The Laryngoscope

The Effectiveness of Tonsillectomy and Partial Adenoidectomy on Obstructive Sleep Apnea in Cleft Palate Patients

Mosaad Abdel-Aziz, MD

Objectives/Hypothesis: The most common cause of pediatric obstructive sleep apnea (OSA) is adenotonsillar hypertrophy (ATH). In cleft palate patients, however, the obstructive effects of ATH are more severe due to narrow airways. The aim of this study was to assess the effectiveness of tonsillectomy and/or partial adenoidectomy on OSA in cleft palate patients.

Study design: Case series.

Methods: Tonsillectomy and/or partial adenoidectomy was performed in 17 repaired cleft palate patients with tonsillar and/or adenoid hypertrophy and OSA. Apnea/hypopnea (A/H) index and minimum O_2 saturation were measured before and after surgery. In addition, because these patients are vulnerable to speech impairment after pharyngeal surgery, auditory perceptual assessment (APA) and nasometric assessment of speech were performed.

Results: The mean preoperative A/H index was 17.6 $\pm$ 3.9, and the mean preoperative minimum O_2 saturation was 88.7 $\pm$ 1.5%. Both parameters improved postoperatively, to 1.9 $\pm$ 2.3 and 93.7 $\pm$ 1.5% respectively, and the changes were significant ($P < 0.001$). In 12 cases (70.6%), A/H indexes were normalized following surgery. Associated comorbidities such as retrognathia and narrow pharyngeal airways may underlie incomplete recovery in some cases. There were no significant postoperative changes in APA and nasalance scores.

Conclusions: In most cases, tonsillectomy and/or partial adenoidectomy is an effective method for treatment of OSA in repaired cleft palate patients presenting with tonsillar and/or adenoid hypertrophy. However, some cases may need further procedures to relieve airway obstruction due to associated comorbidities.

Key Words: Obstructive sleep apnea, cleft palate, polysomnography, tonsillectomy, adenoidectomy.

Level of Evidence: 4

***Laryngoscope,* 122:2563–2567, 2012**

INTRODUCTION

Obstructive sleep apnea (OSA) is a breathing disorder during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation.[1] The prevalence of OSA in the general pediatric population is approximately 2%–3%, with 3%–12% exhibiting primary snoring. Risk factors for OSA include adenotonsillar hypertrophy (ATH), obesity, craniofacial anomalies, and neuromuscular disorders.[2] Changes in human facial morphology associated with cleft palate result in a small midface and a retruded mandible. Cleft patients may have decreased posterior length and height of the maxilla, increased maxillary width, increased width of the nasal cavity, decreased length of the mandible, and mandibular retrognathia, all of which result in reduced size of the pharyngeal airway.[3] Recently, several reports have documented much higher rates of OSA in cleft palate patients compared to non-cleft patients.[2–5]

The most common cause of pediatric OSA in non-cleft patients is ATH. Moreover, cleft palate patients with ATH are more prone to develop OSA due to narrow airways.[4] However, patients with cleft palate generally do not undergo adenoidectomy unless it is absolutely necessary; when the procedure is necessary, conservative or partial adenoidectomy is performed to avoid the development of velopharyngeal insufficiency (VPI).[3,6,7] VPI has also been reported after tonsillectomy without adenoidectomy; although in these cases the problem is mostly transient, it can be permanent.[8,9] On the other hand, the upper pole of the enlarged tonsil may interpose between the velum and posterior pharyngeal wall, interfering with proper velopharyngeal closure; tonsillectomy may improve velopharyngeal function in such patients.[10–12] Proper management of OSA in cleft palate children is required not only to ensure adequate treatment but also to prevent possible complications, such as systemic hypertension, pulmonary hypertension, failure to thrive, neurocognitive impairment, and behavioral problems.[2] It is especially important to avoid reduced or impaired velopharyngeal closure in these patients after adenotonsillectomy. The aim of this study was to assess the effectiveness of tonsillectomy and/or partial adenoidectomy on OSA in cleft palate patients.

From the Department of Otolaryngology, Faculty of Medicine, Cairo University, Egypt.

Editor's Note: This Manuscript was accepted for publication May 21, 2012.

The study was conducted at the Department of Otolaryngology, Cairo University, Egypt.

The authors have no funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Mosaad Abdel-Aziz, MD, 2 el-salam St., King Faisal, Above El-baraka bank, Giza, Cairo, Egypt.
E-mail: mosabeez@yahoo.com

DOI: 10.1002/lary.23507

TABLE I.
Clinical Characteristics of the Patients.

