# **The Effect of Body Position on Sleep Apnea in Children**

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## **Abbreviations**

- OSA Obstructive sleep apnea
- PSG Polysomnogram, polysomnography
- CPAP Continuous positive airway pressure
- AT Adenotonsillectomy
- MRI Magnetic resonance imaging
- SDB Sleep-disordered breathing
- CFD Computational fluid dynamics
- AHI Apnea-hypopnea index
- RDI Respiratory disturbance index

# **Introduction**

 In recent years, the increasing trend of childhood obstructive sleep apnea syndrome (OSA) has generally followed the increasing prevalence of obesity in that age group. Accordingly, between 1980 and 2000, the prevalence of obesity has doubled in the age group  $6-11$  and tripled among children  $12-17$  $12-17$  $12-17$  years of age  $[1, 2]$ , ultimately emerging as a leading cause for morbidity associated with OSA  $[3, 4]$ , and thus sowing the seeds for ill health and loss of economic productivity later in adulthood [5].

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 Adenotonsillectomy has generally been accepted as standard of care for remediation of PSG-proven OSA, in line with observed findings related to mechanical obstruction caused by extra-luminal soft tissues on the pediatric airway  $[6-8]$ . However, despite this, adenotonsillectomy fails to completely address OSA-related symptoms in certain children  $[9, 10]$  $[9, 10]$  $[9, 10]$  in whom altered tone  $[11, 12]$  and adiposity  $[13]$  play a significant role. Besides these factors, body position appears to modulate airflow significantly and is dependent on the stage of sleep  $[14]$ . Thus far, there is data to show both in adults and children that the chief metric of severity of OSA, the apnea-hypopnea index (AHI), is worse in the supine position. Both OSA severity and physiologic indicators of airflow, such as critical closing pressure and the optimum level of CPAP required seem to increase during the supine position during sleep  $[15]$ .

Despite the observed relationship between fixed anatomic factors such as size of pharyngeal soft tissues and the degree of sleep apnea, dynamic control of airway lumen during sleep—particularly *dependence* of oropharyngeal and nasopharyngeal soft tissues due to the effect of gravity in the supine position—may be difficult to quantify, and thus the purpose of this chapter is to lay a foundation for understanding the modulation of the *physiologic* airway by position of the body.

#### **Pathophysiology of Upper Airway Obstruction**

 The upper aerodigestive tract in humans maintains similarity to other mammals at birth. However, at about 18 months postnatally, there is a gradual drive towards compartmentalization of function for the pharyngeal and laryngeal musculatures [16, [17](#page-9-0)]. During sleep, pharyngeal dilators that oppose narrowing of the oropharyngeal airway during wakefulness have decreased tonic activity. In normal individuals, this decrease does not reach the critical closing pressure resulting in maintenance of patency at all times. However, in individuals with OSA, the tonic activation of pharyngeal dilators is enhanced during the awake condition compared to those without OSA. During sleep, the lack of this tonic activation leads to marked reduction in airway patency. Both neurologic and anatomic factors modulate this sleep-related reduction in airway patency. The *neural* hypothesis suggests that in the presence of OSA, there is a tendency towards general reduction of pharyngeal tone, especially within the genioglossus [ [18](#page-9-0) ]. Alternatively, the anatomic hypothesis favors normal sleep-related reduction of tone within the upper airway that potentiates lack of stability within the pharyngeal musculature [19]. Thus the decreased tonic activation of pharyngeal dilators poses an anatomically unfavorable circumstance eventually enhancing dependent collapsibility in children with OSA.

 Subsequent to formation of discrete channels within the upper aerodigestive tract, anomalous anatomic factors related to development can contribute to OSA in infants. It is to be noted that in very young infants, physiologic factors such as central nervous system immaturity can contribute significantly to the pathogenesis of OSA. Craniofacial syndromes, such as Crouzon's and Apert's, and mandibulofacial dysostoses, such as Treacher Collins and Pierre Robin sequence, have distinct roles in changing the upper airway collapsibility either anatomically or by altering the neuromuscular tone  $[20, 21]$ . In this age group, full-term neonates who are obligate nasal breathers are susceptible to limitation of airflow following nasal obstruction due to anatomical anomalies or in the presence of an upper respiratory tract infection.

 Thus far, the role of adenotonsillar hypertrophy in the pathogenesis of OSA in children has been validated by consensus statements from the American Academy of Pediatrics who recommends adenotonsillectomy as the gold standard initial treatment  $[22, 23]$ . Indeed, many authors have reported improvement in nasal airflow subsequent to adenotonsillectomy, resulting in both resolution of symptoms and improvement in quality of life in these children [24]. These changes are directly contingent upon the extent of volumetric reduction of naso- and oropharyngeal soft tissues, and is equivalent to a decompression of static obstructive pathology, as confirmed by polysomnographic and MRI-derived information  $[25]$ .

