Body Position and Obstructive Sleep Apnea in Children

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Study Objectives: In adults, sleep apnea is worse when the patient is in the supine position. However, the relationship between sleep position and obstructive apnea in children is unknown. The objective of this study was to evaluate the relationship between obstructive apnea and body position during sleep in children.

Design: Retrospective analysis of the relationship between body position and obstructive apnea in obese and non-obese children.

Setting: Tertiary care pediatric sleep center.

Patients: Otherwise healthy children, aged 1-10 years, undergoing polysomnography for suspected obstructive sleep apnea syndrome. Obese and non-obese children were evaluated separately.

Interventions: Retrospective review of the relationship between sleep position and obstructive apnea during polysomnography.

Measurements and Results: Eighty polysomnograms from 56 non-obese and 24 obese children were analyzed. Body position was determined by a sensor during polysomnography, and confirmed by review of videotapes. Children had a lower obstructive apnea hypopnea index when supine vs. prone, and shorter apneas when supine then when on their side. There was no difference in apnea duration between the supine and prone positions. Obese and non-obese children showed similar positional changes.

Conclusions: Children with obstructive sleep apnea, in contrast to adults, breathe best when in the supine position.

Key words: Sleep-disordered breathing; supine; prone; upper-airway obstruction; posture

INTRODUCTION

THE OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS) IS COMMON DURING CHILDHOOD, OCCURRING IN APPROXIMATELY 2% OF PRESCHOOL CHILDREN.[1] It can result in significant morbidity.[2] Nevertheless, the factors affecting the severity of OSAS in children have not been thoroughly studied.

In adults, snoring and obstructive apnea are well known to be worse in the supine position.[3-5] In fact, mild OSAS is often treated with positional therapy, and techniques such as attaching tennis balls to the back of pajamas have been advocated to prevent patients from lying supine.[3] The effect of position on obstructive apnea in children older than infancy has not been studied. However, it has been our clinical impression that parents of children with OSAS do not report increased snoring or apnea when their child sleeps in the supine position. We therefore hypothesized that sleep position does not affect the severity of upper airway obstruction in children with uncomplicated OSAS.

In most children, OSAS is associated with adenotonsillar hypertrophy. These children are usually of normal weight, or may even have failure to thrive.[6] The subgroup of obese children with OSAS may have a similar pathophysiology to adults with OSAS, most of whom are obese. We therefore hypothesized that the effect of position on sleep-disordered breathing would differ between obese and non-obese children.

Consequently, we retrospectively analyzed the relationship between sleep position and obstructive apnea in obese and non-obese children with OSAS.

METHODS

Study Group

We retrospectively analyzed consecutive overnight polysomnograms with an apnea hypopnea index ≥1 performed at the Johns Hopkins Pediatric Sleep Center. Studies from otherwise-healthy, prepubertal children aged 1-10 years, referred for evaluation of suspected OSAS, were evaluated. Ten years was used as the higher age cut-off as Tanner pubertal ratings were not available for this retrospective review. Infants were excluded as they would be expected to have anatomic and physiologic differences compared to older children. All children had OSAS related to adenotonsillar hypertrophy and/or obesity. Children with craniofacial anomalies, neuromuscular disease, a history of upper airway surgery or any major medical illnesses other than OSAS were excluded. Subject identifiers were not maintained.

Patients were then separated into obese and non-obese subgroups. Growth percentiles were obtained using standard growth charts (National Center for Health Statistics, adapted by Ross Laboratories). Children were defined as obese if their weight was greater than 120% of their ideal weight for height.[7]

Polysomnography

Polysomnography was performed on a Respironics Alice System (Pittsburgh, PA) and consisted of electroencephalogram

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Table 1—Population characteristics and polysomnography results

	NON-OBESE	OBESE
N	56	24
Age (years)	4±2	7±2[a]
Male, N (%)	33 (59%)	8 (33.4%)[a]
% Ideal Body Weight	99±12	179±5[b]
Body Mass Index (kg/m2)	16±2	27±6[b]
Caucasian, N (%)	19 (34%)	2 (8%)[a]
African American, N (%)	35 (63%)	22 (92%)[a]
Asian, N (%)	2 (4%)	0 (0)
TRT (minutes)	459±71	461±48
TST (minutes)	394+48	400+49
Sleep Efficiency (%)	83±14	87±7
Arousal Index (N/hr)	11±6	11±5
Stage 1 (%TST)	4±3	4±3
Stage 2 (%TST)	46±8	48±7
Slow-Wave Sleep (%TST)	29±9	30±6
REM (%TST)	22±6	19±5[a]
OAI (N/hr)	7±8	5±6
OAHI (N/hr)		
Mean±SD	9±10	8±9
Median (interquartile range)	5 (2,12)	4 (2,13)
SaO_2 nadir (%)	87±10	87±9
Peak $P_{ET}CO_2$ (mm Hg)	52±4	52±5
Duration $P_{ET}CO_2$>50 mm Hg (%TST)	11±23	26±36

