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Radiologic evaluation of adenoids and tonsils in children with obstructive sleep apnea: plain films and fluoroscopy*

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Abstract. Twenty-six children with obstructive sleep apnea were evaluated by lateral neck radiographs during wakefulness, and by polygraphic monitoring and upper airway fluoroscopy during natural sleep. Children with craniofacial abnormalities, palatal surgery, and central nervous system disease were excluded from the study. Moderate or marked enlargement of tonsils and adenoids was noted on lateral neck radiographs of 18 of 26 patients. An objective measure of adenoidal enlargement, the adenoidal-nasopharyngeal ratio, correlated well with subjective judgment of adenoidal size but was not generally more useful than subjective estimation. Upper airway fluoroscopy demonstrated the site and mechanism of obstruction in all patients. Because all children with moderate to marked adenotonsillar enlargement demonstrated obstruction at the adenoidal or tonsillar level on fluoroscopy, we now screen children with suspected sleep apnea with lateral airway radiographs and polysomnography. Fluoroscopy is reserved for children with mild adenotonsillar enlargement, craniofacial dysplasia, prior cleft palate repair, or neuromuscular disorders. These results suggest that the pathogenesis of obstructive sleep apnea in children involves anatomic factors which narrow the upper airway, sleep-related hypotonia of pharyngeal dilator musculature, and compensatory mechanisms to prevent or alleviate asphyxia.

Key words: Apnea, sleep – Adenoids – Tonsils – Airway radiography

Obstructive sleep apnea in childhood may be associated with several seemingly nonrelated conditions: craniofacial dysplasias, neuromuscular disorders, palatal surgery, and the more common enlargement of the tonsils and adenoids [1–6]. Reports have documented the varied presentation of children with obstructive sleep apnea [7, 8]. Hypersomnolence, behavior disorders, secondary enuresis, and failure to thrive have all been attributed to obstructive sleep apnea. When diagnosis is delayed, as it frequently is, serious sequelae may develop, including cor pulmonale and neurologic damage [9–15]. Lack of awareness of pediatric obstructive sleep apnea as a treatable disease and failure of the physician to examine the sleeping child have been mentioned as two reasons for the delay in diagnosis [11]. A recently published report [11] has stressed the importance of correctly diagnosing obstructive sleep apnea as early as possible and has provided clinical criteria for making this diagnosis. Radiologic signs of obstructive sleep apnea in children have not been similarly developed.

We attempted to examine the usefulness of two methods of evaluating the lymphoid tissue on standard lateral neck radiographs in 26 children with obstructive sleep apnea. The more commonly used subjective system of grading tonsillar and adenoidal size was compared to the adenoidal-nasopharyngeal ratio (AN ratio). The AN ratio, devised by Fujioka et al. [16], compares the amount of the lymphoid tissue in the nasopharynx to the size of the nasopharyngeal compartment (Fig. 1), but does not assess tonsillar size.

Upper airway fluoroscopy was performed on these same 26 children. During a period of natural sleep, multiple signs of partial and complete airway collapse, as well as compensatory mechanisms to keep the airway patent were observed. While previ-

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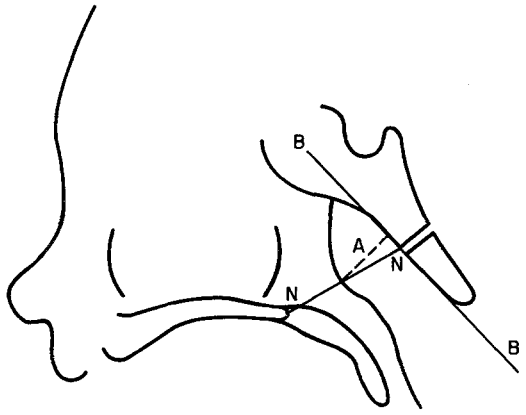

Fig. 1. Measurements necessary for calculation of the AN ratio, modified from Fujioka et al. [16]. Line BB is tangential to the basiocciput. The adenoidal measurement (A), shown by the dotted line, is obtained by drawing a perpendicular line to BB at the point of maximal adenoidal tissue. The measurement of the nasopharynx (NN) is made between the most posterior portion of the hard palate to the anteroinferior aspect of the basisphenoidal synchondrosis

Table 1. Fluoroscopic changes in the upper airway in pediatric obstructive sleep apnea