Patient	Age (years)	Sex	Type of Cleft	Adenoid hypertrophy	Tonsillar size grade	Surgical procedure	A/H index		Minimum O_2 %	
							Pre-	Post-	Pre-	Post-
1	4	Male	BCCLP	Absent	4	T	17	9	88	92
2	3.8	Male	UCCLP	Absent	4	T	20	4	87	93
3	5.5	Female	BCCLP	Present	1	A	13	1	89	96
4	7.3	Male	BCCLP	Absent	4	T	15	0.5	91	94
5	4.2	Female	BCCLP	Present	4	T&A	21	0.5	90	94
6	8	Male	UCCLP	Present	1	A	12	1	90	95
7	5.6	Male	UCCLP	Absent	3	T	10	1	89	95
8	5.1	Female	BCCLP	Absent	4	T	16	3	90	92
9	5.8	Male	UCCLP	Absent	4	T	23	3	89	91
10	6.7	Male	BCCLP	Present	3	T&A	16	0.5	91	96
11	7.7	Male	BCCLP	Absent	3	T	19	0.5	87	94
12	3.6	Female	UCCLP	Present	4	T&A	17	1	86	93
13	5	Female	UCCLP	Present	1	A	21	0.5	88	93
14	6.2	Female	UCCLP	Present	1	A	24	1	88	94
15	10	Male	BCCLP	Present	3	T&A	16	5	89	92
16	4.2	Female	BCCLP	Absent	3	T	22	1	87	93
17	5.6	Male	UCCLP	Present	1	A	18	0.5	88	95

Pre-, preoperative; Post-, postoperative; A/H index, apnea/hypopnea index; BCCLP, bilateral complete cleft lip and palate; UCCLP, unilateral complete cleft lip and palate; T, tonsillectomy; A, partial adenoidectomy; T&A, tonsillectomy and partial adenoidectomy.

MATERIALS AND METHODS

The study was conducted on 17 repaired cleft palate patients who presented with OSA and adenoid and/or tonsillar hypertrophy; nine patients had bilateral complete cleft lip and palate (BCCLP) and eight patients had unilateral complete cleft lip and palate (UCCLP). Two-flap palatoplasty was used for cleft repair in all patients. The study was carried out at the Department of Otolaryngology of Cairo University from February 2008 to January 2011. Table I summarizes the clinical characteristics of the patients. Informed consent was obtained from the parents of all patients, and the principles outlined in the Declaration of Helsinki were followed. In addition, the research protocol was approved by the Ethics Committee of our institute.

Patients underwent extensive pre- and postoperative assessment as follows:

Preoperative Assessment

Medical history was obtained from the parents of the patients, with emphasis on symptoms suggestive of OSA, including nocturnal symptoms (snoring with difficult breathing, observed apnea, restless sleep) and daytime symptoms (daytime somnolence).[13] Only patients with a positive history were included in the study. Physical examination was performed for assessment of associated cranio-facial abnormalities, middle ear effusion, and size of the tonsils. The size of the tonsils was scored as 0–4 based on the percentage of oropharyngeal airway occupied by the two tonsils.[14] Adenoid size and nasopharyngeal airway space were assessed by lateral cephalometric x-ray.[15] Also retrognathia was diagnosed on lateral cephalometry when the gonial angle was more obtuse than normal, the condyle extended distally from the ramus, and ramus to body proportions were deviant because of the greater reduction in body length.[16] Patients with tonsillar hypertrophy grade 3 or 4 were subjected to tonsillectomy, patients with adenoid hypertrophy were subjected to adenoidectomy, and patients with tonsillar hypertrophy grade 3 or 4 and adenoid hypertrophy were subjected to adenotonsillectomy. Patients with a history of adenotonsillectomy and those who underwent secondary corrective surgery (such as pharyngeal flap or sphincter pharyngoplasty) to improve their speech were excluded, because such corrective procedures usually obturate the velopharyngeal port at the expense of the cross-sectional area of the airway.

