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REVIEW

Growth failure and sleep disordered breathing: A review of the literature

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Summary

Objective: While otolaryngologists consider growth failure an absolute indication for tonsillectomy and adenoidectomy (T&A), they may not be accustomed to screening for poor growth, and thus unlikely to consider it when recommending a T&A. This paper will (a) familiarize otolaryngologists with the definition, prevalence, and etiology of growth failure and (b) review the published findings that examine the inter-relationship among sleep disordered breathing, growth failure, and adentonsillar hypertrophy in children.
Methods: This paper is divided into three sections. The first section presents a brief overview of growth failure for the otolayngologist. The second section reviews the evidence base linking sleep disordered breathing, growth failure, and adenotonsillar hypertrophy in children. The anthropometric outcomes of children presenting for T&A, or having sleep symptoms assessed, are presented. The third section presents pilot data (*n* = 28) on the prevalence of growth failure and sleep disordered breathing among children presenting for T&A at our institution.
Results: Among children presenting for T&A or having sleep symptoms assessed, growth failure was at least twice the expected rate in six of eight published studies. Across these six studies, this rate ranged from a low of 6% of children <3rd percentile for weight and 6% <3rd percentile for height in one study, to a high of 52% who were <3rd percentile in weight in a second study, and 44% who were ⩽5th percentile for height in a third. Among children presenting for T&A at our own institution, 14% were ⩽5th percentile in height, and 11% were ⩽5th percentile in weight. Among children under 6 years of age, 21% were either ⩽5th percentile in weight and/or height.

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Conclusions: Published studies, as well as our own pilot data support the hypothesis that SDB, secondary to adenotonsillar hypertrophy increases the risk of growth failure in children. Adenotonsillar hypertrophy and sleep disordered breathing may be unrecognized risk factors in the etiology of growth failure. Otolaryngologists can play an important role in identifying growth failure, and referring children to the appropriate specialists.

Contents

1. Introduction

Evidence starting from the 1980s supports a causal relationship between sleep disordered breathing (SDB) and growth failure (GF) in children [1–4]. For most children, adenotonsillar hypertrophy is the primary factor leading to SDB; tonsillectomy and adenoidectomy (T&A) is curative in 80% of cases [5,6]. Nearly every study finds that otherwise healthy children experiencing GF show significant catch up growth following T&A. The American Academy of Pediatrics identifies GF as a serious complication of untreated obstructive sleep apnea [7].

Yet, primary care providers' differential diagnosis of GF in children does not routinely include an assessment of upper airway obstruction [8–13]. Only half of pediatricians surveyed identify a relationship between poor growth and obstructive sleep apnea (OSA) [14]. Pediatric subspecialties in genetics [15], GI [16,17], and endocrine [18] do not routinely include such an assessment in their differential diagnosis of GF either. This is not surprising, given that the underlying cause of GF is nearly always attributed to under-nutrition, despite multiple biologically plausible pathways from SDB to GF. For over 20 years, there has been an as yet unheeded call for assessment of SDB in children with growth problems [4,19–24].

Otolaryngologists identify GF as an absolute indication for T&A; dysphagia, caused by hypertropic tonsils and/or adenoids is generally cited as the primary causal mechanism [25]. With rare exception [26], the otolaryngology literature does not discuss the potential role of growth hormone [1] and/or

energy expenditure disturbances [27] in SDB-related GF. In contrast, the association between SDB and GF is well documented in the sleep disorders literature [7,21,28]. Furthermore, a survey of otolaryngologists' reasons for performing T&A among school age children, did not present growth disorders as an option, despite 59% of procedures being reported for obstructed breathing of any type and 39% for OSA [25].

This paper is divided into three sections. The first section presents a brief overview of GF for the otolayngologist. In the second section, we review the literature on SDB, GF, and adenotonsillar hypertrophy and present a model of the proposed associations. The third section presents pilot data on the prevalence of both SDB symptoms and GF among children presenting for T&A at our institution.