I. Signs of complete or partial inspiratory collapse
Prevertebral soft tissues move anteriorly and appear thickened
Tonsils move posteriorly or down into airway
Palate moves against adenoids or posterior pharyngeal wall, or elongates
Jaw moves backwards (occasionally)
Tongue moves backwards (occasionally)
II. Compensatory mechanisms to maintain airway patency
Widening of nasopharynx above obstruction
Jaw opening, jaw rocking
Arousal from sleep

ous authors have advocated videofluoroscopy in the evaluation of obstructive sleep apnea [1, 2, 17–19], no prior report has been directed to the findings in a large pediatric series. Also, few guidelines have been established to help the radiologist decide what are appropriate indications for videofluoroscopy. On the basis of our results, we recommend specific patient groups in whom fluoroscopy will be useful and discuss the pathogenesis of pediatric obstructive sleep apnea.

Definition of terms

Apnea has been defined as a cessation of respiratory flow. Three kinds of apnea – obstructive, central, and mixed – must be differentiated [20]. Obstructive apnea occurs when continued respiratory efforts fail to produce air flow because of mechanical blockage. Central apnea is said to be present when the airway remains patent but respiratory efforts cease. Mixed apnea contains periods of both central and obstructive apnea.

In adults and older children, the obstructive sleep apnea syndrome has been diagnosed when there are at least 30 episodes of obstructive apnea longer than ten seconds within a seven-hour sleep period [21, 22]. However, the definition of sleep apnea in infants and children has been evolving as data are accumulated [2, 23, 24]. Fifteen seconds without respiratory efforts in infants less than 52 weeks and ten seconds without respiratory efforts in older infants have been proposed as criteria for central apnea [25]. Because Carskadon et al. [24] found no obstructive or mixed apnea in 22 normal children, even shorter obstructive apneas may be abnormal [11]. Furthermore, many children with obstructive sleep apnea have relatively few complete obstructions, but nearly constant partial obstruction during sleep as indicated by hypercarbia, snoring, and paradoxical inward movement of the chest on inspiration [11].

In this study, as in a prior paper [11], we diagnosed obstructive sleep apnea syndrome: (1) when episodes of partial or complete airway obstruction during sleep were associated with abnormal transcutaneous oxygen and end-tidal carbon dioxide values on polygraphic monitoring, and (2) when there were sequelae of the sleep-related asphyxias, such as failure to thrive, cor pulmonale, or neurobehavioral disturbances. Total obstruction was indicated by lack of airflow at the nares or mouth. Partial obstruction was said to be present when expired P_ACO_2 rose to 45 mmHg.

Subjects and methods

Patient selection

Twenty-six children (21 boys and five girls) aged seven months to ten years were studied. No child in this group had a craniofacial abnormality, neural or neuromuscular impairment, or prior cleft palate repair.

Lateral neck radiographs

Airway radiographs were performed using a modification of the method described by Joseph et al. [26]. This technique employs geometric magnification, selective filtration, and high kilovoltage. Lateral neck films were taken during inspiration with the patient awake and sitting upright and the head supported by a chin strap. A staff pediatric radiologist, without knowledge of the fluoroscopic results, subjectively graded tonsillar and adenoidal size as normal, or having mild, moderate, or marked enlargement. The AN ratio was computed (by SKF) in 22 children. Mean AN ratios for our patients were compared with the norms of Fujioka et al. [16], using a two-tailed one sample t-test. The average AN ratio for 908 normal patients from six months to eleven years was calculated from Table 1 in the report by Fujioka et al. [16].

Fluoroscopy and polygraphic monitoring

Fluoroscopy and polygraphic monitoring were performed during an afternoon nap in a large, darkened room. To enhance sleep, the children were kept up after 2 AM on the night preceding the exam. Sedative or hypnotic drugs were not used. With the child supine, lateral projection horizontal-beam fluoroscopy was used to observe the entire upper airway from nose to trachea. When possible, the child underwent fluoroscopy during a completely obstructive apneic episode. If no such episodes occurred, fluoroscopy was performed during periods of partial airway obstruction, as indicated by snoring, inspiratory retractions, the use of accessory muscles of respiration, and hypercarbia. In selected patients, it was necessary to introduce barium into the nasopharyngeal airway to better define airway size. The 45-s to 2-min exam was recorded on videotape.