All patients were assessed by overnight polysomnography (PSG) for at least 6 hours (lab-based study) in a quiet, dark room. Apnea/hypopnea (A/H) index and minimum O_2 saturation were measured, and cases with an A/H index > 1 were included in the study. A/H index was categorized as follows: ≤ 1.0, normal; 1.1–5.0, mild; 5.1–15.0, moderate; and > 15.0, severe.[4] Speech analysis was performed in the phoniatric unit using auditory perceptual assessment (APA) and nasometry. APA included measurement of hypernasality, nasal emission of air, and weak pressure consonants, and parameters were graded on a 5-point scale (0 to 4) in which 0 indicates normal and 4 indicates severe hypernasality. A lower score on this scale indicates less dysfunction. Assessment of nasalance (nasally emitted acoustic energy is compared to the orally emitted energy) was performed using a Nasometer (Kay Elemetrics, model 6200), which provides an acoustic measure of movement of the vibrational energy through the vocal tract. Nasometric data were obtained while the patients read or repeated standardized Arabic nasal and oral sentences.

Surgical Procedure

Under general anesthesia with oral endotracheal intubation and retraction of the soft palate with two rubber catheters, a 70° Hopkins 4-mm nasal endoscope was introduced through the mouth. A camera (Karl Storz GmbH & Co KG; Tuttlingen, Germany) was mounted on the endoscope and the endoscopic view was projected on a monitor. Trans-oral endoscopic partial adenoidectomy was performed,[17] in which the upper half of the adenoid was removed using adenoid curette and St. Claire Thompson forceps, while the lower half was retained to maintain velopharyngeal competence. Coagulation diathermy was

used for hemostasis. After partial adenoidectomy and insertion of nasopharyngeal pack, conventional dissection tonsillectomy with cold instruments was performed. After removal of the tonsils, hemostasis was carried out with minimal use of bipolar diathermy, and the nasopharyngeal pack was removed. The following aspects were given special attention: 1) During adenoidectomy, no tight retraction was exerted on the soft palate to avoid injury to repaired clefts; 2) During tonsillectomy, emphasis was placed on minimal tissue loss, avoidance of injury to muscles of the pillars, and cold dissection during the procedure, with minimal use of diathermy to avoid postoperative fibrosis that can affect velopharyngeal functions.[18] Patients with middle ear effusion underwent myringotomy and insertion of ventilation tubes. Upon awakening, patients were extubated and placed in the lateral position, and then transferred to the postanesthesia care unit (PACU) for observation of respiration and oxygen saturation.

Postoperative Care and Follow-up

All patients received oral antibiotics and paracetamol for 1 week. Patients were discharged from the hospital on the third postoperative day, except in cases that developed postoperative airway compromise; these children stayed in monitored hospital beds until the condition resolved. Children were seen at the end of the first and second postoperative weeks for assessment of wound healing. After 3 months, patients underwent PSG with recording of the same parameters that were employed preoperatively. Patients with an A/H index ≤ 1 were considered to have converted to normal.[4] APA and nasometric assessment were also performed using the same parameters employed preoperatively.

Statistical Methods

Data were coded and summarized using Statistical Package for Social Sciences version 17.0 for Windows. Quantitative variables are presented as mean $\pm$ standard deviation, and categorical data as frequency and percentage. Comparison between unilateral and bilateral cleft cases regarding pre- and postoperative results was done using an independent sample *t* test. The paired two-sample *t* test was used to compare pre- and postoperative data in the same group. $P < 0.05$ was considered statistically significant.

RESULTS

The study examined 17 repaired cleft palate patients (10 boys and seven girls) diagnosed to have OSA with adenoid and/or tonsillar hypertrophy, with mean age at surgery of 5.8 years. The parents of 11 patients stated that their children complained of snoring and obstructive sleep breathing after repair of their clefts while the condition had been observed in the other six patients before palatoplasty. Preoperative examination showed that none of the patients was syndromic, although retrognathia was detected in four patients who had been diagnosed with Pierre Robin Sequence. Seven patients had tonsillar hypertrophy grade 4 in which the tonsils impinged on more than 75% of the airway, five patients had grade 3 hypertrophy in which the tonsils impinged on 50%–75% of the airway, and five patients had grade 1 in which the tonsils impinged on less than 25% of the airway.

Lateral cephalometric x-ray showed a narrow pharyngeal airway in all cases, with remarkable nasopharyngeal narrowing in one patient. Adenoid hypertrophy was detected in nine cases, while a tonsillar shadow appeared to occupy most of the oropharyngeal airway in 12 cases.

Operative decision was taken according to the findings seen in every individual case during the preoperative assessment, however, no intraoperative unexpected pathology was found. Five cases underwent partial adenoidectomy, eight cases underwent tonsillectomy, and four cases underwent both procedures. Middle ear effusion was detected bilaterally in six cases that underwent myringotomy and insertion of ventilation tubes.