2. Review of growth failure: definition, prevalence, and etiology

Criteria for GF vary widely. The most common are: weight-for-age ⩽5th percentile; crossing of two major percentile lines; height-for-age ⩽5th percentile, and weight-for height ⩽5th percentile. Nutritional factors are paramount in low weight-for-age and weight-for-height, while endocrine or skeletal growth issues tend to manifest in linear growth (i.e., height-for-age) [18,29]. The prevalence of GF is difficult to estimate, due to differences in definition. Among children under 2 years of age, a population-based screening program in England identified 3% with GF [30]. In the U.S., the

National Health and Nutrition Examination Survey found the proportion of children aged 24—71 months who were ≤5th percentile to be: 4.1% for weight-for-height [31], 5.2% for height-for-age, and 2.7% for weight-for-age [32].

GF in younger children, i.e., 3 years of age and under, is referred to as "failure to thrive" (FTT) [33]. While FTT is used as a clinical label, it is, more aptly, a syndrome in which social or developmental delays may also be present [16]. The relationship between these delays and growth problems is inconsistent and often contingent on etiology [34]. The etiology of FTT is multi-factorial. Historical distinctions between organic and non-organic failure to thrive are now eschewed given what is often mixed etiology. Population-based studies find that <5% of children with FTT have major organic disease [35]. Neither SDB, nor adenotonsillar hypertrophy are commonly cited as an "underlying cause" of GF or FTT. Rather, the *underlying cause of failure to thrive is nearly always attributed* to insufficient usable nutrition due to:

- the child's inability to feed properly/inadequate intake (e.g., poverty, neurological dysfunction, dysphagia);
- inadequate absorption or utilization of adequate nutrition (malabsorption syndromes);
- a disease process that creates added metabolic requirements (e.g., asthma, cardiac failure, thyroiditis) [36].

Psychosocial issues, such as the "will to grow" [29] and deprivation or neglect can affect growth [37,38]. Severe stress or deprivation can affect the rate of linear growth and growth hormone secretion [37]. A revised diagnostic classification system of feeding disorders in infants and toddlers exists [39]. Feeding behaviors of children with FTT may, for example, result in reduced energy intake [40], as a function of an abnormal appetite or energy-intake-regulating mechanism [41]. The overall contribution of behavioral, psychological, and environmental factors to FTT is difficult to discern, and as yet unknown [42].

GF among children older than 3 years of age is most often attributed to normal variation, ascribed to either constitutional growth delay or familiar short stature [43]. Children with constitutional growth delay first evidence reduced growth between 3 and 6 months of age, fall <2S.D. below the mean for height by 3 years of age, and then parallel the normal growth rate while remaining <3rd percentile. Bone age is delayed relative to chronological age [44]. In contrast, familiar short stature is assumed to be genetic as there is decreased mean parental height and no delay in bone age. Whether constitutional growth delay and familiar short stature are distinct conditions has been questioned [45]. Growth hormone or thyroid deficiency is diagnosed through endocrine testing. Growth hormone deficiency among children referred to endocrine clinics has been reported as 14% [46] and 23% [47], while 5% of short stature children (population-based) have endocrine disorders of any type [48].

3. Causal pathway: from sleep disordered breathing to growth failure

Sleep disordered breathing is relatively common in children; parents often seek treatment for snoring, mouth breathing, or OSA. SDB is a pathophysiologic continuum spanning snoring, upper airway resistance syndrome, obstructive hypoventilation, and OSA. While the exact prevalence of SDB in children is unknown, snoring may occur in 3—12%, while OSA may occur in 1—10% [49,50]. Just 20% of pediatricians screen for SDB [51]. OSA peaks at 2—6 years of age, because of the relative adenotonsillar hypertrophy found at this time [52]. OSA is attributable to a combination of AT hypertrophy and neuromuscular tone, rather than structural abnormalities alone [53].

We performed a Medline review of English language studies to identify the incidence of growth failure among children presenting for T&A, or having OSA assessed. We using the following search terms: "failure to thrive," "growth failure," "growth delay," "growth impairment," and "tonsillectomy," "adenoidectomy," "adenotonsillectomy," "adenotonsillar hypertrophy," "tonsillary hypertrophy," "tonsil and/or adenoid," or "polysomnography," Only published studies that provided incidence data on sex adjusted height-for-age or weight-for-age at or below 10% (versus z-score, group percentile, or no data) were selected for inclusion. In all of the studies that we identified, children with chronic conditions or craniofacial conditions were excluded, except one, which included seven dysmorphic infants.