 Given this information, the success of adenotonsillectomy in remediation of symptoms in obese children with OSA is suboptimal. For example, while the success rate is in excess of 80 % in non-obese children with OSA, it drops to less than 40 % in overweight and obese children, with the accepted norm that adenotonsillectomy improves, yet does not resolve, OSA-related symptoms in the second group  $[26]$ .

Immaturity correlates significantly with the severity of OSA, with spontaneous neck flexion, glottic closure, and a highly compliant chest wall contributing to events associated with cessation of airflow in more than half of infants presenting with apneic events  $[27]$  that are very responsive to CPAP-based treatment. These events are known to occur with increased association and frequency with sleep and are most pronounced in deep stages.

 While both adenotonsillar hypertrophy and extraluminal adiposity represent static factors that change the size and shape of airway lumen, dependence and position aggravate sleep-disordered breathing in a dynamic fashion. A schematic of the relative contributions of these factors are shown in Fig. [1](#page-3-0). Based on the structural model suggested by Isono and colleagues  $[15]$ , less force is required in an anteroposterior dimension to compress the airway in the supine position compared with that in a lateral position, thus increasing the tendency towards collapse.

Various authors have highlighted fluid dynamics that are passively altered in association with increased upper airway collapsibility in children with OSA. Some understanding of fluid-volume physics of the upper airway is of potential importance to determine the poor response seen in some children with OSAS to adenotonsillectomy.

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**Fig. 1** Influences of dynamic and static forces on the airway (cylinder). (a) and (b) show, respectively, the focally obstructed airway in adenotonsillar *vs* . an airway that is attenuated along its entire length. Shape of the airway also constitutes a risk—specifically, a more *elliptical* or *eccentric shape*. Similarly, a larger cross-sectional area in a representative airway as shown in (d) is more prone to collapse when compared to (c). Differences in the tendency to collapse are illustrated to the right

## **Biomechanics of Upper Airway Obstruction**

## *History of Fluid Models*

Historically, fluid models have found use in the analysis of nasal airway obstruction and for preoperative planning prior to functional septorhinoplasty in adults. In addition, inspiratory peak flow limitation has been found to correlate very well with functional limitations in allergic rhinitis. A significant, negative, nonlinear relationship was also found between minimum cross-sectional area and intranasal resistance. Furthermore, acoustic rhinometry demonstrated that small intrusions into the lumen of the nose that might be missed by rhinoscopy produce an exponentially greater increase in nasal resistance measurements by rhinomanometry [28].

Computational fluid dynamics is a branch of fluid physics that uses computerbased mathematical algorithms to model and determine the parameters affecting



Fig. 2 Upper airway models of an OSA airway (a) and a normal control (b) signifying the overlap area (*red*) with the palatine tonsils below and lateral to the oropharynx and nasopharynx overlap the adenoids superior and posterior to the nasopharynx. In this location, there is a significant drop in cross-sectional area. This leads to both increases in turbulence and reduction of airflow. For data from reconstructed MR images, see Xu et al.  $[31]$ 

fluid flow. With advances in computational power and distributed processing, airflow characteristics in a geometrically altered airway can be delegated to the individual subunits of a central processing unit (CPU) of a computer.

Historically, the upper airway and its associated flow resistance has been modeled either as an elliptical orifice with local airway obstruction in a 2-D model of pharyngeal airflow  $[29]$  or that of a 3-D conduit to study the effects of area variation and turbulence  $\lceil 30 \rceil$  as a function of pressure exerted by extraluminal soft tissues. Indeed, as discussed in the following sections, effects of dependence (gravity) on luminal collapsibility are shown by computational models of the upper airway and confirmed by clinical and PSG-derived data from pediatric populations.

#### *Techniques for Modeling the Obstructed Airway*

 Modeling the obstructed airway encompasses several parameters that may be incorporated into two broad types (Fig. 2). In most models  $[31, 32]$ , various fluid dynamic parameters such as pressure drops, velocity distribution, wall shear stress, and distribution of wall force induced by pressure at various locations within the airway are calculated with various inspiratory flow rates. However, in these models, deformability of the airway wall—either induced by gravity or by extra luminal soft tissues—is not taken into account, and thus, the rendition of data from this



 **Fig. 3** Cross-sectional anatomy from normal breathing and apneic episodes ( *solid* and *broken loops* ). *NP* nasopharynx, *OL* overlap area, *OP* oropharynx, *SG* supraglottis

technique suffers from oversimplification. In the second type of approach that is also computationally intensive, a finite element model (FEM)<sup>1</sup> may be established with contribution from luminal variability as a function of deformability and material stress providing more realistic anatomical models. In either of these types of models, both CT- or MRI-based volumetric data may be acquired for computational reconstructions.

