Obstructive Sleep Apnea and Weight Abnormalities in Children

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Abbreviations

AHI	Apnea-hypopnea index
AT	Adenotonsillectomy
ATH	Adenotonsillar hypertrophy
BIA	Bioelectrical impedance analysis
Bi-PAP	Bi-level positive airway pressure
BMI	Body mass index
CPAP	Continuous positive airway pressure
cwp	Centimeters of water pressure
EEG	Electroencephalographic
EMG	Electromyogram
FTT	Failure to thrive
GF	Growth failure
GFBP-3	Growth factor binding protein
GH	Growth hormone
IGF-1	Insulin-like growth factor
OSA	Obstructive sleep apnea
OSDB	Obstructive sleep-disordered breathing
TcPCO2	Transcutaneous CO2 level

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Case 1: Toddler Presenting with Failure to Thrive

History

A 20-month-old full-term male presented to the emergency department (ED) with 2 days of cough and congestion. His parents reported trouble breathing and choking episodes while sleeping as well as difficulties taking solid foods. His oral intake started declining approximately 4–5 months ago. He coughed and choked with attempts to eat solids, and therefore had to be fed with pureed/mashed or baby foods. He has been able to drink liquids and takes 5–6 large bottles of whole cow's milk daily in addition to apple juice and water. The parents believe he has lost weight over this time, but they are unsure of the exact amount. There is reported noisy breathing that is worse when going to bed at night. The family feels as though they need to observe him throughout the night to make sure he won't stop breathing. They routinely prop him up but feel his breathing is very shallow as though "he is using his whole body to breathe." Parents also commented on loud snoring and jerking movements during sleep. Further evaluation in the ED revealed unremarkable chest X-ray and two-view airway X-ray, which were significant for the prominence of the palatine tonsils and adenoid hypertrophy.

Past Medical and Surgical History

The child's past medical history was remarkable for sickle cell trait and constipation.

Medications and Allergies

There were no reported food or medication allergies or need for medication use.

Family History and Social History

Family history was remarkable for myocardial infarction in maternal aunt who died at age 4 and multiple family members affected by obstructive sleep apnea.

Review of Systems

Review of medical systems was unremarkable.

Physical Examination

Height: 80 inches
Weight: 22 pounds

The child appeared to be malnourished and had mild respiratory distress with suprasternal and intercostal retraction. Copious nasal secretions and considerable tonsillar hypertrophy were noticed on oropharyngeal examination. His exam was otherwise unremarkable.

Differential Diagnosis

- Inadequate caloric intake (neglect, behavioral problems associated with decreased oral intake such as oral aversion or dysphagia)
- Inadequate nutrient absorption (celiac disease, cystic fibrosis)
- Increased metabolism (hyperthyroidism, congenital heart disease, obstructive sleep apnea/sleep-disordered breathing)
- Defective utilization (genetic disorders, metabolic disorders, others)

The child's nutritional and gastrointestinal review of systems were consistent with an appropriate oral intake and no symptoms suggestive of malabsorption. Absence of dysmorphism and unremarkable family history made genetic disorder less likely. The presence of significant tonsillar hypertrophy and observed sleep-related apneas and dyspnea were suggestive of obstructive sleep-disordered breathing.

Diagnostic Testing

A pediatric polysomnography was obtained that demonstrated frequent brief oxygen desaturations to 70%, dyspnea, apneas, and rising level of transcutaneous carbon dioxide (TcPCO2) to 58–60 mmHg noted during the child's sleep (Fig. 12.1). Laboratory data revealed normal complete blood count and an unremarkable basic metabolic panel. The child tested positive for Metapneumovirus virus.

Diagnostic Polysomnogram (PSG):

Total sleep time:	315 minutes
Latency to sleep:	2 minutes
REM latency:	40 minutes
Sleep efficiency:	88%
Apnea-hypopnea index (AHI):	88 events per hour of sleep
REM AHI:	120 events per hour of sleep
Mean sleep % SpO2:	96%
Min sleep % SpO2:	75%
TcPCO2 > 50 mmHg:	35% of total sleep time

Assessment

The patient's acute respiratory distress was likely due to metapneumovirus bronchiolitis. His chronic symptoms of snoring, nocturnal choking episodes, and noisy