Table 1 summarizes anthropometric data from seven studies of children presenting for T&A (n = 4 for suspected OSA), and one study of children undergoing overnight polysomnography. The incidence of GF was at least twice what would be expected in six of these eight studies. For example, in Selimoglu's study, 10% of children were below the 2.5th percentile in weight-for-age and height-for-age. In Williams' study, nearly half of the children were below the 5th percentile in weight-for-age and

Table 1 GF incidence[a] in children having T&A[b], or OSA symptoms assessed[c]

Study	Inclusion criteria	Age	GF definition	GF incidence
Ahlqvist Rastad[b] [57] (Sweden, n = 122)	Tonsillar obstruction	1.8—15.8 years	Weight <2S.D. (≈2.5%)	<1% (1/122)
Ylmaz[b] (Turkey, n = 41)	Obstructive adenotonsillar hypertrophy	4—8 years	Height <3rd %ile	3% (1/32)
Selimoglu[b] [23] (Turkey, n = 49)	Obstructive adenotonsillar hypertrophy	4—12 years	Weight and height <2S.D. (≈2.5%)	10% Wt and Ht (3/29)
Brouillette[b] [4] (USA, n = 22)	Suspected OSA, on retrospective review	19/22, <5 years old	Weight <10th %ile and "improved" post-op	27% (6/22)
Williams[b] [20] (USA, n = 41)	Suspected OSA, on retrospective review	6—36 months	Weight ≤5th %ile; height ≤5th %ile	46% Wt (19/41); 44% Ht (15/34)
Freezer[b,c] [22] (USA, n = 38)	Suspected OSA, clinical hx	13 months, median	Weight <3rd %ile	52% (15/29)
Li[b] [93] (Hong Kong, n = 35)	Suspected OSA, 2° to adenotonsillar hypertrophy	4—10 years	Weight <3rd %ile; height <3rd %ile	5.7% Wt (2/35); 5.7% Ht (2/35)
Wang[d] (USA, n = 82)	Referred for overnight PSG, due to symptoms suggestive of OSA	18 months—15 years, mean = 6.7 years	Weight <10th %ile	33% Wt (5/15 with GF are OSA); 20% of those w/OSA are GF

[a] Only includes studies w/GF incidence data, vs. z-scores, a group percentile, or no data.
[b] Studies of children presenting for AT surgery.
[c] Included seven dysmorphic infants.
[d] Study of children in whom both GF and OSA symptoms were assessed.

height-for age. Thus, these studies support the hypothesis that SDB, secondary to adenotonsillar hypertrophy increases the risk of GF in children.

Removal of the nasopharyngeal airway obstruction consistently yielded greater than expected gains in weight, and sometimes in height, among children identified with GF [4,19,20,22,23,54], as well as children in whom GF is not identified [3,21,55—58], often allowing children to reach their growth potential. Overall, 13 studies [3,4,19—23,54—59] report pre- and post-T&A weight. In 12 of these 13 studies, post-T&A weight changes (follow-up range = 10 weeks—42 months) were greater than expected. In the 11 studies reporting pre- and post-T&A height [3,19—23,57—60,64], greater than expected increases in height were observed in 8 out of 11 studies. And, in an RCT of T&A that *excluded children with suspected obstructive sleep apnea* there were no differences in height or weight at 6- or 24-month follow-up [60], suggesting that nocturnal upper airway obstruction is a critical link between SDB and GF. The limitations of these studies are that (a) they were not purposefully designed to measure the association between SDB

and GF, (b) small sample sizes preclude controlling for potential confounds, and (c) there were no a priori power analyses.