Simultaneous monitoring of heart rate, ECG, expired carbon dioxide and transcutaneous oxygen levels was carried out during the entire nap. Mercury strain gauges placed around the thorax and abdomen measured respiratory efforts and documented continued efforts in the absence of airflow (obstruction) and thoracic and abdominal motion out of phase with each other. Obstructive apneic episodes greater than 10 s were counted and the number of apneas per h calculated for the test period. Central apneic episodes were tallied separately.

Five of these children were completely restudied after tonsillectomy and adenoidectomy had alleviated the symptoms. Four additional children felt to be normal when all tests were analyzed were also studied with fluoroscopy. Fluoroscopy in these nine children showed minimal variation of pharyngeal airway dimensions with breathing.

Results

Lateral neck radiographs

In 21 of the 26 children, enlargement of the tonsils and/or adenoids was identified. Eighteen of these children had moderate to marked enlargement of the tonsils and adenoids. The degree of tonsillar hypertrophy was parallel to the degree of adenoidal enlargement in nine children and was disparate in twelve. In these twelve children, the adenoids were judged to be moderately or markedly enlarged in the presence of only mild tonsillar hypertrophy. There were three children with pediatric obstructive sleep apnea whose tonsils and/or adenoids were only mildly enlarged. Five children had airway studies interpreted as normal.

The AN ratio was calculated in 22 children. Technical problems (poor positioning, over-collimation) precluded measurement of the standard diameters on the lateral neck radiographs in the other four. Nineteen children had an AN ratio within two standard deviations of the mean value for their respective age (Fig. 2). Twenty-one children had an AN ratio at or above the mean value. Only three children had an AN ratio which was greater than two standard deviations above the mean. Thus, 23 (88%) of the 26 children with proven pediatric obstructive sleep apnea would have been missed if screened using the AN ratio alone.

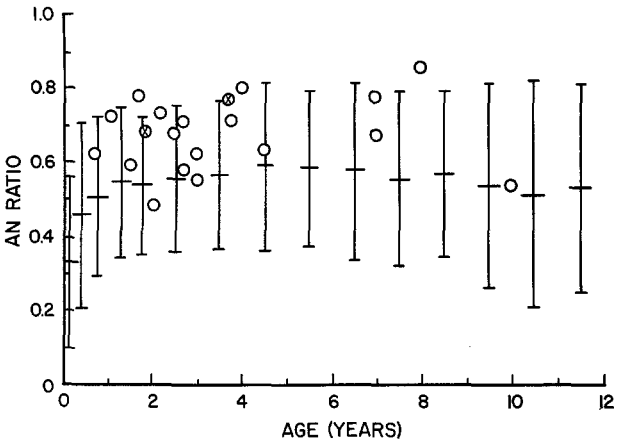

Fig. 2. Calculated AN values of children with obstructed sleep apnea. Each circle represents the AN ratio of one child. Each crossed circle represents two children with identical AN ratios. The mean AN value (long horizontal bar) and two standard deviations above and below the mean (shorter horizontal bar) are indicated

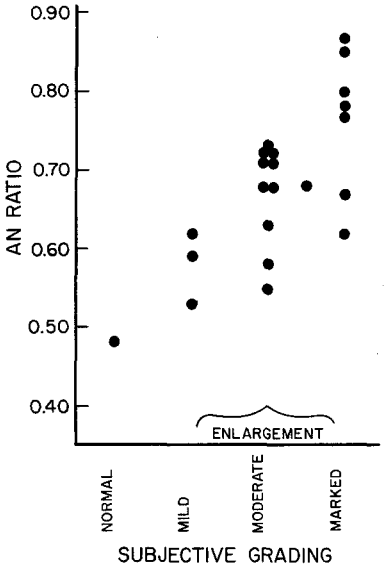

Fig. 3. Comparison of measured AN ratio to subjective grading system of adenoids. While there is some overlap of subjective grading, the experienced radiologist usually separated the different levels of adenoidal enlargement

The mean AN ratio of the 22 children with pediatric obstructive sleep apnea was 0.67 ± 0.10 ($\pm$ SD). This mean was significantly ($p < 0.001$) greater than the mean value of 0.55 derived for the 908 normal infants and children reported by Fujioka et al. [16]. The lower 99% confidence limit for the mean AN ratio of children with pediatric obstructive sleep apnea was greater than the mean AN ratio of normal patients of any age.