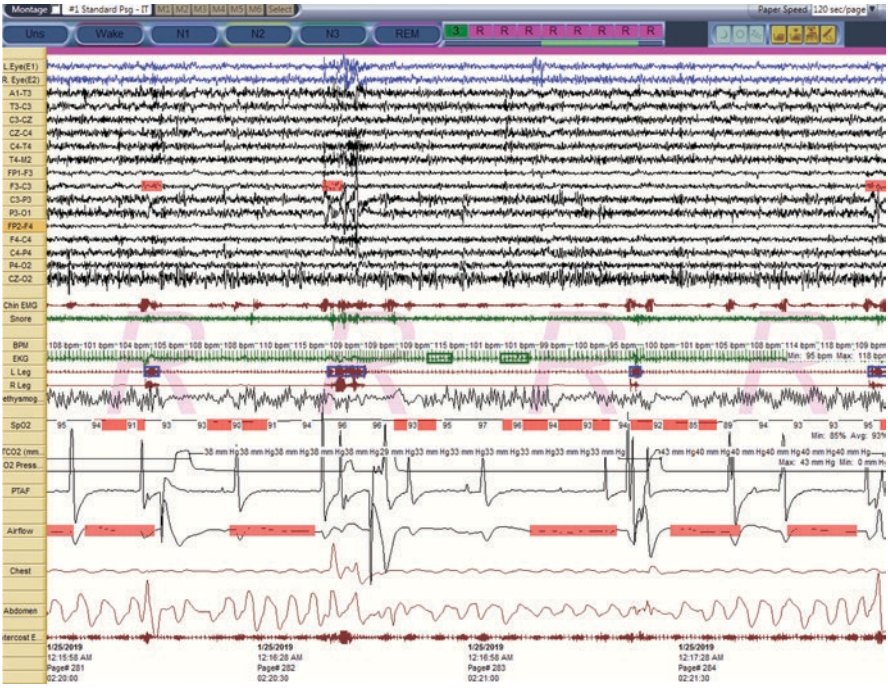


Fig. 12.1 Obstructive apneas in REM sleep associated with periodic oxygen desaturations, 120-second window
L Eye and R eye/EOG – electrooculogram; A1-T3, T3-C3, C3-CZ, CZ-C4, C4-T4, T4-M2, CZ-O2/EEG – electroencephalogram; chin EMG – chin electromyograms; Snore – sound channel; BMP and ECG – electrocardiogram; L-leg and R-leg – left and right anterior tibial EMG; Plethysmog – Plethysmography; SpO2 – oxyhemoglobin saturation by pulse oximetry; ETCO2 – end tidal carbon dioxide; PTaF – nasal pressure; Airflow – thermistor derived oronasal flow; Chest – chest respiratory excursion; Abd – abdominal respiratory excursion; Intercost E – intercostal myogram

breathing were attributed to a new diagnosis of severe OSA associated with profound gas exchange abnormalities, acute on chronic hypercapnic respiratory failure, ATH, and failure to thrive (FTT).

Diagnosis: Pediatric OSA associated with growth failure

Treatment

The patient was started on CPAP therapy at 13 cmH2O, which resulted in resolution of hypercapnia, hypoxia, snoring, dyspnea, and sleep consolidation. The patient was discharged home with CPAP at 13 cmH2O. He was able to tolerate CPAP therapy for a month. Adenotonsillectomy (AT) was performed 1 month later following the resolution of bronchiolitis symptoms. At a follow-up visit 3 months after initial

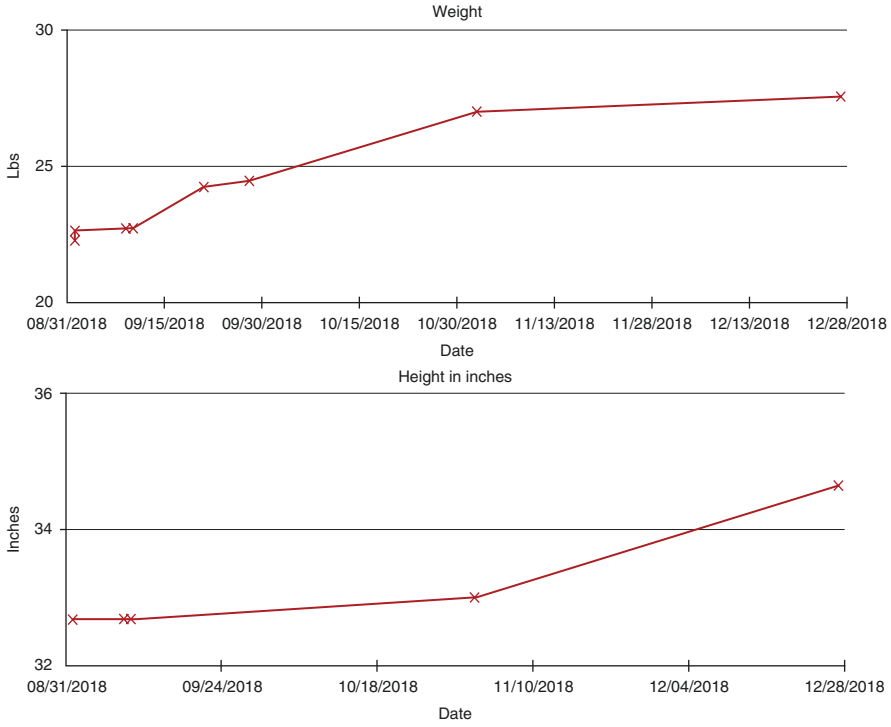


Fig. 12.2 Patient's growth chart demonstrates a proportional increase in weight and height since the administration of continuous positive airway pressure (CPAP) therapy in 9/2018 and following AT in 11/18

admission and 2 months post AT, the family reported complete resolution of sleep apneas, nocturnal dyspnea, restlessness, and snoring. There was 5.5 pound weight gain and 2 inch height increase (Fig. 12.2). The child's oral intake improved dramatically, though he continued working with speech therapy. Repeat sleep study demonstrated complete OSA resolution (Fig. 12.3).

Discussion

This case illustrates a well-known complication of OSA: failure to thrive. Normal growth is the result of a complex interaction between genetic, hormonal, and environmental/nutritional factors. OSA is one of the important factors frequently omitted from the differential diagnosis of FTT by pediatricians, otolaryngologists, family physicians, and endocrinologists.