All data displayed as mean±SD unless otherwise specified.
([a])p<0.05 ([b])p<0.001
TRT, total recording time; TST, total sleep time; REM, rapid eye movement; OAI, obstructive apnea index; OAHI, obstructive apnea hypopnea index; SaO_2, arterial oxygen saturation; $P_{ET}CO_2$, end-tidal PCO_2.

(C3/A2, C4/A1, O1/A2), electrooculograms (right and left), submental and tibial electromyograms, arterial oxygen saturation by pulse oximetry (Nellcor N-1000, Van Nuys, CA), oximeter pulse waveform, end-tidal carbon dioxide tension (Nellcor N-1000; Van Nuys, CA), oronasal airflow (three-pronged thermistor), thoracic and abdominal wall motion (piezoelectric transducers), electrocardiography, and body position sensor. Children were also monitored and recorded on videotape, using an infrared video camera, and were continuously observed by a polysomnography technician. Children accompanied by a parent arrived in the laboratory at 20:00, and studies were terminated at 5:00.

Assessment of Body Position

Patients were allowed to spontaneously assume body positions. They were categorized as being in the prone, supine, or side position based on the position of their torso. Head and neck position was not considered. Body position was identified primarily by the Alice position sensor, situated on the thoracic belt. This was double-checked by reviewing the technician study notes. All technicians routinely filled out a flow sheet in which body position was entered in a dedicated column every hour. In addition, for the first ten patients, body position was confirmed by review of videotapes. In all cases, the videotape agreed with the body sensor.

The Alice System body position sensor utilizes a mercury tilt and vibration switch to determine the position of the patient. The switch design consists of 12 electrically isolated contacts that are spaced thirty degrees apart in a circle around the periphery of the switch. A ball of mercury is in the center of this circle of contacts. As the switch is rotated, the mercury ball will make contact with one or more of the 12 contacts, thereby indicating the body position.

Data Analysis

All polysomnograms were scored by a registered sleep technologist and subsequently reviewed by a physician experienced in pediatric sleep medicine. All data were analyzed by a single investigator (LBP). Standard criteria were used for scoring sleep architecture.[8,9] Respiratory events were scored using accepted pediatric standards.[10] As children have a higher respiratory frequency than adults, and frequently desaturate even with short apneas, all obstructive apneas greater than or equal to two breaths duration were counted.[11] Hypopneas were defined as a qualitative reduction in thermistor airflow ≥50% in the presence of paradoxical respiratory efforts, associated with desaturation ≥3% and/or arousal.[12] The obstructive apnea hypopnea index (OAHI) was defined as the number of hypopneas and obstructive and mixed apneas per hour of total sleep time. For the purpose of this study, any polysomnogram with at least one obstructive event per hour was evaluated. Central apneas were not analyzed due to the very small number of central apneas present.

Statistical Methods

Statistical analysis was performed using STATA version 6.0.[13] Histograms and box plots were created for outcome measures to show the data spread and help identify outlying observations. Categorical patient demographic information was compared between obese and non-obese patients using the chi-squared goodness-of-fit test. Two-sided t-tests for independent samples were performed when comparing the means of normally distributed continuous variables for the obese and non-obese groups, while the Wilcoxon rank-sum test was performed for comparing the distributions of non-normally distributed continuous variables between these two groups. Because sleep and apnea variables were recorded for each child in the three positions, we expected that outcome measures would be correlated within the individual patient. In order to take this correlation into account, regression analyses of apnea on body position were performed using generalized estimating equations (GEE method).[14] Since apnea events consist of count data, the regression of apnea on position was modeled by using the Poisson distribution with an offset for time in each position. Because the data were over-dispersed, the Huber-White robust estimator of variance was used to obtain proper estimates of the standard errors for the regression coefficients.[15] In the regression analysis, an indicator variable for position was placed in the model, with the supine position being the base of comparison. The STATA procedure lincom, which calculates point estimates and confidence intervals for linear combinations of regression coefficients, was used to allow us to compare relative rates of apnea between the side and prone positions.[15] We also controlled for age and obesity in this analysis. Some patients did not have any prone sleep. Therefore, an indicator variable of 1 (prone sleep present) vs. 0 (no prone sleep occurred) was used to analyze whether apnea in other positions differed between those patients with some prone sleep versus