The AN ratio in each patient was compared to the subjective grading of adenoidal size (Fig. 3). While there was some overlap among the various

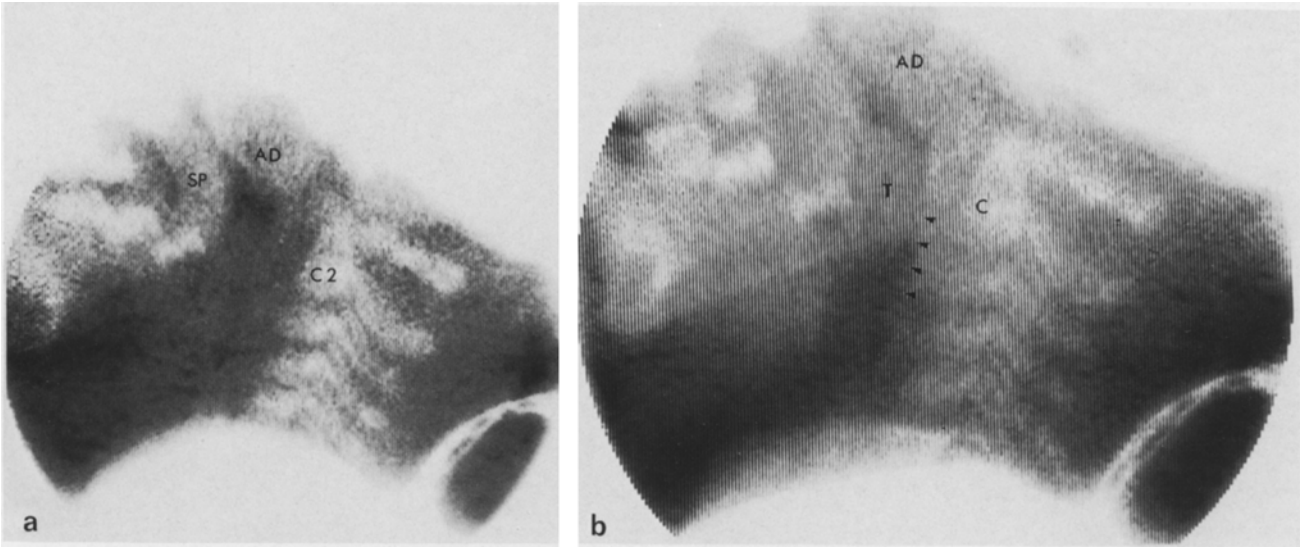

Fig. 4. a An occluded inspiration. Collapse of the airway has produced poor definition of almost all airway landmarks. **b** Expiratory phase in the same patient. The posterior pharyngeal wall (arrowheads) is distinct. The enlarged tonsil can now be appreciated. (AD = adenoids, SP = soft palate, C_2 = second cervical vertebra)

degrees of adenoidal enlargement, the radiologist generally categorized adenoids with lower AN ratios as normal or mildly enlarged. Similarly, with few exceptions, adenoids which measured large were interpreted as moderately to markedly enlarged.

Upper airway fluoroscopy

All of these 26 children experienced upper airway obstruction during sleep. By correlating the various physiological parameters with the fluoroscopic observation, it became evident that partial airway obstruction was more frequent than complete airway obstruction. Isolated partial airway obstruction, occurring in approximately 65% of our population, was frequently associated with profound physiological or behavioral disturbances. Partial airway obstruction combined with periods of intermittent complete airway obstruction was present only in 35% of the children studied.

The fluoroscopic signs of complete pharyngeal airway occlusion were not subtle. During inspiration, the air column became obliterated (Figs. 4 and 5). The air-soft tissue interface around the tonsils, epiglottis, and other airway structures was lost as adjacent structures abut each other. After several occluded breaths, the child opened the pharyngeal airway by changing the position of his tongue, head, or body, thus allowing respiration to resume (Figs. 4 and 5). Often arousal or semiarousal from sleep preceded airway reopening.

In every patient with physiological indications of obstruction, we noted thickening of the prevertebral soft tissues below the point of obstruction during inspiration (Fig. 6). Only during obstructed inspirations did the posterior pharyngeal wall move forward, narrowing the airway. No child with central apnea and no child studied after surgical relief of obstructive sleep apnea demonstrated this change.

The tongue, tonsils, and palate may change position and configuration during obstructed respiration. On inspiration, the tongue may appear to be displaced posteriorly. The tonsils may elongate and be drawn inferiorly and, to a lesser extent, posteriorly. The changes in the palate were less constant. The palate may remain unchanged throughout respiration, may become the site of obstruction if it is apposed to the precervical soft tissues, or may elongate and be drawn into the airway like the tonsil.

