.6	0	42.9	28.6	0	NA	42.9	42.9	14.3	0	0	NA
Group 2	V	50.0	10.0	40.0	0	0	0	NA					
UPPP-TE	O	100.0	NA	NA	0	0	NA						
before UAS	T	0	70.0	30.0	NA	NA	NA						
n = 10	E	90.0	0	10.0	0	0	NA						
Group 3 UAS without UPPP-TE	V	50.0	25.0	12.5	0	12.5	0	NA					
	O	75.0	NA	NA	0	25.0	NA						
n = 8	T	0	100.0	0	NA	NA	NA						
	E	62.5	37.5	0	0	0	NA						

AP = anteroposterior; C = complete; CC = concentric; DISE = drug-induced sleep endoscopy; E = epiglottis level; LL = latero-lateral; N = none; O = oropharynx level; P = partial; Re-DISE = repeated DISE; T = tongue base level; UAS = upper airway stimulation; UPPP-TE = uvulopalatopharyngoplasy with tonsillectomy; V = velum level.

for which obstruction was also present at the oropharynx, tended to have more difficult therapy response path. In situations when other adjustments such as changes in electrode configuration[9,23] do not improve outcomes, UPPP-TE as adjunctive multilevel therapy could help solve the problem with the therapy response. On the other hand, there is a large portion of cases of our UAS cohort, namely group 3, that show impressive improvement using subjective and objective measures without any soft palate surgery.

Based on our data, UPPP-TE should be considered in UAS implanted patients with insufficient results after treatment optimization. Although positive effects of UPPP-TE may vanish over time,[24] our 2-year follow-up supports sustainable OSA treatment in cases of UAS combined with UPPP-TE. Nonetheless, UAS is a long-term therapy that needs a close collaboration between implanting otolaryngology and sleep medicine clinicians.

There are several limitations of our retrospective analysis. Despite the large number of cases with 2-year follow-up, these were the first cases of our center which are subjects to effects of learning curve.[25] Such a learning curve includes not only the implantation technique but also patient selection and DISE evaluation. Despite

TABLE III.
AHI Reduction at Re-DISE: 12-Month and 24-Month Follow-up

		Month 12	Month 24
Entire cohort	AHI reduction by > 50% + AHI < 20/hour	84.0%	80.0%
n = 25	AHI < 15/hour	68.0%	72.0%
	AHI < 5/hour	36.0%	24.0%
Group 1	AHI reduction by > 50% + AHI < 20/hour	71.4%	85.7%
UPPP-TE after UAS	AHI < 15/hour	28.6%	71.4%
n = 7	AHI < 5/hour	0%	0%
Group 2	AHI reduction by > 50% + AHI < 20/hour	90.0%	80.0%
UPPP-TE before UAS	AHI < 15/hour	80.0%	70.0%
n = 10	AHI < 5/hour	40.0%	50.0%
Group 3	AHI reduction by > 50% + AHI < 20/hour	87.5%	75.0%
UAS without UPPP-TE	AHI < 15/hour	87.5%	62.5%
n = 8	AHI < 5/hour	25.0%	12.5%

AHI = Apnea-Hypopnea Index; Re-DISE = repeated drug-induced sleep endoscopy; UAS = upper airway stimulation; UPPP-TE = uvulopalatopharyngoplasty-tonsillectomy.

the generally accepted good retest variability,[19] several slight changes in collapse patterns could be seen (Table II), in addition to relevant identification of the CCC in one of the cases.

No data for repeated DISE were collected in groups 2 and 3 because this could not be done outside of clinical studies for ethical reasons; thus, direct comparison of the effect of UAS on palatal closure in those groups is unavailable. Of the first 30 UAS cases in our center, five cases were not included. No follow-up could be done after insufficient PSG at month 2 for one case. Two other cases underwent UPPP-TE after UAS implantation but were lost to M24 follow-up. One patient with insufficient tongue motion had a stimulation cuff repositioned and was therefore excluded. For one patient, only partial outcome assessment was available.

CONCLUSION

Additional soft palate surgery intervention can improve insufficient response after UAS implantation if the obstruction is identified at the level of velum and oropharynx. Using this approach, high therapy response levels at 2-year follow-up could be achieved with additional treatment. Unless indicated by anatomic findings, due to high therapy response in patients with untreated soft palate, it is not necessary to routinely consider UPPP-TE prior to offering PAP intolerant patients UAS.

Acknowledgments

Author contributions. Armin Steffen and Katrin Hasselbacher: conception and design, data acquisition, data analysis and interpretation, drafting the article, final approval, accountability for all aspects of the work; Nils Abrams: conception and design, data acquisition, data analysis and interpretation, drafting the article, final approval, accountability for all aspects of the work; Maria V. Suurna and Barbara Wollenberg: data analysis and interpretation, drafting the article, final approval, accountability for all aspects of the work.