Table 2—Duration of sleep in each position

	Supine TST (minutes)	Prone TST (minutes)	Side TST (minutes)
All patients	81 (34,210)	10 (0,51)	252 (162,318)[a]
Non-obese	93 (34,219)	9 (0,37)	252 (162,318)
Obese	68 (37,165)	21 (0,63)	255 (185,305)
	REM time (%TST)	**REM time (%TST)**	**REM time (%TST)**
All patients	12 (1,29)	0 (0,18)	55 (37,72)[a]
Non-obese	11 (0,28)	0 (0,19)	57 (39,74)
Obese	14 (7,36)	1 (0,11)	47 (32,62)[b]

All data displayed as median (interquartile range). TST, total sleep time.
([a])p< 0.0001 for supine vs. prone, prone vs. side and supine vs. side. There was no significant difference in the time spent in each position between non-obese and obese patients during total sleep.
([b])p=0.023 for REM time in obese vs. non-obese patients.

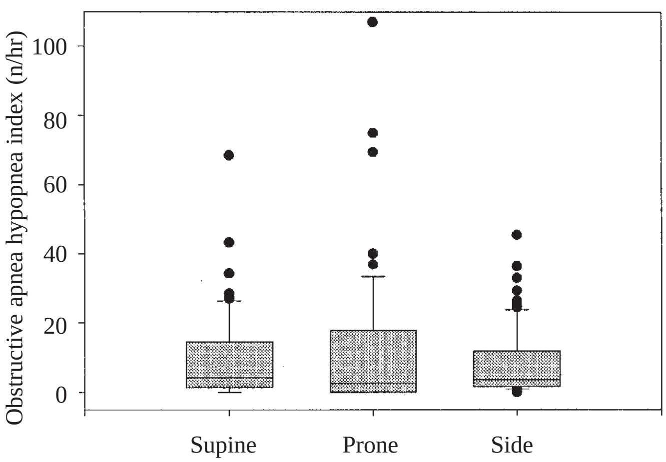

Figure 1—A box plot of the obstructive apnea hypopnea index (OAHI) for all patients in the supine, prone and side positions is shown. The box represents the 25th percentile, median and 75th percentile. The whiskers represent the 10th and 90th percentiles. Outliers are represented by circles. An extreme outlier with an OAHI of 200/hr (supine) is not shown. The OAHI was increased in the prone vs the supine position (p=0.031).

those with no prone sleep. Data that were normally distributed are shown as mean±standard deviation; skewed data are shown as median and interquartile range.

RESULTS

Study Group

Studies from 80 children (56 non-obese and 24 obese) were analyzed. Patient characteristics are shown in Table 1. The obese group was significantly older and more likely to be female and African American than the non-obese group. However, sleep characteristics and the degree of sleep-disordered breathing were similar between the two groups, except for a slight decrease in REM sleep in the obese population.

Total Sleep Time and Position

There was no difference in total sleep time between obese and non-obese patients (Table 1). Children slept predominantly in the side position, followed by the supine and prone positions (Table 2; p<0.0001 for supine vs. prone, prone vs. side and supine vs. side). There was no significant difference in the time spent in each position between non-obese and obese patients.

Obstructive Apnea Index and Position

The OAHI did not differ significantly between the two groups (Table 1). Although it did not quite reach statistical significance, there was a tendency for obese patients to have a higher OAHI during supine sleep than non-obese patients (14 [2,20] vs. 4 [1,9] /hr, p=0.053); no difference was observed between the two groups in the other positions. For the group as a whole, the OAHI in different positions is shown in Figure 1. Table 3 shows the total apnea number and sleep times in different positions used for calculating the generalized estimating equations. For the group as a whole, the relative risk of apnea in the prone position was 1.68 (95% C.I. 1.05, 2.69; p=0.031) compared to the supine position. There was no significant difference in OAHI between the other positions. These results were similar when the model was adjusted for those patients who spent any time prone vs. those who had no prone time. Similar results were noted for the obese subgroup, and when age was added into the model.