Jaw motion, negligible during normal respiration, may be accentuated. One child, examined early in our experience, exhibited a marked "in and out" motion of the jaw. We have previously published figures demonstrating the movement of his jaw [11]. In other patients, lesser degrees of posterior jaw motion were intermittently seen to accompany obstructed inspirations.

A few compensatory mechanisms to keep the airway patent either preceded or did not require the patient's arousal from sleep. Although the airway below the site of obstruction collapsed during inspiration, the airway above an obstruction characteristically dilated on inspiration. During episodes of tonsillar obstruction, the intraoral airway may increase by jaw opening, a rocking jaw. Similarly, the nasopharyngeal airway may widen as the soft palate des-

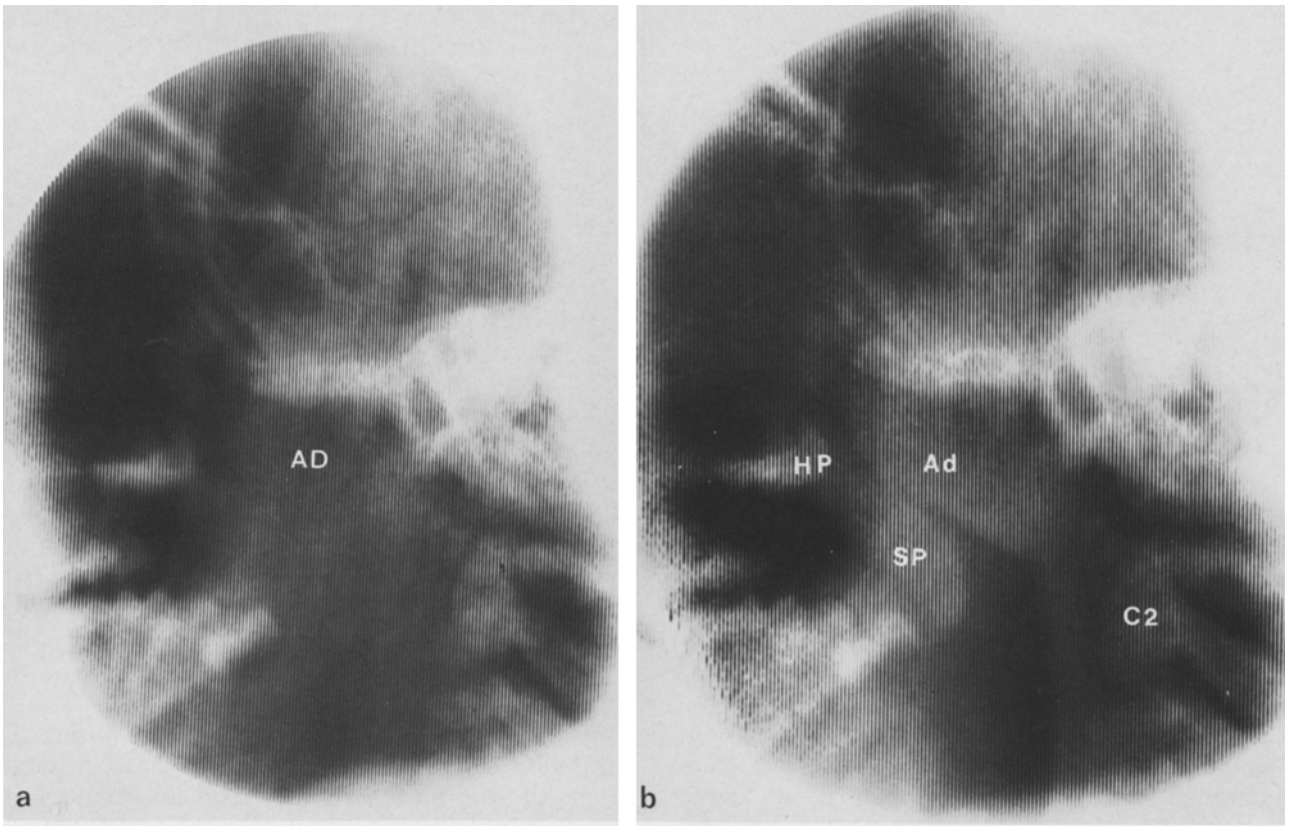

Fig. 5. a An occluded inspiration. Except for bony structure, airway landmarks have been obscured. All soft-tissue planes have merged. **b** Expiratory phase in same patient. Marked adenoidal hypertrophy is evident (AD) in this child who had undergone prior tonsillectomy. (AD = adenoids, SP = soft palate, HP = hard palate)