There are multiple biologically plausible pathways from SDB to GF. In adults, OSA is associated with increased sleep energy expenditure [61]. Marcus found increased caloric expenditure among children with SDB due to labored breathing in overnight monitoring. Children with the lowest weight z-scores had the highest energy expenditures [27]. A comparison of children with OSA versus controls, found a non-significant trend toward greater sleeping energy expenditure among those with OSA [62]. Nocturnal hypoxemia [1,63] and metabolic alkalosis [64] have also been implicated for effects upon growth.

Of all the potential pathways, growth hormone hypotheses have received the most attention. Impaired growth hormone secretion in children with SDB may result from interruptions in slow-wave sleep, when a large proportion of growth hormone is secreted [65]. Compared with controls, growth-impaired children have higher levels of insulin-like growth factor binding protein 3 (IGFBP-3) [65,66]

and lower levels of insulin-like growth factor (IGF-1) [67]. As 70—80% of IGF-1 binds to IGFBP-3, increased IGFBP-3 levels restrict the bioavailability of IGF-1 [67]. Among children who underwent T&A, there are increased post-operative levels of circulating levels of IGF-1 [68,69], IGFBP-3, or both IGF-1 and IGFBP-3 [54,70]. Levels of leptin, a hormone associated with both fatness and bone metabolism during human development [71], are correlated with IGF-1 levels in infants [72] and children [73]. Decreased weight may result in low leptin and IGF-1 levels, and thus short stature [74].

GH deficiency itself may affect sleep architecture. Differences in sleep architecture were observed between children with short stature and GH deficiency, compared with normally growing children [75]. Abnormalities in sleep architecture have also been observed in children without classical GH deficiency [76,77]. GH treatment improved sleep quality by altering sleep architecture in children with psychosocial dwarfism [77], GH deficiency [78], and Smith Magenis syndrome [79], and in adults [80]. There are no systematic studies of SDB in children with GF, nor any comparing short stature children with and without GH deficiency. Second, data on the effect of GH deficiency upon sleep architecture are limited to small case reports [1,76,77,79,80]; only one compares children with and without GH deficiency [1].

In addition, several feeding disorders are direct complications of either SDB or adenotonsillar hypertrophy. SDB can cause neurological dysfunction, which can interfere with a child's feeding ability [81,82]. Feeding fatigue, secondary to adenotonsillar hypertrophy-induced upper airway obstruction may result in inadequate intake and weight gain [83]. Pharyngeal dysphagia from adenotonsillar hypertrophy can elicit pain and obstruct food entry into the esophagus, thereby reducing appetite [82,84]. Leanness is observed in some children with constitutional growth delay, familial short stature,

or idiopathic short stature [85,86]. Not all children with adenotonsillar hypertrophy will experience SDB, and vice-versa.

Fig. 1 presents the proposed model of the relationship between SDB, adenotonsillar hypertrophy, and GF.

4. Pilot study assessing SDB and GF in children presenting for T&A

4.1. Introduction

We conducted a pilot study to assess the prevalence of SDB symptomology and GF, among a sample of children presenting for T&A surgery at an urban children's hospital. Based upon the accumulated literature, we expected that this sample of children — who were not specifically selected for GF — would exhibit greater rates of SDB symptomology compared with the general pediatric population. Two pediatric otolaryngologists collected data from parents/guardian of all children ($n = 28$) undergoing adenotonsillectomy just prior to the surgery from November 4, 2004 to January 6, 2005. After consulting with the affiliated medical center's institutional review board, data collection was permitted to proceed without informed consent. This decision was based upon the activity being considered preparatory to research under HIPAA regulations.

4.2. Methods

In accordance with this being a pilot study, we sought to minimize the burden of data collection both upon the families and the pediatric otolaryngologists. The two pediatric otolaryngologists conducting the assessments recorded the following data, in checklist format, in the hospital's preoperative setting:

- Demographics—DOB and sex.
- Symptoms of sleep disordered breathing—respiratory pauses during sleep (yes/no); snoring (yes/no).
- Health problems—parental concerns about growth (yes/no) and chronic health problems (yes/no).
- Tonsillar obstruction—tonsillar size (1—4) was obtained from the patient chart, according to the criteria of Brodsky, and recorded by the pediatric otolaryngologist at an office visit prior to surgery. Parent report of swallowing difficulty (yes/no) was also recorded.
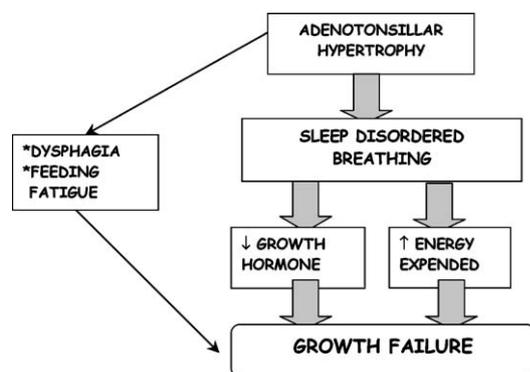

Fig. 1 Theoretical model relating adenotonsillar hypertrophy and SDB to GF.

- Anthropometrics—height and weight data were obtained from the medical clearance form completed by the child's primary care provider up to 1 week prior to surgery, and converted to age- and sex-adjusted weight-for-age and height-for-age percentiles using EpiInfo. Thus, anthropometric assessment was not standardized across children.

Adenotonsillectomy was performed with electrocautery dissection, without microdebrider or coblation. None of the children underwent PSG prior to surgery.

5. Results

Our pilot study data are shown in Table 2. Data are stratified by < and ≥6 years, as 6 years represents the median age, and is the upper age at which adenotonsillar hypertrophy peaks. Parent-reported rates of snoring (96%) and pauses in sleep respiration were high (78%). Also as anticipated, GF rates (defined as ⩽5th percentile in weight and/or height for age) were high, relative to the <5% one would expect to find in the general population. That is, 18% of the entire sample met *either* criteria for GF, as did 21% of children under 6. Seven percent of children met *both* criteria for GF. Obesity is a known risk factor for obstructive sleep apnea; 32% of the sample was obese.

We present the characteristics of the 5 children meeting criteria for GF, compared with the 23 children not identified as having GF. As shown in

Table 3 Characteristics of GF+ vs. GF-children

	GF+ (n = 5)	GF− (n = 23)
Tonsil size = 4, % (n)	80 (4)	17 (4)
Pauses in sleep respiration, % (n)	80 (4)	78 (18)
Difficulty swallowing, % (n)	40 (2)	0 (0)
Snoring, % (n)	80 (4)	100 (23)
Parent concerns re: growth, % (n)	60 (3)	9 (2)

Table 3, a greater proportion of children with GF had tonsil size = 4, difficulty swallowing, and parent reported growth-concerns.

6. Discussion

Sleep disordered breathing, which spans a continuum from primary snoring to obstructive sleep apnea, is a risk factor for growth failure in children. This paper discusses the prevalence and definition of growth failure for the otolaryngologist. Several potential causal mechanisms linking SDB and GF are discussed. Data from a pilot study assessing both SDB symptoms and GF in children presenting for T&A are presented. These findings support our view that otolaryngologists can play an important role in identifying GF, and referring children to the appropriate specialists.

Otolaryngologists are familiar with the etiology and sequelae of upper airway obstruction in children. However, otolaryngologists may not always recognize the risk that co-morbid adenotonsillar hypertrophy and SDB pose for growth failure. Chil-