Four cases that had retrognathia developed postoperative airway obstruction caused by edema of the tongue base; these cases showed oxygen desaturation of less than 90%. Following oxygen supplementation via face mask and hydrocortisone treatment (3 mg/kg), oxygen saturation improved and re-intubation was not required. Difficult breathing was eliminated after 24 hours; however, these four patients were discharged from the hospital on the fourth postoperative day. No patients developed postoperative hemorrhage or infection.

Preoperative polysomnographic data showed OSA in all cases. The A/H index was moderate in four patients and severe in 13, with a mean A/H index of 17.6 ± 3.9, and a minimum O_2 saturation of $88.7 \pm 1.5\%$. Postoperatively, A/H index was normalized in 12 cases, improved by two categories in four patients, and improved by one category in one patient, with a postoperative mean A/H index of 1.9 ± 2.3 and a minimum O_2 saturation of $93.7 \pm 1.5\%$ (Table I). Comparison between the preoperative and postoperative A/H index showed significant improvement, with $P < 0.001$. In addition, comparison between the preoperative and postoperative minimum O_2 saturation showed significant improvement, with $P < 0.001$.

Improved ($n = 12$) and partially improved ($n = 5$) patients did not differ in age; mean ages were 5.15 years and 5.74 years respectively. The groups did not differ in preoperative severity of OSA ($p = 0.625$); mean A/H index for improved cases was 17.3 ± 4.3, and for partially improved cases 18.4 ± 3. Among partially improved patients, retrognathia was detected in four, and high arched palate with narrow nasopharyngeal airway (radiographic finding) in one. Patients with retrognathia were subjected to tongue base reduction using radiofrequency coblation, and the case of narrow nasopharyngeal airway was observed. Radiofrequency coblation was performed under general anesthesia, a protective thermal sheath probe (XP Coblation; Arthro-Care ENT, Austin,Texas) was used at a setting of 600 joules, the probe was initially inserted at the apex of the circumvallate papillae and a subsequent lesion was created 1-cm anterior to the first treatment site.

The difference between cases with BCCLP and cases with UCCLP regarding pre- and postoperative severity of A/H index was not significant, with $P = 0.651$ and $P = 0.471$, respectively. However, there was significant postoperative improvement in each individual group, with $P < 0.001$.

Speech was not adversely affected by surgery. The mean preoperative baseline of APA was 5.43 ± 1.12, while the postoperative value was reduced to 4.32 ± 1.45. The difference between preoperative and postoperative scores was not significant, with $P > 0.05$. Moreover, nasalance was not worsened after surgery. The preoperative scores were 33 ± 2.5 for the nasal sentences and 13 ± 2.1 for the oral sentences, while postoperative scores declined to 31 ± 3.2 for the nasal sentences and 12.4 ± 2.2 for the oral sentences. The changes were not statistically significant.

DISCUSSION

Dysfunction of the palatal muscles controlling the soft palate in conjunction with structural abnormalities of the maxilla and mandible producing a small nasopharyngeal airway underlie the high risk for sleep-related breathing disorders in children with a cleft palate. The risk of OSA is estimated to be between 22% and 65% in these children. The risk is increased by surgical procedures to correct the structural abnormalities, which further reduce the cross-sectional area of the airway.[2,3] Sometimes tonsils and adenoids appear enlarged not due to enlargement of the lymphoid tissues, but rather due to narrow bone structures that many children with OSA present. Adenotonsillectomy may be performed because elimination of the soft tissues will allow better breathing, but this simple surgery will not resolve other anatomical problems that may be present in children with OSA.[19] However, adenotonsillectomy is the most common surgical treatment for pediatric OSA in the non-cleft population.[4,20]

The adenoid lies in the posterior pharyngeal wall and may act as a pad against the palate facilitating velopharyngeal closure. Thus it can compensate for a short or a poorly mobile palate. Following adenoidectomy, this mechanism of compensation is eliminated and VPI can result. For this reason, adenoidectomy is contraindicated in patients with cleft palate unless it is highly needed.[3,6,7] On the other hand, tonsillar hypertrophy may lead to VPI because the tonsil may have a significant upward extension with its upper pole impinging between the palate and posterior pharyngeal wall, and in such cases tonsillectomy has been reported to relieve symptoms.[10–12] Auditory perceptual assessment and nasometric assessment showed no worsening effects of surgery on the speech of our patients. In fact some improvement (not statistically significant) was achieved that may be attributed to removal of hypertrophied tonsils.