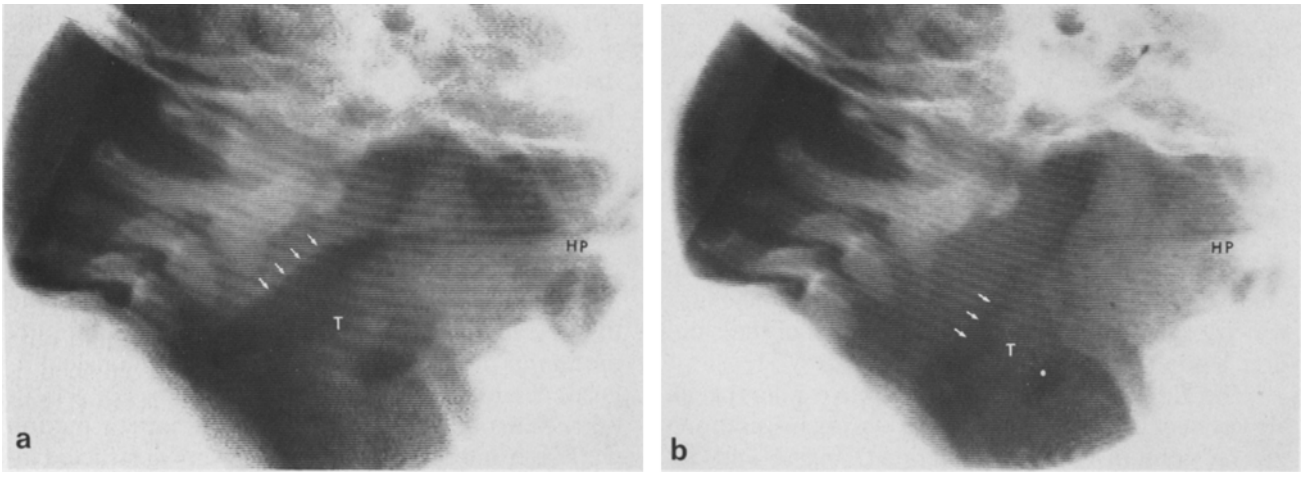

Fig. 6. a The tonsil is clearly seen as the hypopharynx is normally distended during expiration. The posterior pharyngeal wall (arrows) defines the anterior margin of the precervical soft tissues. **b** During inspiration, collapse of the airway makes tonsillar mass harder to define. Precervical soft tissues appear to thicken. (HP = hard palate, T = tonsils)

cends. Generally these mechanisms were ineffective in establishing or maintaining a patent airway. Table 1 summarizes the fluoroscopic signs of airway collapse and the observed compensatory mechanisms.

Treatment and follow-up

Nineteen children underwent tonsillectomy and adenoidectomy. One child underwent tracheostomy and two were treated only with decongestants. Three

children have been lost to followup, and one is awaiting treatment. Twelve of the 19 children had a full polysomnographic study performed after tonsillectomy and adenoidectomy, showing improvement in monitored parameters. Five of these children underwent brief postoperative fluoroscopy of the airway; in each instance, inspiratory pharyngeal collapse was not seen. Parents reported improvement in the sleep pattern, behavior, etc. in 17 of the 19 children. Post-operative improvement was documented with polygraphic monitoring in the child who required tracheostomy.

Discussion

In adults, obstructive sleep apnea is believed to be due to a physiologic abnormality in an anatomically normal pharyngeal airway [28]. The usual treatment is a tracheostomy to bypass the collapsing segment.

Pediatric obstructive sleep apnea is very different. Anatomic abnormalities of the upper airway (craniofacial dysplasia, tonsillar and adenoidal enlargement, obstructive pharyngeal flap) are frequently present and interact with a sleep-related physiologic or neuromuscular defect in upper airway maintenance to produce partial or complete airway obstruction [2, 4, 29]. In contrast to most adults, surgical correction of the anatomic abnormality produces relief from pediatric obstructive sleep apnea [12].