Growth failure (GF) and failure to thrive (FTT) are defined as inadequate growth in early childhood. Both entities are currently described solely based on anthropometrical measurements, primarily based on the child's weight and height. Although

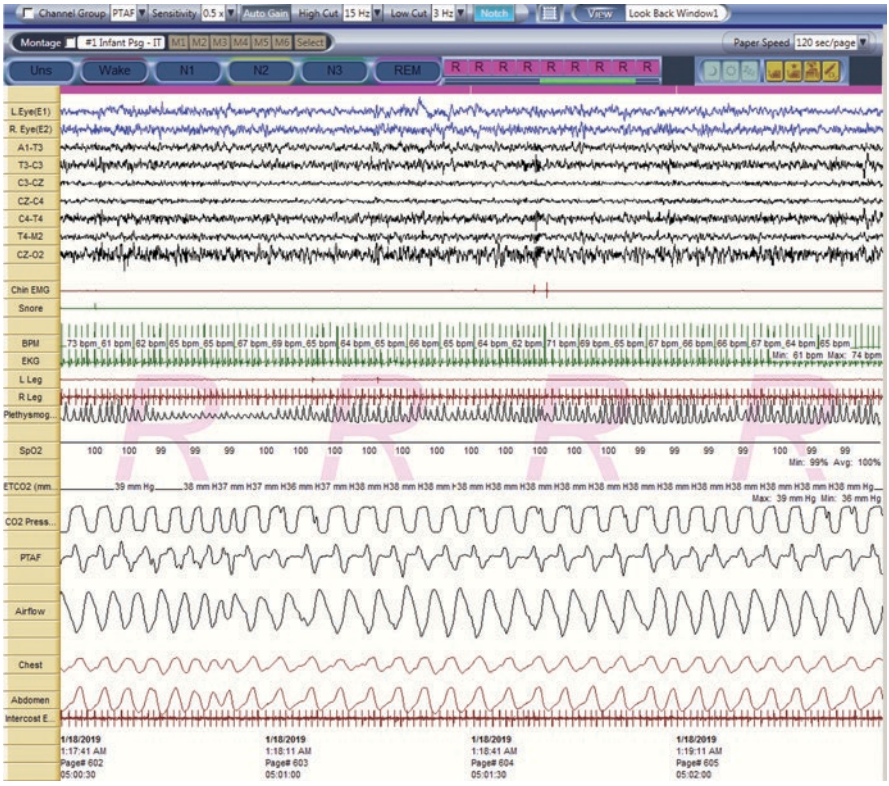


Fig. 12.3 Resolution of REM sleep-related obstructive events, hypoxia, and hypercapnia following AT, 120-second window
L Eye and R eye/EOG – electrooculogram; A1-T3, T3-C3, C3-CZ, CZ-C4, C4-T4, T4-M2, CZ-O2/EEG – electroencephalogram; chin EMG – chin electromyograms; Snore – sound channel; BMP and ECG – electrocardiogram; L-leg and R-leg – left and right anterior tibial EMG; Plethysmog – Plethysmography; SpO2 – oxyhemoglobin saturation by pulse oximetry; ETCO2 – end tidal carbon dioxide; PTaF – nasal pressure; Airflow – thermistor derived oronasal flow; Chest – chest respiratory excursion; Abd – abdominal respiratory excursion; Intercost E – intercostal myogram

there is no universal consensus on the definition of childhood FTT, the term is frequently used for infants and children with weight below the fifth percentile for their sex and corrected age [88]. Supporting definitions include weight for length below the fifth percentile, body mass index for age below the fifth percentile [17], or a sustained decrease in growth velocity in which weight for age or weight for length/height falls by two major percentiles over time [88]. Thus, valid weight and height measurements over time are required for the recognition of failure to thrive [38]. In the case, serial measurements of the child's weight and height were not available; however, the patient's weight and height upon presentation to the hospital were <5% for age.

The prevalence of FTT depends on the population studied and recognition criteria used. It is estimated that in the United States, FTT may occur in up to 5–10% of children [64, 68, 88].

Pediatric OSA was formally recognized in 1976 [34]. OSA is most prevalent in younger children ages 2–8, when tonsil and adenoid size is largest relative to the upper airway diameter [58]. Historically OSA has been most reported in young children, thus many original reports published in 1980s described correlation between severe OSA, adenotonsillar hypertrophy (ATH), and growth failure (GF) in young children [8, 15, 25]. Guilleminault et al. in 1981 reported a "spectacular increase in weight" as well as height increase 2 months after AT, particularly in children with FTT and OSA. Since then, multiple publications confirmed an association between OSA, ATH, and FTT, mostly in children younger than age 10 [13, 19, 24, 37, 46, 54, 59, 63, 77].

The underlying etiology of growth retardation in OSA is still not fully understood. Commonly proposed mechanisms include dysphagia due to hypertrophy of tonsils and adenoids, alterations in taste and poor appetite [78], dysregulation of energy supply/energy expenditure balance due to the increased work of breathing during sleep [10, 56, 59, 78], and nocturnal growth hormone (GH) secretion disruption [7, 13, 14, 67, 80, 81]. Additional proposed mechanisms include intermittent hypoxia associated with OSA, which may result in metabolic compensation required to maintain adequate growth; OSA-related behavioral alterations (e.g., hyperactivity) associated with potentially increased caloric demand; and chronic inflammation causing the growth-inhibiting effects [47]. To date, none of these mechanisms have been conclusively proven to be a cause for FTT in children with OSA. For instance, one pediatric study by Marcus et al. found increased caloric expenditure among children with OSA due to labored breathing. In their study, children with the lowest weight had the highest energy expenditures [59]. However, other studies did not detect a significant difference in sleep energy expenditure between children with and without OSA [6, 10, 56].

Impairment in GH production has been considered the main mechanism underlying growth impairment in children with OSA [6, 7, 13, 58]. GH is secreted in a pulsatile, circadian rhythm that peaks at night, mainly during deep slow-wave or stage N3 sleep [12, 26, 27]. GH production stimulates production of insulin-like growth factor (IGF-1), the major mediator of somatic growth, in the liver and other target tissues [82]. IGF-1 then binds to growth factor binding protein (GFBP-3) [11, 28, 40]. Most IGF-1 binds to IGFBP-3; therefore, increased IGFBP-3 levels restrict the bioavailability of IGF-1 leading to GH deficiency [57]. Compared to controls, children with growth failure have lower levels of IGF-1 [57] and higher levels of IGFBP-3 [45, 66]. Interestingly, IGF-1 increases significantly with postoperative OSA resolution [7, 14, 63]. There are still unanswered questions surrounding the issue of FTT, GH production, OSA, and deep sleep disruption. Deep slow wave sleep complete disappearance, as well as a moderate reduction in REM sleep, was reported by some publications [22, 36]. Yet, later studies have not discovered any changes in the amount of deep sleep in children with OSA before and after OSA resolution [47, 59]. It appears that major OSA-related sleep disruption in children typically occurs outside of stage N3 sleep and is more common in REM sleep [22, 30, 61]. Therefore, the absence of deep sleep disruption when GH is mainly released in a pulsatile manner cannot explain impairment in GH production.