We treated OSA of 17 repaired cleft palate patients with tonsillectomy and/or partial adenoidectomy. Cold dissection was used for removal of tonsils with minimal usage of diathermy to avoid postoperative scarring of the palate. Adenoidectomy was performed using a transoral endoscopic approach, as direct endoscopic vision could facilitate precise removal of the upper obstructive adenoidal tissues and clearance of choanae. We achieved significant improvement of the A/H index, with complete relief of OSA in 12 cases (70.6%). The condition of five patients (29.4%) was partially improved: four patients by two categories and one patient by one category. Persistence of OSA after adenotonsillectomy in a few cleft palate patients may be attributed to the presence of associated comorbidities such as retrognathia and narrow pharyngeal airway. For patients with Robin sequence, some authors recommended tongue lip adhesion to relieve airway obstruction, reserving the more aggressive mandibular distraction osteogenesis to nonresponding cases.[3,21] We used tongue base reduction to widen the airway for those cases, with distraction reserved for patients with persistent symptoms. Wootten and Shott[22] have achieved good results using radiofrequency coblation combined with genioglossus advancement in treatment of pediatric patients with OSA refractory to adenotonsillectomy. Our study showed no significant effect of the type of palatal cleft on occurrence of the disease.

Although many studies have documented the effectiveness of adenotonsillectomy in treatment of OSA in the general pediatric population,[19,20] little is known about the effectiveness of the procedure in eliminating the disease in cleft palate patients. Rose et al.[23] found significant narrowing of the anterior-posterior dimension of the pharynx in patients with cleft palate compared to non-cleft controls, although there were no significant differences in the size of the tonsils and adenoids between the groups. Antony and Sloan[24] found airway obstruction in 5.7% of cleft palate patients following palatoplasty, and reported that the risk is increased in patients with Pierre Robin Sequence or additional congenital anomalies. Indeed, Liao et al.[25] found no OSA in their cleft palate patients prior to repair; however, all cases demonstrated OSA during the early postoperative period. The condition resolved in 80% of their patients within 3 months, while 10% experienced persistent OSA after 6 months.

Muntz et al.[4] studied 24 cleft palate patients with OSA who underwent tonsillectomy and/or adenoidectomy without PSG. They found subjective improvement in all patients, but recommended a post-treatment PSG since some of the children studied did not achieve complete resolution after surgery due to the multifactorial causes of the obstruction. Even in a general pediatric population, Guilleminault et al.[19] found persistent OSA following adenotonsillectomy in 46.2% of patients, as measured with PSG, although parents reported improvement in 92.6% of their children.

Our study highlights the need for PSG monitoring of cleft palate patients presenting with OSA, since the disease persisted in five cases (29.4%) despite successful adenotonsillectomy. Brietzke and Gallagher[20] advised the use of postoperative PSG especially in children with a high probability of residual OSA, such as patients with craniofacial disorders. Although it is an expensive technique and may not be easily accessible, the authors have opted for use of PSG in uncomplicated pediatric patients as a routine postoperative screen for residual OSA.

Our study has some limitations. As a case series, it constitutes a small sample size. Prospective multi-center

studies are needed to adequately assess alternative non-adenotonsillectomy procedures to improve the airway, and also to assess the effects of palatal cleft types and methods of repair on the occurrence of OSA.

CONCLUSION

In most cases, tonsillectomy and/or partial adenoidectomy is an effective method for treatment of OSA in repaired cleft palate patients presenting with tonsillar and/or adenoid hypertrophy. However, some cases may need further procedures to relieve airway obstruction due to associated comorbidities.

Acknowledgments

We are grateful to the staff of the Phoniatric Unit in our department who carried out the speech evaluation.