Duration of Obstructive Apnea and Position

There was no significant difference in the mean apnea/hypopnea duration between non-obese and obese patients (10 [9,13] vs. 10 [8,12] seconds, respectively). Mean duration did not differ significantly between the two groups in any particular position. For the group as a whole, the mean apnea duration was 12 (10,14) seconds in the prone position, 11 (10,14) seconds in the side position, and 11 (9,13) seconds in the supine position. In order to determine whether significant differences existed between different positions after controlling for obesity, regression analysis was performed. This showed a significant increase in apnea duration in the side vs. the supine position (p=0.034). There was no significant difference in duration of apnea between the prone and supine or prone and side positions.

Table 3—Total number of apneas and sleep time in different body positions

	ENTIRE NIGHT			REM SLEEP		
	Apneas (N)	TST (min)	OAHI (N/hr)	Apneas (N)	TST (min)	OAHI (N/hr)
		All Patients			All Patients	
Supine	1102	9867	7	650	1600	24
Prone	490	2484	12	269	759	21
Side	2714	19501	8	1667	4352	23
		Non-obese			Non-obese	
Supine	684	7125	6	427	1110	23
Prone	314	1537	12	199	577	21
Side	2012	13595	9	1273	3227	24
		Obese			Obese	
Supine	418	2742	9	223	490	27
Prone	176	947	11	70	183	23
Side	702	5906	7	394	1125	21

TST, total sleep time; REM, rapid eye movement; OAI, obstructive apnea index; OAHI, obstructive apnea hypopnea index

Effect of REM Sleep

The effect of REM sleep was analyzed separately. Obese patients had slightly less REM sleep (Table 1). However, there was no significant difference between obese and non-obese children in the REM time in each position (Table 2). As with total sleep time, during REM the children slept mainly on their side, followed by the supine and prone positions (Table 2). Analyses were therefore limited by the short amount of time spent in the non-side positions. Two percent of children had no REM sleep on their side, 23% had no REM sleep supine and 55% had no REM sleep prone. The amount of REM sleep differed significantly between all three positions controlling for obesity ($p=0.001$ for prone vs. supine; $p<0.001$ for side vs supine and $p<0.001$ for side vs prone), and between obese and non-obese groups controlling for position ($p=0.023$).

The OAHI was higher during REM sleep than over the entire sleep period (14 [6,34] vs 4 [2,12]hr, $p<0.001$), consistent with previous reports.[16] There was no significant difference in REM OAHI between obese and non-obese groups, and no significant effect of body position on REM apnea index.

There was no significant difference in the mean REM apnea duration between non-obese and obese patients (17 [12,20] vs. 17 [11,21] seconds, respectively). Mean duration did not differ between obese and non-obese children in each position. Regression analysis showed that REM apnea duration was significantly longer in the side than the supine position ($p=0.022$) after controlling for obesity. No significant differences were found between the side and prone position, or between the obese and non-obese groups.

DISCUSSION

The main finding of this study is that children with OSAS breathed best when supine. Children had fewer obstructive apneas when they were supine compared to prone, and the apneas were shorter in the supine position. Thus, the pattern of obstructive apnea in children is very different from that of adults, who have consistently been shown to have less apnea when in the prone position. Contrary to expectations, obese children had a similar pattern to non-obese children. Although significant positional differences in OAHI were not noted during REM sleep (possibly due to the limited amount of time spent in the non-side position during REM), apneas were shorter in the supine position during REM.

In adults, obstructive apneas are more frequent and longer when patients sleep in the supine position compared to the side position (most studies in adults did not evaluate the prone position due to the short duration of time spent in that position).[3-5] This clinical finding has been corroborated by physiologic studies showing a higher critical closing pressure (Pcrit) in the supine compared to the lateral position.[17] In contrast, the current study shows that children obstruct least when supine. To the best of our knowledge, there are no previous studies evaluating the effect of position on obstructive apnea in young children. However, data on infants support our findings in older children. Although sleep position in infants has not been shown to significantly affect the rate or duration of central or obstructive apneas,[18,19] preliminary data suggest that the infant upper airway is more collapsible when the infant is prone.[20] This may be one of the factors accounting for the increased risk of sudden infant death syndrome (SIDS) in the prone position.[21,22]

In this study, the children with OSAS were most likely to sleep on their side, followed by supine and prone. Thus, their sleep position preferences were similar to that of normal children.[23]