Key Learning Points

1. Obstructive sleep apnea (OSA) is a common problem among children and is recognized as a cause of significant medical morbidity.
2. Growth failure (GF) is a serious complication of younger children with OSA. The most common cause of GF in younger children with OSA is adenotonsillar hypertrophy (ATH). Children with GF should be screened for OSA and referred for possible adenotonsillectomy.
3. Etiology of GF in young children with OSA remains elusive, and further research is needed to determine the cause and identify children at risk.

Case 2: Loud Snoring, Poor Sleep Quality, and Daytime Sleepiness in an Obese Teenager

History

A 14-year-old male with morbid obesity but no other known medical problems presented for evaluation of nightly loud snoring, observed pauses in breathing, snorts, gasps, frequent nocturnal awakenings, nocturnal enuresis, occasional sleepwalking, morning headaches, and daytime sleepiness. He was referred for evaluation by an ENT specialist who diagnosed the patient with adenoid hypertrophy. ENT did not recommend adenoidectomy and referred him to the sleep medicine department for further evaluation and potential CPAP therapy. The patient denied any further sleep-related symptom, such as insomnia, restlessness at night, or abnormal behaviors or movements such as sleepwalking or sleep talking. He did not report any similar breathing concerns during the daytime, although he did report that he had difficulty performing exercises in his physical education class and would become short of breath if he had to perform any exertional activity. He also reported persistent sleepiness during the daytime despite an adequate amount of sleep time. This was unchanged on weekends and on vacations or holiday breaks from school. He did not have any symptoms of sleep paralysis or cataplexy.

Differential Diagnosis

- Sleep-disordered breathing including primary snoring, obstructive sleep apnea, obesity hypoventilation syndrome, and central sleep apnea
- Gastroesophageal reflux
- Nocturnal asthma
- Psychiatric illnesses including panic attacks
- Nocturnal seizures

Sleep Schedule/Sleep Hygiene

The patient sleeps alone in his bed and tends to use his smart phone in the 30 minutes prior to sleep. He usually falls asleep on his side but tends to change position over the course of the night. He believes he wakes up more often when he is on his back.

Time in bed:	9:00 pm
Lights out:	9:15 pm
Sleep onset latency:	5 minutes or less
Number of awakenings:	3–4 per night, usually due to breathing events
Wake after sleep onset time:	10–15 minutes
Wake time:	7:00 am
Naps:	None
Total sleep time:	Approximately 9–10 hours per night

Scales/Questionnaires

STOP-BANG Scale:	5 points (high risk of obstructive sleep apnea)
Epworth Sleepiness Scale:	14 points (excessive daytime sleepiness)

Past Medical and Surgical History

- None

Allergies and Medications

- None

Family History

- Hypertension, mother and father
- Depression, father
- Obstructive sleep apnea, father, paternal grandfather

Social History

He is currently in the ninth grade in high school and reports enjoying school. His grades have recently started to suffer due to excessive sleepiness during the day. He finds himself consuming more caffeine in the last year, and usually drinks 2–3 cups of coffee on weekdays to help him stay awake. He does not use tobacco, drink alcohol, or use other recreational drugs.

Physical Examination

Vital Signs

Blood pressure:	132/70 mmHg
Heart rate:	78 beats per minute
Respiratory rate:	14 breaths per minute
Height:	5 feet 6 inches
Weight:	255 pounds
BMI:	40 kg/m^2

Physical Exam

General:	Obese body habitus. No apparent distress.
HEENT:	Mallampati Class III. Neck circumference 17 inches. Mild tonsillar hypertrophy. Funduscopic examination is normal, normal range of motion. No nasal flaring, no swelling of the turbinates, and no septal deviation.
Respiratory:	Clear to auscultation bilaterally, no wheezing/rhonchi/crackles
Cardiovascular:	Regular rate and rhythm, no murmur, rubs, or gallops. No carotid bruit.
Extremities/Skin:	No cyanosis, clubbing, or edema. Acanthosis nigricans in skin folds and multiple abdominal striae.
Neurologic:	Alert and fully oriented. Cranial nerves II-XII are intact. Motor strength 5/5 in upper and lower extremities. Sensory exam intact to light touch, pinprick, and vibration. Reflexes 2+ throughout, normal gait.

Differential Diagnosis

This patient's normal neurological exam, negative neurological history, and normal review of systems as well as associated snoring and obese body habitus made central apnea diagnosis less plausible. Absence of frequent or chronic cough, wheezing, and dyspnea, or previous inhalers use made a diagnosis of asthma unlikely. Psychiatric history was not consistent with anxiety or nocturnal panic attacks. Morbid obesity, male gender, large neck circumference, crowded airways, and family history positive for OSA were suggestive of obstructive sleep apnea, possibly associated with obstructive hypoventilation and/or obesity hypoventilation syndrome.

Thus, at this point an in-lab sleep study with PaCO2 monitoring was ordered to formally evaluate for sleep-disordered breathing.