BIBLIOGRAPHY

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.
2. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005;365:1046–1053.
3. Arzt M, Young T, Finn L, Skatrud JB, Bradley TD. Association of sleep-disordered breathing and the occurrence of stroke. *Am J Respir Crit Care Med* 2005;172:1447–1451.
4. Heiser C, Thaler E, Boon M, Soose RJ, Woodson BT. Updates of operative techniques for upper airway stimulation. *Laryngoscope* 2016;126(suppl 7):S12–S16.
5. Heiser C, Hofauer B, Lozier L, Woodson BT, Stark T. Nerve monitoring-guided selective hypoglossal nerve stimulation in obstructive sleep apnea patients. *Laryngoscope* 2016;126:2852–2858.
6. Boon M, Huntley C, Steffen A, et al. Upper airway stimulation for obstructive sleep apnea: results from the ADHERE registry. *Otolaryngol Neck Surg* 2018:19459981876489.
7. Gillespie MB, Soose RJ, Woodson BT, et al. Upper airway stimulation for obstructive sleep apnea: patient-reported outcomes after 48 months of follow-up. *Otolaryngol Head Neck Surg* 2017;156:765–771.
8. Strollo PJ, Soose RJ, Maurer JT, et al. Upper-airway stimulation for obstructive sleep apnea. *N Engl J Med* 2014;370:139–149.
9. Steffen A, Sommer JU, Hofauer B, Maurer JT, Hasselbacher K, Heiser C. Outcome after one year of upper airway stimulation for obstructive sleep apnea in a multicenter German post-market study. *Laryngoscope* 2018;128:509–515.
10. Heiser C, Maurer JT, Hofauer B, Sommer JU, Seitz A, Steffen A. Outcomes of upper airway stimulation for obstructive sleep apnea in a multicenter German postmarket study. *Otolaryngol Neck Surg* 2017;156: 378–384.
11. Steffen A, Frenzel H, Wollenberg B, Konig IR. Patient selection for upper airway stimulation: is concentric collapse in sleep endoscopy predictable? *Sleep Breath* 2015;19:1373–1376.
12. Hasselbacher K, Bruchhage K-L, Abrams N, Steffen A. About the development of therapy concepts using sleep endoscopy in OSA patients with PAP.
13. Vanderveken OM, Maurer JT, Hohenhorst W, et al. Evaluation of drug-induced sleep endoscopy as a patient selection tool for implanted upper airway stimulation for obstructive sleep apnea. *J Clin Sleep Med* 2013; 9:433–438.
14. Hasselbacher K, Seitz A, Abrams N, Wollenberg B, Steffen A. Complete concentric collapse at the soft palate in sleep endoscopy: what change is possible after UPPP in patients with CPAP failure? *Sleep Breath*. 2018. doi: 10.1007/s11325-018-1657-z.
15. Mahmoud AF, Thaler ER. Upper airway stimulation therapy and prior airway surgery for obstructive sleep apnea. *Laryngoscope* 2018;128:1486–1489.
16. Heiser C, Edenharter G, Bas M, Wirth M, Hofauer B. Palatoglossus coupling in selective upper airway stimulation. *Laryngoscope* 2017;127: E378–E383.
17. Safiruddin F, Vanderveken OM, de Vries N, et al. Effect of upper-airway stimulation for obstructive sleep apnoea on airway dimensions. *Eur Respir J* 2015;45:129–138.
18. Steffen A, Hartmann JT, Konig IR, et al C. Evaluation of body position in upper airway stimulation for obstructive sleep apnea: is continuous voltage sufficient enough? *Sleep Breath*.
19. De Vito A, Carrasco Llatas M, Vanni A, et al. European position paper on drug-induced sedation endoscopy (DISE). *Sleep Breath* 2014;18:453–465.
20. Soose RJ, Woodson BT, Gillespie MB, et al. Upper airway stimulation for obstructive sleep apnea: self-reported outcomes at 24 months. *J Clin Sleep Med* 2016;12:43–48.
21. Woodson BT, Soose RJ, Gillespie MB, et al. Three-year outcomes of cranial nerve stimulation for obstructive sleep apnea. *Otolaryngol Neck Surg* 2016;154:181–188.
22. Ong AA, Murphey AW, Nguyen SA, et al. Efficacy of upper airway stimulation on collapse patterns observed during drug-induced sedation endoscopy. *Otolaryngol Neck Surg*. 2016;154:970–977.
23. Heiser C. Advanced titration to treat a floppy epiglottis in selective upper airway stimulation. *Laryngoscope* 2016;126:S22–S24.
24. Lysdahl M, Haraldsson PO. Uvulopalatopharyngoplasty versus laser uvulopalatoplasty: prospective long-term follow-up of self-reported symptoms. *Acta Otolaryngol* 2002;122:752–757.
25. Thaler ER, Schwab RJ. Single-institution experience and learning curve with upper airway stimulation. *Laryngoscope* 2016;126:S17–S19.