The central theme of simulations of fluid dynamics of the airway is the Navier-Stokes (NS) equation, with or without Reynolds averaging, among others, such as the Spalart-Allmaras model. A detailed description of all the mathematical models is beyond the scope of this section. Briefly, NS equations are useful for describing behavior of fluids and have found applications to model weather, ocean currents, and vascular flow patterns. One of the fundamental characteristics of biological flow patterns is that of *turbulence* , which is the time-dependent chaotic behavior seen in fluid flow. This is relevant to flow within the upper airway in children with OSA, which is characteristically turbulent, with worsening seen in focally narrowed sections such as the nasal valve area and the retropalatal and retroglossal segments.

Xu et al.  $[31]$  studied computational fluid models of the upper airway using MRIderived volumetric information and showed luminal restriction as shown within an overlap area in Fig. 3. This illustrates the airflow patterns as derived from the data shown by the same authors, re-rendered two dimensionally. The highlighted area points to the site of maximum obstruction and is enhanced during sleep. The overlap area is normally located between the adenoids posterosuperiorly within the nasopharynx and palatine tonsils inferolaterally within the oropharynx in patients with OSA. Importantly, a turbulent jet is expected downstream of the site of obstruction [\[ 33](#page-10-0) ]. The authors concluded that in children, area restriction in the pharynx may be

<sup>&</sup>lt;sup>1</sup>FEM involves replacement of continuous geometry with a set of objects with a fixed number of simple components. Elements connected at nodal points that may be common to two or more adjacent elements. A set of elements is referred to as *mesh.*

as important as nasal resistance in driving airway collapse in OSA. Thus, respiratory system models that include apnea mechanics may benefit from including pharyngeal restriction along with airway collapse properties. These CFD models may also be used as a benchmark for building simplified models of pressure drop in the pharynx to analyze larger numbers of subjects or for incorporation into dynamic system models.

## **Impact of Dependence on Airway Collapsibility: Theoretic Models**

 As described in the previous section, the upper airway is susceptible to collapse from extraluminal pressure exerted within the retroglossal and retropalatal areas with considerable reduction of luminal diameter at the overlap area. Huang et al. [34] recently completed a FEM modeling study to understand the circumferential deformability of the airway using material properties such as Young's modulus and Poisson ratio. In this study, the lumen is characteristically an ellipse that has variable material properties anteroposteriorly and laterally (Fig. 4 ). The pharynx in a patient with OSA is analogous to a collapsible conduit when the pharyngeal constrictors are completely relaxed. The degree of structural collapse during sleep is inversely proportional to the diameter of the awake airway due to increase in kinetic energy of fluid flow through these segments.

 When deformability across the longitudinal plane is considered, the material properties including compliance and stiffness of the airway are opposed by extraluminal, compressive force exerted by gravity. In this scenario, it is critical to understand that the airway patency is maintained as a balance between intrinsic mechanical properties of the pharynx and the neural control of pharyngeal dilators.



Fig. 4 Influence of dependence on change in shape and structural collapsibility of the pharyngeal airway. Here, lateral position causes the airway to be more *circular* in shape with a larger pressure gradient required to collapse compared to the supine position. In the latter, the radius of curvature ( *red* ) is 50 % that of a circular airway inducing a collapse in the longitudinal axis

It may thus be postulated that the improvement of pharyngeal luminal diameter due to the lateral position may be due to structural change or increase in dilator tone. Using optical coherence tomography, the physical changes in shape and orientation have been studied before [35]. In this study, the authors report that the airway changes from a transversely oriented elliptical shape when supine to a more circular shape when in the lateral recumbent posture. Laplace's law states that at equilibrium, the pressure gradient across a concave surface is directly proportional to the wall tension and inversely proportional to its radius of curvature. It follows that the pressure gradient required to compress the airway varies inversely as a function of its radius of curvature. Hence, as the transverse elliptical airway changes to a more circular shape with change to the lateral posture, its propensity to collapse decreases as a function of the reduction in radius of curvature of its anterior and posterior walls. In proportions, this can be expressed as ratio of the lengths of the major and minor axes of the elliptical cross section.