Diagnostic Testing

Diagnostic Polysomnogram (PSG):

Total sleep time:	345 minutes
Latency to sleep:	7.5 minutes

REM latency:	87 minutes
Wake after sleep onset:	24.5 minutes
Sleep efficiency:	76.1%
Apnea-hypopnea index (AHI):	83 events per hour of sleep
REM AHI:	115 events per hour of sleep
Mean sleep % SpO2:	97%
Min sleep % SpO2:	70%
End-tidal CO2:	28% of total sleep time at EtCO2 > 50 mmHg
Periodic limb movement index (Fig. 12.4):	3.5 limb movements per hour

Assessment

The patient's clinical history coupled with his polysomnography is consistent with severe OSA, associated with significant nocturnal hypoxemia and hypoventilation. In addition, his clinical exam is suggestive of morbid obesity and insulin resistance, both of which are likely related to the sleep-disordered breathing.

Diagnosis: Pediatric severe OSA associated with obesity

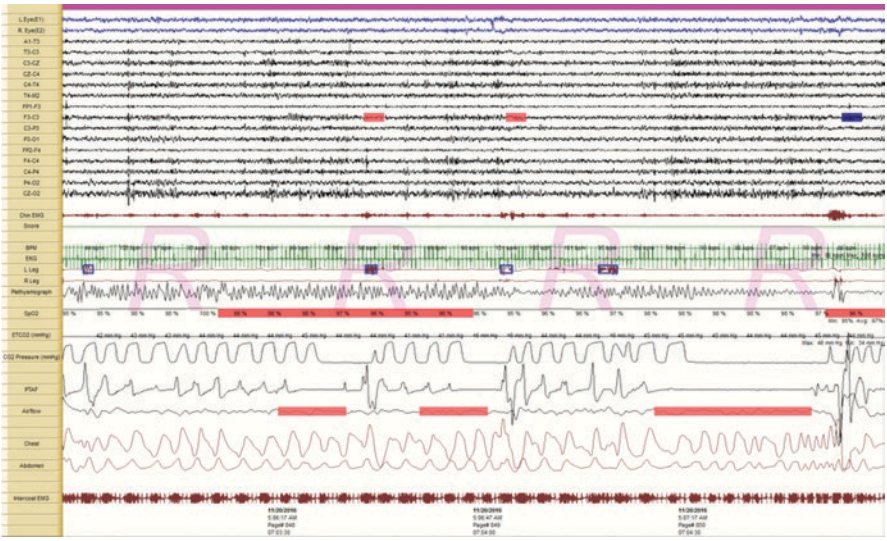


Fig. 12.4 Obstructive events in a 14-year-old child with morbid obesity, 120-second window. Red horizontal bars represent obstructive apneas in REM sleep
L Eye and R eye/EOG – electrooculogram; A1-T3, T3-C3, C3-CZ, CZ-C4, C4-T4, T4-M2, CZ-O2/EEG – electroencephalogram; chin EMG – chin electromyograms; Snore – sound channel; BMP and ECG – electrocardiogram; L-leg and R-leg – left and right anterior tibial EMG; Plethysmog – Plethysmography; SpO2 – oxyhemoglobin saturation by pulse oximetry; ETCO2 –end-tidal carbon dioxide; PTaF – nasal pressure; Airflow – thermistor derived oronasal flow; Chest – chest respiratory excursion; Abd – abdominal respiratory excursion; Intercost E – intercostal myogram
This baseline sleep study demonstrated severe OSA, with evidence of nocturnal hypoventilation and hypoxemia.

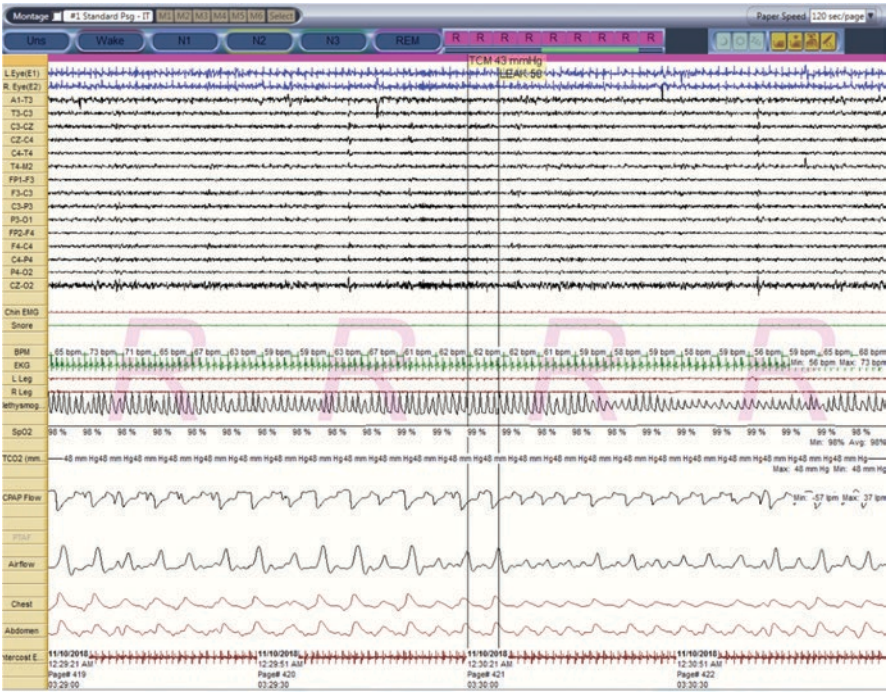


Fig. 12.5 Resolution of obstructive events, hypoxia, and hypercapnia with application of Bi-level PAP therapy at 16/11 cwp, 120-second window
L Eye and R eye/EOG – electrooculogram; A1-T3, T3-C3, C3-CZ, CZ-C4, C4-T4, T4-M2, CZ-O2/EEG – electroencephalogram; chin EMG – chin electromyograms; Snore – sound channel; BMP and ECG – electrocardiogram; L-leg and R-leg – left and right anterior tibial EMG; Plethysmog – Plethysmography; SpO2 – oxyhemoglobin saturation by pulse oximetry; ETCO2 – end-tidal carbon dioxide; PTaF – nasal pressure; Airflow – thermistor derived oronasal flow; Chest – chest respiratory excursion; Abd – abdominal respiratory excursion; Intercost E – intercostal myogram