BIBLIOGRAPHY

1. Section on Pediatric Pulmonology, Subcommittee on obstructive sleep apnea syndrome, American Academy of Pediatrics. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2002;109:704–712.
2. Robison JG, Otteson TD. Increased prevalence of obstructive sleep apnea in patients with cleft palate. *Arch Otolaryngol Head Neck Surg* 2011; 137:269–274.
3. MacLean JE, Hayward P, Fitzgerald DA, Waters K. Cleft lip and/or palate and breathing during sleep. *Sleep Med Rev* 2009;13:345–354.
4. Muntz H, Wilson M, Park A, Smith M, Grimmer JF. Sleep disordered breathing and obstructive sleep apnea in the cleft population. *Laryngoscope* 2008;118:348–353.
5. MacLean JE, Waters K, Fitzsimons D, Hayward P, Fitzgerald DA. Screening for obstructive sleep apnea in preschool children with cleft palate. *Cleft Palate Craniofac J* 2009;46:117–123.
6. Maryn Y, Van Lierde K, De Bodt M, Van Cauwenberge P. The effects of adenoidectomy and tonsillectomy on speech and nasal resonance. *Folia Phoniatr Logop* 2004;56:182–191.
7. Abdel-Aziz M, Dewidar H, El-Hoshy H, Aziz AA. Treatment of persistent post-adenoidectomy velopharyngeal insufficiency by sphincter pharyngoplasty. *Int J Pediatr Otorhinolaryngol* 2009;73:1329–1333.
8. Haapanen ML, Ignatius J, Rihkanen H, Ertama L. Velopharyngeal insufficiency following palatine tonsillectomy. *Eur Arch Otorhinolaryngol* 1994;251:186–189.
9. Wachtel JM, Kuehn DP, Weiss KS. Hypernasality after tonsillectomy without adenoidectomy in an adult. *Otolaryngol–Head Neck Surg* 2000;122: 112–113.
10. Shprintzen RJ, Sher AE, Croft CB. Hypernasal speech caused by tonsillar hypertrophy. *Int J Pediatr Otorhinolaryngol* 1987;14:45–56.
11. Hu TL, Yun C, Wang R, Chen PKT, Lo LJ. Management of velopharyngeal insufficiency in the presence of enlarged tonsils: comparing a one-stage versus two-stage treatment result. *J Plast Reconstr Aesthet Surg* 2008; 61:883–888.
12. Abdel-Aziz M. Hypertrophied tonsils impair velopharyngeal function after palatoplasty. *Laryngoscope* 2012;122:528–532.
13. Muzumdar H, Arens R. Diagnostic issues in pediatric obstructive sleep apnea. *Proc Am Thorac Soc* 2008;15:263–273.
14. Brodsky L. Modern assessment of tonsils and adenoids. *Pediatr Clin North Am* 1989;36:1551–1569.
15. Fujioka M, Young LW, Girdany BR. Radiographic evaluation of adenoidal size in children: adenoidal-nasopharyngeal ratio. *AJR Am J Roentgenol* 1979;133:401–404.
16. Kozak FK, Ospina JC. Characteristics of normal and abnormal postnatal craniofacial growth and development. In: Flint PW, Haughey BH, Lund VJ, et al., eds. *Cummings Otolaryngology Head and Neck Surgery.* 5th ed. Philadelphia, PA: Elsevier Mosby; 2010:2612–2637.
17. El-Badrawy A, Abdel-Aziz M. Transoral endoscopic adenoidectomy. *Int J Otolaryngol* 2009; doi: 10.1155/2009/949315.
18. McKerrow WS, Clarke R. Tonsillectomy. In: Gleeson M, Browning GG, Burton MJ, Clarke R, Hibbert J, Jones NS, et al., eds. *Scott-Brown's Otorhinolaryngology, Head and Neck Surgery.* 7th ed. London: Oxford University Press; 2008:1229–1241.
19. Guilleminault C, Yu-shu H, Glamann C, Li K, Chan A. Adenotonsillectomy and obstructive sleep apnea in children: a prospective survey. *Otolaryngol–Head Neck Surg* 2007;136:169–175.
20. Brietzke SE, Gallagher D. The effectiveness of tonsillectomy and adenoidectomy in the treatment of pediatric obstructive sleep apnea/hypopnea syndrome: a meta-analysis. *Otolaryngol–Head Neck Surg* 2006;134: 979–984.
21. Schaefer RB, Gosain AK. Airway Management in patients with isolated Pierre Robin sequence during the first year of life. *J Craniofac Surg* 2003;14:462–467.
22. Wootten CT, Shott SR. Evolving therapies to treat retroglossal and base of tongue obstruction in pediatric obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 2010;136:983–987.
23. Rose E, Thissen U, Otten J, Jonas I. Cephalometric assessment of the posterior airway space in patients with cleft palate after palatoplasty. *Cleft Palate Craniofac J* 2003;40:498–503.
24. Antony AK, Sloan GM. Airway obstruction following palatoplasty: analysis of 247 consecutive operations. *Cleft Palate Craniofacial J* 2002;39: 145–148.
25. Liao YF, Yun C, Huang CS, et al. Longitudinal follow-up of obstructive sleep apnea following Furlow palatoplasty in children with cleft palate: a preliminary report. *Cleft Palate Craniofac J* 2003;40:269–273.