During normal inspiration, the diaphragm and other respiratory muscles contract and generate negative inspiratory pressure, drawing air into the lungs. Simultaneously, other "inspiratory muscles" in the upper airway contract to maintain or dilate the nasal, pharyngeal, and laryngeal airways, allowing inspiratory airflow. Both animal and human studies have demonstrated that the pharynx will collapse unless supported by contraction of these airway-maintaining muscles [30, 31]. Recently, Konno et al. [32] demonstrated negative inspiratory pressures of -5 to -25 cm H_2O within the esophagus of pediatric patients with obstructive sleep apnea-pressures several times those needed to collapse the airway of a human infant [31]. Remmers et al. [28] documented the presence of negative intraluminal pharyngeal pressure in adult patients with upper airway occlusion during sleep and stressed the role of airway-monitoring muscles in counteracting the generated negative transmural pressure.

The genioglossus muscles, the major group of airway-dilating muscles, originate on the anterior mandible and insert on the hyoid bone and the posterior part of the tongue. By contracting, they pull the tongue forward and out of the airway during inspiration. Sauerland and co-workers [33] have shown that, in human adults, the activity of the genioglossus muscles decreases during sleep. In adult patients with obstructive sleep apnea, it has been postulated that this sleep-related hypotonia of the genioglossus muscles allows negative inspiratory pressure to pull the tongue backward, obstructing the pharyngeal airway [28]. In children, the changes of the airway musculature have not been as thoroughly documented.

The tendency of obstructive apnea to manifest itself during sleep has been also elucidated by physiological studies. During sleep, there is an overall decreased tonus in the muscles responsible for airway patency [12, 32, 34]. The elevated initial negative inspiratory pressure produced by the hypertrophied tonsils and adenoids interacts with the decreased muscle tone to draw the airway inward, causing a further decrease in caliber and creating the need for greater inspiratory pressures. Frequent arousals from sleep which allow muscle tone to return interrupt this cycle.

The role of the hypertrophied tonsils and adenoids in obstructive sleep apnea may be explained by a basic principle of physics. Hagen-Poiseuille's Law states that resistance to flow is proportional to $1/(\text{radius of the canal})^4$. Small changes in the radius of a canal cause marked changes in the resistance to flow through the canal. Marked increases in resistance to airflow follow any narrowing of the airway by lymphoid tissue, or structural diminutions of the airway by craniofacial abnormalities. A large negative inspiratory pressure is necessary to draw air through the patent but narrowed airway. The negative inspiratory pressure tends to draw the walls of the pharyngeal airway even closer together, further increasing inspiratory airway resistance.

Correct determination of tonsillar/adenoidal size, whether judged clinically or radiographically, is vital in the evaluation of children with suspected pediatric obstructive sleep apnea. Tonsillar size is generally appraised subjectively. On lateral neck radiographs, the adenoids may be measured directly and compared to age-matched norms [35–37]. This method fails in children with craniofacial dysplasia; "normal" adenoids can be obstructive in a structurally small nasopharynx. Alternatively, adenoidal size has been expressed in relation to nearby bony landmarks [16, 38]. The measurements to produce a ratio may be cumbersome, or precluded on available radiographs if the technique is too light, making landmarks hard to define, or if over-collimation has excluded any of the necessary landmarks.

Our measurements of the AN ratio of pediatric patients with obstructive sleep apnea and comparison with the normal data of Fujioka et al. [16] represent the first attempt to quantitate the degree of nasopharyngeal lymphoid enlargement in pediatric