The mechanism for the positional change in obstructive apnea is unclear. Although awake adults with OSAS have narrowing of the upper airway when in the supine position compared to being erect,[24] upper airway dimensions are similar in the supine and lateral recumbent positions.[25] Furthermore, studies during wakefulness may not accurately reflect changes that occur during sleep. In adults, the supine position can lead to prolapse of the mandible, along with the base of the tongue, leading to upper airway obstruction.[26] In children with OSAS, obstruction usually occurs at the levels of the adenoids or soft palate, rather than the tongue;[27] thus, prolapse of the tongue is probably less important. It is possible that external pressure from the bedding materials on the mandible in the prone position may cause posterior displacement of the mandible, compromising the airway. Another possibility is that children have a decreased lung capacity when prone, due to pressure on a compliant thorax. Decreases in lung capacity have been shown to result in narrowing of the upper airway.[28,29] Alternatively, children with OSAS may do worse in the prone position because the dependent tonsils become more congested. None of these mechanisms, however, would explain the differences between the supine and lateral positions. Another possibility is that children in the prone or lateral position have flexion of their neck, which can lead to increased upper airway collapsibility.[30,31] In the current, retrospective study, we were unable to assess the degree of neck flexion or rotation. It is unlikely that the increase in obstructive apnea noted in non-supine positions was due to artifact. Two sensors of airflow (thermistor and end-tidal PCO_2) were used. Furthermore, obstructive events were all associated with paradoxical breathing, and frequently with desaturation and/or arousal; and patients were observed on video, with obstructive breathing documented by the technicians.

There are some technical limitations to this study. This was a retrospective study, with no standardization of body position. Furthermore, the position of the head and neck was not assessed. Therefore, a prospective study evaluating body position is warranted. Study groups were not directly comparable, as the obese children were more likely to be older, female, and African American than the non-obese children. This is not surprising, as it is consistent with current demographic trends.[32] However, polysomnographic data were comparable between the obese and non-obese groups, with the only difference being the percentage of REM time.

This study included patients with OSAS secondary to adenotonsillar hypertrophy or obesity only. Children with OSAS secondary to craniofacial disease were excluded, due to the heterogenousity of the conditions represented. Some patients with craniofacial anomalies are thought to have improved breathing in the prone condition, particularly patients with micrognathia or macroglossia.[33,34] The findings of this study, therefore, should not be extrapolated to patients with craniofacial anomalies.

In summary, we have shown that children with OSAS breathe best when in the supine condition. This was a retrospective study with a relatively small sample size, and further, prospective studies are warranted. In the interim, however, we recommend that consideration be made to encourage children with OSAS to sleep in the supine position pending treatment. However, as obstructive apneas occurred in all positions, definitive therapy other than positioning is required.