**Table 2** Assessment of SDB and GF in children prior to T&A surgery

	Total sample (n = 28)	<60 months old (n = 14)	≥60 months old (n = 14)
Age range (mean), years	2.3—16.6 (7.5)	2.3—5.7 (4.19)	6.0—16.6 (10.75)
Child snores (yes)	96% (27/28)	100% (14/14)	93% (13/14)
Difficulty swallowing (yes)	7% (2/28)	7% (1/14)	7% (1/14)
Parent concerns regrowth (yes)	18% (5/28)	21% (3/14)	14% (2/14)
Pauses in sleep respiration (yes)	79% (22/28)	93% (13/14)	64% (9/14)
Chronic health prob/anomaly (yes)	14% (4/28)	21% (3/14)	7% (1/14)
Tonsil size			
2	11% (3/28)	21% (3/14)	0% (0/14)
3	61% (17/28)	50% (7/14)	71% (10/14)
4	29% (8/28)	29% (4/14)	29% (4/14)
Ht-for-age ⩽5th %ile	14% (4/28)	14% (2/14)	14% (2/14)
Wt-for-age ⩽5th %ile	11% (3/28)	14% (2/14)	7% (1/14)
Both Ht- and Wt-for age ⩽5th %ile	7% (2/28)	7% (1/14)	7% (1/14)
Growth failure (GF+) (Ht-for-age and/or Wt-for-age ⩽5th %ile)	18% (5/28)	21% (3/14)	14% (2/14)
BMI ≥95th %ile	32% (9/28)	29% (4/14)	36% (5/14)

dren with co-morbid GF and SDB are at increased risk for the developmental, behavioral, and/or neurocognitive sequelae of untreated SDB. Up to 50% of young children with GF are never identified [87–89] even by experienced clinicians [90].

The data from our small sample are congruent with our hypothesis that SDB secondary to adenotonsillar hypertrophy may be associated with GF. The prevalence of both GF and SDB in our pilot study of children about to undergo T&A was high. Defining GF as ≤5th percentile in weight and/or height for age, one would expect <5% of the sample to meet criteria for GF. Instead, 18% of the entire sample met *either* criteria for GF, as did 21% of children under 6. Thus, these data are in the mid-range of data

disorders (e.g., insomnia) versus SDB, and (b) had not been validated by nocturnal polysomnography [92].

Both the American Thoracic Society [28] and the American Academy of Pediatrics [7] have called for research on the risk factors for growth failure, including the prevalence of growth failure among children with sleep disorders. Future research should identify the prevalence of co-morbid SDB and GF in children from primary care sites, and in children referred to GI and endocrine specialists, compared with controls. Ideally, both groups would undergo overnight polysomnography. Such research would enable us to begin identifying the extent to which adenotonsillar issues contribute to growth failure.

Growth Failure (GF+) in ENT Sample
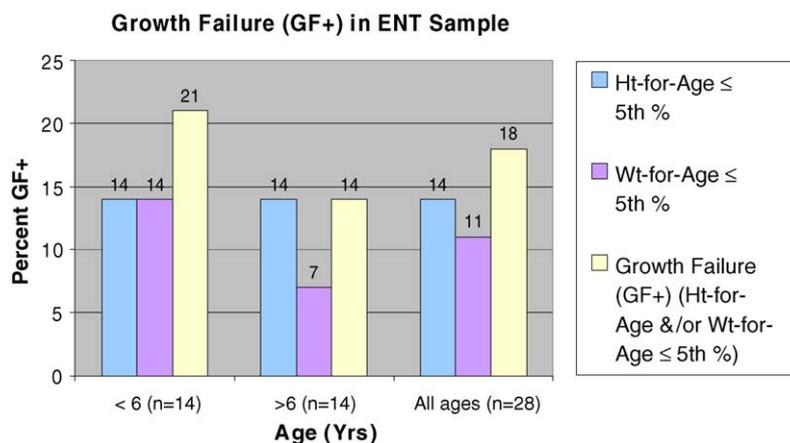
presented by others. Seven percent of children met *both* criteria for GF. Obesity is a known risk factor for obstructive sleep apnea; 32% of the sample was obese. Rates of snoring (96%) and pauses in sleep respiration were high (78%).

While anthropometric data for children with suspected SDB and/or presenting for T&A have been published, just one study examines the incidence of SDB in children selected for short stature. Parents of 33% (53/158) of children retrospectively identified from endocrine clinic records completed a sleep disorders screening questionnaire. Of these, 27% (15/53) met criteria for SDB and were referred for overnight polysomnography. However, none of the 10/15 who agreed to polysomnography showed evidence of nocturnal respiratory disturbances [91]. This study has several limitations. The low response rate may have resulted in selection bias—just 6% of the original sample underwent nocturnal polysomnography. Furthermore, the screening questionnaire used for referral (a) primarily assessed other sleep

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