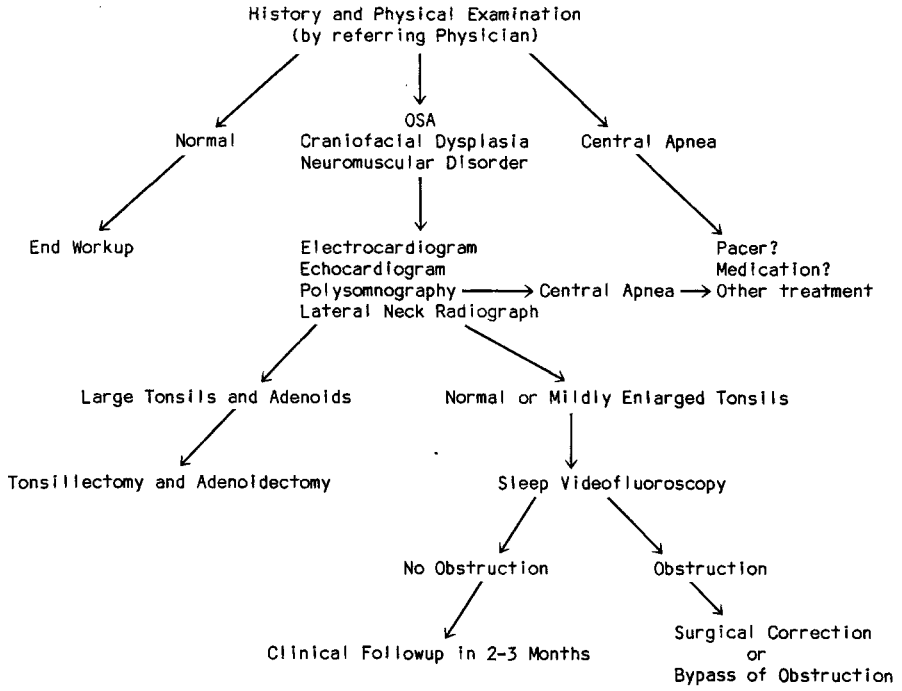

Fig. 7. The evaluation of a child referred with suspected obstructive sleep apnea has evolved during our study and now usually follows the above algorithm

patients with pediatric obstructive sleep apnea. We were surprised that only three of the 26 children with pediatric obstructive sleep apnea had an AN ratio greater than two standard deviations above the mean value, defined as abnormal by Fujioka et al. [16]. It appears that even mild to moderate adenoidal enlargement may be associated with significant sleep-related breathing difficulty. For this reason, we have not made any decisions regarding patient management based on the AN ratio alone. Of note, we have not seen pediatric obstructive sleep apnea due to adenoidal-palate obstruction in any patients with an AN ratio less than 0.5.

Our results indicate that even mild to moderate adenotonsillar enlargement may cause significant breathing difficulties during sleep. Clearly, more data are needed in this area, both to establish normal values for tonsil size in infancy and childhood and to quantitate the degree of adenotonsillar enlargement that results in obstructive sleep apnea. Also the neuromuscular component of sleep apnea needs to be further elucidated.

The present study, as well as previous fluoroscopic studies of smaller numbers of patients [1, 2, 17–19], suggests that the mechanism of pharyngeal collapse and obstruction is complex and may vary from patient to patient. Anatomic factors must be involved because the children get better after tonsillectomy and adenoidectomy. Physiologic factors must also be involved because the problem is sleep related. The forward displacement of the posterior pharyngeal wall during obstructed respirations may be caused by hypotonia of muscles in this area or may be secondary to the increased negative inspiratory pressures. Similarly, hypotonia or elevated inspiratory pressure may be the basis of tonsillar and palatal motion during obstructed respirations. The pharyngeal airway above the point of obstruction is seen to widen because of changes in jaw or palate position. This compensatory dilatation appears to represent the contraction of pharyngeal dilators shielded from and unopposed by negative inspiratory pressure.

Fluoroscopy of the upper airway appears to be quite useful in diagnosing the site of airway obstruction and has considerably expanded our understanding of the pathogenesis of obstructive sleep apnea in infants and children. However, fluoroscopy is not necessary for the clinical management of most pediatric patients with obstructive sleep apnea. In our referral population, when craniofacial abnormalities and primary central nervous system disorders had been excluded and if the tonsils and adenoids were enlarged on lateral airway radiographs, fluoroscopy always has demonstrated obstruction at the adenoidal and/or tonsillar levels. Accordingly, tonsillectomy and adenoidectomy have always been recommended. The majority of pediatric patients with obstructive sleep apnea fit this clinical description: large adenoids and/or tonsils, otherwise normal craniofacial morphology, and an absence of any underlying neurologic disorder [2, 3, 6, 11]. Therefore, fluoroscopy can be safely eliminated from the preoperative evaluation of those pediatric patients with a strong clinical history of respiratory abnormalities

(stridorous respirations, sternal retractions) and an abnormal polysomnographic study, if a standard lateral neck radiograph demonstrates adenoidal and/or tonsillar enlargement.

On the basis of these results, we no longer utilize the AN ratio in evaluating patients referred with suspected pediatric obstructive sleep apnea. All children referred with pediatric obstructive sleep apnea do undergo a polysomnographic study regardless of the subjective grade of tonsillar enlargement. Fluoroscopy is reserved for children whose adenotonsillar enlargement falls into the 0–1 + range, or for children with craniofacial dysplasia, neurological disorders, or prior cleft palate surgery (Fig. 7).

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