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REFERENCES

1. Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. Am J Respir Crit Care Med 1999;159:1527-1532.
2. American Thoracic Society. Cardiorespiratory sleep studies in children: Establishment of normative data and polysomnographic predictors of morbidity. Am J Respir Crit Care Med 1999;160:1381-1387.
3. Kavey NB, Blitzer A, Gidro-Frank S, Korstanje K. Sleeping position and sleep apnea syndrome. Am J Otolaryngol 1985;6(5):373-377.
4. George CF, Millar TW, Kryger MH. Sleep apnea and body position during sleep. Sleep 1988;11(1):90-99.
5. Oksenberg A, Silverberg DS, Arons E, Radwan H. Positional vs nonpositional obstructive sleep apnea patients: anthropomorphic, nocturnal polysomnographic, and multiple sleep latency test data. Chest 1997;112(3):629-639.
6. Brouillette RT, Fernbach SK, Hunt CE. Obstructive sleep apnea in infants and children. J Pediatr 1982;100(1):31-40.
7. Kleinman RE. Obesity. In: Rudolph AM, ed. Pediatrics. Norwalk, CT: Appleton & Lange, 1987:205-208.
8. Rechtschaffen A, Kales A. A manual of standardized terminology: techniques and scoring systems for sleep stages of human subjects. Los Angeles, CA: UCLA Brain Information Service/Brain Research Institute, 1968.
9. Sleep disorders atlas task force. Guilleminault C. EEG arousals: scoring rules and examples. Sleep 1992;15:173-184.
10. American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 1996;153:866-878.
11. Marcus CL, Omlin KJ, Basinki DJ, Bailey SL, Rachal AB, Von Pechmann WS et al. Normal polysomnographic values for children and adolescents. Am Rev Respir Dis 1992;146(5 Pt 1):1235-1239.
12. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force [see comments]. Sleep 1999;22(5):667-689.
13. Stata Statistical Software: Release 6.0. College Station, TX: Stata Corporation, 1999.
14. Diggle PJ, Liang K-Y, Zeger SL, ed. Analysis of longitudinal data. Oxford: Clarendon Press, 1995.
15. Stata reference manual release 6. College Station, Texas: Stata Press, 1999.
16. Goh DYT, Galster PMCL. Sleep architecture and respiratory disturbances in children with obstructive sleep apnea. Am J Respir Crit Care Med 2000;162:682-686.
17. Penzel T, Moller M, Becker HF, Knaack L, Peter JH. Effect of sleep position and sleep stage on the collapsibility of the upper airways in patients with sleep apnea. Sleep 2001;24(1):90-95.
18. Orr WC, Stahl ML, Duke J, McCaffree MA, Toubas P, Mattice C et al. Effect of sleep state and position on the incidence of obstructive and central apnea in infants. Pediatrics 1985;75(5):832-835.
19. Kahn A, Groswasser J, Sottiaux M, Rebuffat E, Franco P, Dramaix M. Prone or supine body position and sleep characteristics in infants. Pediatrics 1993;91(6):1112-1115.
20. Ishikawa T, Isono S, Saito J, Tanaka A, Nishino T. Prone position increases collapsibility of the passive pharynx in infants and small children. Am J Respir Crit Care Med 2000;161:A805.
21. Positioning and sudden infant death syndrome (SIDS): update. American Academy of Pediatrics Task Force on Infant Positioning and SIDS. Pediatrics 1996;98(6 Pt 1):1216-1218.
22. Kahn A, Groswasser J, Rebuffat E, Sottiaux M, Blum D, Foerster M et al. Sleep and cardiorespiratory characteristics of infant victims of sudden death: a prospective case-control study. Sleep 1992;15(4):287-292.
23. De Koninck J, Lorrain D, Gagnon P. Sleep positions and position shifts in five age groups: an ontogenetic picture. Sleep 1992;15(2):143-149.
24. Yildirim N, Fitzpatrick MF, Whyte KF, Jalleh R, Wightman AJ, Douglas NJ. The effect of posture on upper airway dimensions in normal subjects and in patients with the sleep apnea/hypopnea syndrome. Am Rev Respir Dis 1991;144(4):845-847.
25. Jan MA, Marshall I, Douglas NJ. Effect of posture on upper airway

dimensions in normal human. Am J Respir Crit Care Med 1994;149(1):145-148.
26. Safar P, Escarraga LA, Chang F. Upper airway obstruction in the unconscious patient. J Appl Physiol 1959;14(5):760-764.
27. Isono S, Shimada A, Utsugi M, Konno A, Nishino T. Comparison of static mechanical properties of the passive pharynx between normal children and children with sleep-disordered breathing. Am J Respir Crit Care Med 1998;157(4):1204-1212.
28. Series F, Cormier Y, Desmeules M. Influence of passive changes of lung volume on upper airways. J Appl Physiol 1990; 68(5):2159-2164.
29. Hoffstein V, Zamel N, Phillipson EA. Lung volume dependence of pharyngeal cross-sectional area in patients with obstructive sleep apnea. Am Rev Respir Dis 1984;130(2):175-178.
30. Thach BT, Stark AR. Spontaneous neck flexion and airway obstruction during apneic spells in preterm infants. J Pediatr 1979;94(2):275-281.
31. Thut DC, Schwartz AR, Roach D, Wise RA, Permutt S, Smith PL. Tracheal and neck position influence upper airway airflow dynamics by altering airway length. J Appl Physiol 1993;75(5):2084-2090.
32. Mei Z, Scanlon KS, Grummer-Strawn LM, Freedman DS, Yip R, Trowbridge FL. Increasing prevalence of overweight among US low-income preschool children: the centers for disease control and prevention pediatric nutrition surveillance, 1983 to 1995. Pediatrics 1998;101:e12.
33. Caouette-Laberge L, Bayet B, Larocque Y. The Pierre Robin sequence: review of 125 cases and evolution of treatment modalities. Plast Reconstr Surg 1994;93(5):934-942.
34. Benjamin B, Walker P. Management of airway obstruction in the Pierre Robin sequence. Int J Pediatr Otorhinolaryngol 1991;22(1):29-37.