Treatment

The patient underwent a Bi-level titration study with successful OSA control at Bi-level pressure 16/11 cmH2O (Fig. 12.5). BPAP therapy at 16/11 cmHO was ordered and the patient was referred to a weight management program. However, during subsequent visits to the Sleep Medicine Clinic, the patient was noticed to have continuous weight gain despite consistent use of the BPAP device and appropriate control of OSA with Bi-level PAP therapy (Fig. 12.6).

Discussion

The pediatric OSA profile has changed over the last two decades. Obesity rather than FTT became frequently reported in relationship to OSA. Earlier studies

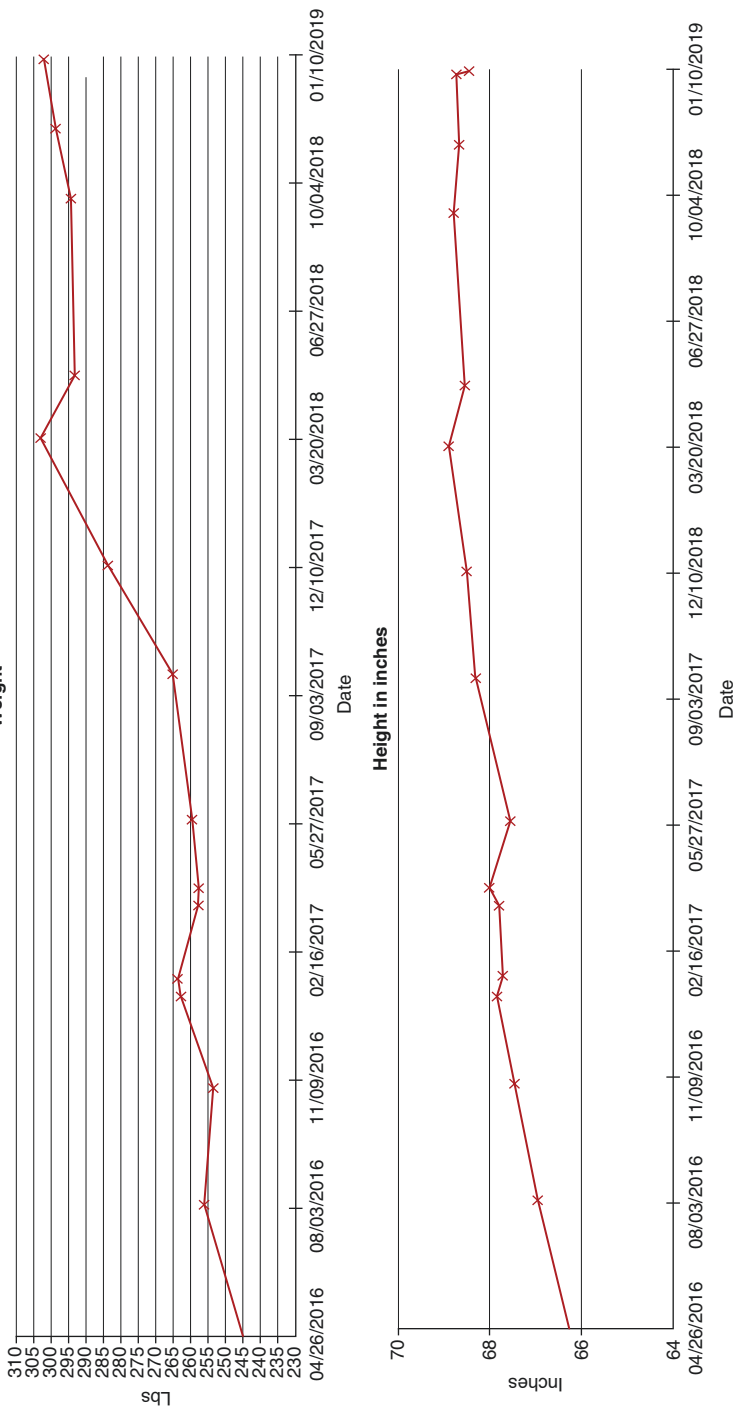


Fig. 12.6 Continuous weight gain despite consistent use of Bi-level PAP device and appropriate control of OSA with Bi-level PAP therapy

reported more frequent OSA association with GF. For instance, a 1992 retrospective study found that of 34 patients with OSA confirmed by polysomnography, only four were obese, whereas 15 weighed under the tenth percentile [55]. In the mid-2000s the relationship between weight and OSA changed due to the rise of pediatric obesity in the United States, with a noticeable increase in childhood obesity occurring at progressively younger ages [48, 86]. This change in dynamics was well documented in the study by Gozal et al. in which less than 15% of all symptomatic habitually snoring children were obese (body mass index or BMI > 95% percentile for age and gender) in the early 1990s, but more than 50% of children referred to the sleep center for suspected SDB in early 2000s fulfilled the criteria for obesity [33]. Subsequently there has been a shift from the classic presentation of children with OSA, ATH, and FTT to children with obesity with and without ATH. This epidemiological change triggered an intense interest in exploring the relationship between OSA and weight gain and OSA and obesity in particular [20].