#### **Clinical Correlates of Theoretic models**

 For several years, it has been known that sleeping in prone position can increase the amount of time spent sleeping, particularly the duration of quiet sleep [36]. In addition, it has also been known that prone sleeping is associated with reduced responsiveness to arousal stimuli [37]. Using the theoretic and modeling-derived data described in the previous section, we review the correlation of sleep position with clinically derived data, specifically in children, OSA parameters.

Pereira et al. [38] examined the variability of respiratory disturbance index (RDI) in children less than 3 years of age with sleep position from PSG data. The authors found that RDI positively correlated with the amount of time spent in the supine position, thus raising questions about PSGs that underestimate the severity of OSA by not sampling the postural variability of sleep apnea. This data is in agreement with theoretic models that show enhanced collapsibility in the supine position caused by dependent forces that act on the extraluminal soft tissues.

 In another study from the same institution and authors, infants aged 8–12 months [39] referred for suspicion of sleep-disordered breathing were retrospectively reviewed. AHI variation was determined as a function of body position. This study attempted to characterize the relationship between the hypoxic episodes associated with OSA and the age at which infants begin to change body position. This study showed that there was no significant effect of body position on sleep-disordered breathing, although REM sleep represented a significant risk factor for OSA. This result assumes significance as sudden infant death syndrome (SIDS) is currently the most common cause of postnatal death in the United States and has known association with OSA  $[40]$ .

 Isono and colleagues [ [15 \]](#page-9-0) determined atonic properties of the human pharynx by inducing a deep plane of anesthesia and examining the effectiveness of lateral position in improving structural properties of each pharyngeal segment, consequently

eliminating neuromuscular factors. Although the study was in adults, their results showed a convincing reduction in pharyngeal closing pressures in the lateral position. However, this is not in agreement with findings from patients with artificially occluded nasal airways where there is a small yet nonsignificant reduction in pharyngeal closing pressures.

#### **Effect of Body Position on OSA: Role of Current Therapies**

 In summary, a number of studies have evaluated the effect of body position during sleep on OSA and have not effectively agreed on a consensus. While one study showed an increase in AHI in the supine position [38], another showed the opposite [\[ 41](#page-10-0) ]. Some of these differences may be related to study design. This is a fundamental difference when compared with adults in prone position that improves OSA by reduction of tongue prolapse. Given the multitude of results, no recommendations can be made for maintaining a particular position of sleep other than to avoid the supine position which results in the maximum amount of airway obstruction. There is need for more data in this regard to establish firmer guidelines.

 The gold standard for treatment of OSAS in children continues to be adenotonsillectomy. There is overwhelming evidence to support adenotonsillectomy in remediation of OSA symptoms showing uniform improvement in PSG criteria in majority of the children undergoing the procedure. Obesity appears to limit the usefulness of this procedure. A meta-analysis of adenotonsillectomy in obese children showed that 88 % of obese patients still had a postoperative AHI > 1 per hour [26]. It is thus of critical importance to stress on weight management in these children including referrals as indicated.

 Intranasal corticosteroids are useful for initial trial in children with mild OSA. They cause a dose-dependent reduction of proliferation of nasopharyngeal soft tissue, and this is supported by both level I clinical and histopathologic evidence shown in tonsils harvested from children with OSA [\[ 42](#page-10-0) ]. In children with orthodontic abnormalities that are directly related to OSA, use of dental appliances is controversial. Thus far, the data is very limited with one study showing a reduction of snoring and restless sleep in the background of a small sample size and nonblinded study design. Despite this, dental appliances may be useful in reduction of anomalous occlusion and consequently improve OSA. Other therapies such as highflow oxygen that circumvents the inspiratory flow limitation and hypoxic spells may provide a physiologic solution, but the use and transport of such a device would be cumbersome. Typically, it is thought that children requiring lower levels of CPAP could potentially be candidates for high-flow oxygen delivery devices such as Vapotherm™.

Thus far, a number of anatomic and neuromotor aspects of airflow within the upper airway have been discussed for critical analysis of positional changes associated with OSA. Despite the few studies performed that investigated PSG-derived information to assess changes during sleep, there has been no consensus, although <span id="page-9-0"></span>age and the stage of sleep appear to affect the severity. Obesity indeed plays a role in OSA treatment and alters the response to adenotonsillectomy. Weight management remains a critical issue that prevents and reduces the extraluminal compression of the upper airway during sleep. Computational tools have helped us understand the physical changes in flow that take place as a function of apneic episodes. Despite this, more level I clinical evidence is necessary at this stage to establish firm recommendations for alteration of posture during sleep in children >1 year of age.

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