Obesity is defined as a BMI at or above the 95th percentile for children and teens of the same age and sex; being overweight is defined as a BMI at or above the 85th percentile and below the 95th percentile for children and teens of the same age and sex. BMI is calculated as weight in kilograms divided by the height in meters squared (CDC website [16]). BMI is a simple yet imperfect tool for evaluation of obesity. It does not distinguish excessive weight due to abundance of fat mass from being overweight due to excess lean mass. BMI is the most commonly used measure for assessing obesity in adults. Despite the likelihood of misclassification of the small percentage of individuals whose high BMI is due to lean muscle mass (e.g., athletes), the vast majority of individuals with high BMI have excess body fat. BMI estimation in children is more complicated as an individual child's BMI score has to be compared to the BMI of other children of the same age and gender. The BMI-for-age reference in the United States is based on nationally representative data from boys and girls ages 2–20 years collected between 1963 and 1980 available through the CDC website (CDC website). Morbidly obese children's BMI could not be plotted on the standard CDC BMI percentile chart because their BMI points were above the chart cutoff. Conversely, children with low BMI were also difficult to track on the standard percentile chart. BMI *z*-scores, also called BMI standard deviation scores, were later introduced to measure relative weight adjusted for child age and sex. Z-scores are particularly useful to monitor changes in patients with extreme BMIs, that is, BMI above the 99th percentile or below the first percentile. A z-score describes how far a child's BMI is from the population mean for his/her age and sex, expressed as a multiple of the population standard deviation. The value of a z-score can be negative or positive depending on whether a child's BMI is smaller or larger than the population mean for his/her age and sex. The further a child's BMI is away from the population mean for his/her age and sex, the larger the absolute value of his/her z-score [5]. Most early studies [14, 27, 55] reporting association between OSA and weight abnormalities focused on children's weight and height; however, recent studies also take into consideration BMI *z*-scores [24, 47, 63, 90]. Obese

children are at a higher risk for OSA as the prevalence of OSA among obese children and adolescents can be as high as 60% [85]. The severity of OSA seems to be proportional to the degree of obesity [43, 69, 76]. At any level of OSA severity, the likelihood of excessive daytime sleepiness for obese children is greater when compared to non-obese children [20]. Many obese children with OSA have ATH [74]; however, the pathophysiology of OSA in obese children is complex and cannot be explained by ATH alone. Other proposed mechanisms include genetic predisposition, race [71], increased deposition of fat in the parapharyngeal fat pads near and within the soft palate contributing to airway obstruction (although, no consistent relationship between measures of fat distribution and pediatric OSA in children has been found to date) [84], local upper airway inflammation and mucosal swelling from recurrent vibration, and elevated levels of inflammatory cytokines. Other contributing factors include systemic inflammation, elevated leptin level, lower lung volumes, increased airway collapsibility, and gas exchange abnormalities [9, 46]. There appears to be a reciprocal relationship between OSA and obesity in adults [42]. In obese adults, OSA causes dysregulation of hunger/satiety-regulating hormones, sleep disruption, fatigue, and sleepiness as well as behavioral changes such as poor dietary choices and lack of physical activity, as well as an alteration of energy balance between energy intake and energy expenditure [42, 72]. There is a considerable similarity between adult OSA and pediatric OSA profiles. As in adults with OSA, pediatric OSA with obesity is associated with insulin resistance, hypertension, and an increase in inflammatory markers [9, 50]. There is now a plethora of evidence suggesting that OSA in adults contributes to or exacerbates cardiovascular disease, especially in the context of obesity [42, 65]. Although long-term data on childhood obesity and OSA-related effects on cardiovascular structure and function are currently not available, data from short-term studies focusing on blood pressure regulation, cardiac function, autonomic dysfunction, and endothelial properties suggest a similar pattern in obese children and obese adults with OSA [4, 23, 73, 79, 83]. Therefore, some investigators speculated that OSA associated with obesity is a different entity than OSA associated with FTT and subsequently proposed to divide OSA in children into type I (OSA without associated obesity) and type II pediatric OSA (OSA with associated obesity) [20, 50]. As opposed to children with ATH and FTT, obese children with OSA may not have adenotonsillar hypertrophy, often present at a slightly later age, and are more likely to have a clinical presentation resembling the adult OSA phenotype [32]. There has been accumulating evidence of metabolic sequelae of OSA and obesity in children such as insulin resistance and diabetes [18, 49], dyslipidemia [18, 75], cardiovascular morbidity including hypertension [3, 35, 39, 60], endothelial dysfunction [31], right ventricular hypertrophy [21], and left ventricular remodeling hypertrophy [4] as well as systemic inflammation [9]. These complications resemble findings in adults with OSA and are now recognized as important public health issues.

Adenotonsillectomy and Weight Gain

AT is recommended as the first step in the management of pediatric OSA in both non-obese and obese children with ATH by the American Academy of Pediatrics

[1]. In the United States, the number of tonsillectomies has actually declined significantly since the 1970s. In the past, approximately 90% of AT in children were performed for recurrent infection; now 80% of AT are performed for obstructive sleep problems (American Academy of otolaryngology-head and neck surgery).

The first documented case of improved growth after AT in a child with FTT was reported in 1893 [41]. Since then, there have been multiple reports demonstrating postsurgical weight gain and GF resolution in children undergoing AT [13, 19, 25, 37, 47, 70]. Interestingly, the research suggests that AT can not only lead to normalization of weight in children with GF but also increases the risk of obesity in overweight and obese patients [13, 15, 47, 78, 83].

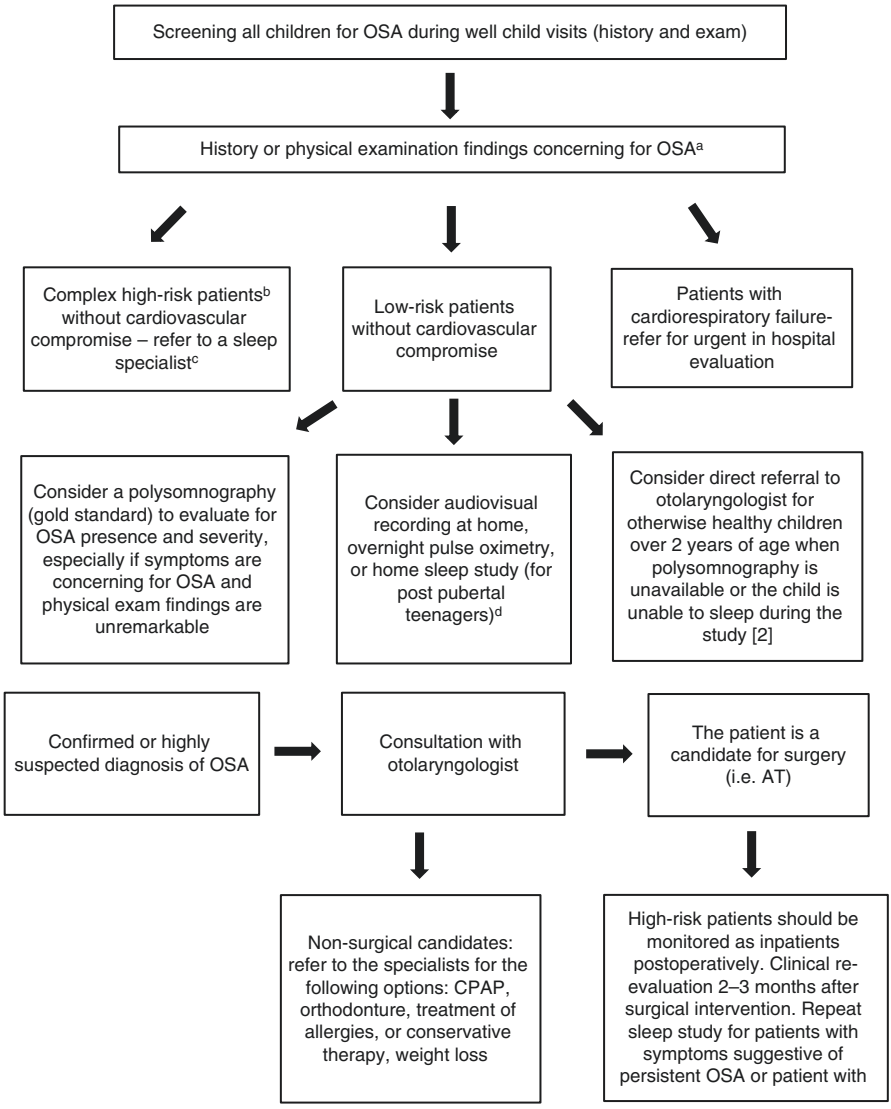
Most studies have focused on evaluating growth and obesity after AT based on anthropometric measurements such as BMI. Although BMI is a simple method to evaluate obesity, it does not distinguish between lean body mass and body fat [51]. This would be an important consideration as obesity is defined by an abnormal percentage of adipose tissue in the body, and children have a higher percentage of lean body mass than adults [52]. Bioelectrical impedance analysis (BIA) appears to be a relatively reliable, simple, and noninvasive method evaluating body fat and lean body mass [87]. One prospective study [54] used BIA to evaluate the difference between the rate of weight increase among children ages 6–9 with chronic tonsillitis and adenotonsillar hypertrophy following adenoidectomy or AT versus healthy controls. This study demonstrated an improvement in BMI in the surgical group without an increase in body fat percentage. However, children in this study were not screened for OSA [54].

Resolution of GF in children with OSA undergoing AT is attributed to increased postoperative levels of circulating IGF-1 and IGFBP-3 [7, 44, 53, 54, 89], reduced upper airway and systemic inflammation [53], increased caloric intake [62] due to unhealthy food choices [29], decreased nocturnal caloric expenditure due to lower work of breathing during sleep, resolution of intermittent hypoxemia, and reduction in wakefulness caloric expenditure due to reduction and/or resolution of hyperactivity [44, 70].

Key Learning Points

- Obese children with OSA may not have ATH but do have pathophysiological changes and metabolic derangements similar to adults with OSA.
- Obese children with OSA are more likely to develop excessive daytime sleepiness than non-obese children with OSA.
- Although normalization of growth post-AT is beneficial in the setting of GF, an exaggerated increase in weight gain in overweight and obese children could increase their risk for OSA recurrence and obesity-related morbidity.

Screening Children for Obstructive Sleep Apnea, Proposed Algorithm Based on American Academy of Pediatrics Clinical Practice Guideline: Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome, 2012



- [a]Historical findings associated with OSA include habitual snoring with labored breathing, observed apnea, mouth breathing, sleep with an arched back, parasomnia, restless sleep, daytime neurobehavioral abnormalities or sleepiness, and others. Physical findings may include growth abnormalities, obesity, mouth breathing, malocclusion, signs of nasal obstruction, adenoidal facies, enlarged tonsils, hypertension, and others. Note that some patients may have no abnormalities on examination.
- [b]Complex, high-risk patients include infants younger than 12 months and children with craniofacial disorders, Down syndrome, neuromuscular disorders (including cerebral palsy), chronic lung disease, sickle cell disease, central hypoventilation syndromes, persistent asthma, or genetic, metabolic, or storage disease.
- [c]Subspecialist refers to a physician with expertise in sleep disorders in children. This physician may be a pulmonologist, neurologist, or other physician with experience in the management of sleep-disordered breathing in children.
- [d]AASM: does not recommend home sleep apnea testing for OSAS diagnosis in children (AASM Practice Parameters 2017; Kirk et al. 2017).

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