

Sleep disorders in children

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Sleep problems, including problems at bedtime and frequent night waking, affect 30%–40% of infants and children before school age.¹ Effects of sleep disorders on the health of the child may include poor growth, adverse behavioural and learning effects and, for the child and family, worsened mental health, and poor quality of life.² The likelihood that important and treatable sleep disorders go unrecognised is increased because many parents do not mention their concerns to their general practitioner, or the doctor does not ask about or identify the issues.^{3,4} Simple management strategies can be effective at a primary care level. An important role of the GP or general paediatrician is to identify children's sleep problems and to differentiate those who would benefit from referral to specialty services.

Average sleep times vary with age, and community surveys indicate considerable variability in sleep requirements, to the extent that normative values are sometimes debated. However, systematic review of the literature can guide general recommendations for sleep duration at different ages.⁵ Newborn infants sleep 16–18 hours per day in cycles of 3–4 hours (day and night). After 6 months of age, healthy infants can sleep for more than 6 hours at night without a feed. By 18 months of age, sleep patterns usually mature to overnight sleep plus one daytime nap. By school age, sleep consolidates into a single night sleep of 11–12 hours. Sleep duration continues to slowly reduce from about 10 hours in prepubescent children to 8 hours by 16 years of age. Individual children and adolescents may benefit from longer sleep times than these average figures, and enquiry about daytime functioning is an important part of assessing adequacy of sleep.^{5,6}

Initial screening is an important aspect of identifying sleep issues in children and the first step in providing timely advice and intervention. An example of a mnemonic to remind physicians of important aspects of history-taking regarding sleep quality in children is BEARS: B = bedtime (settling) problems; E = excessive daytime sleepiness; A = night awakenings; R = regularity and duration of sleep; S = snoring.⁷ Parents define the presence of children's sleep problems, so evaluation of the validity of parental expectations is also important. Age-specific common non-respiratory sleep problems are tabulated in Box 1.⁸

Non-respiratory disorders

Sleep phenomena or parasomnias in children

Parasomnias are undesirable motor, autonomic or experiential phenomena that occur exclusively or predominantly during sleep.⁹ Parasomnias are common in childhood — examples include bruxism (teeth grinding, 6%–10%), sleep terrors (0.7%–2%) and somnambulism (sleep walking, up to 7%).¹⁰ A simplified summary of parasomnias with their prevalence rates is provided in Box 2.⁸ Benign parasomnias may run in families, increase in frequency with any condition that causes sleep deprivation or sleep fragmentation such as fever, and tend to improve with age (Box 2).⁸

Summary

- Sleep disorders are very common in childhood and are often amenable to simple advice and parental education.
- Questions about sleep should be an integral part of every paediatric consultation.
- Children with underlying syndromes or complex medical conditions often have multiple sleep issues.
- Excessive sleepiness in children requires careful history-taking and consideration of specialised investigation.
- Obstructive sleep apnoea (OSA) is a common condition in childhood with important health implications.
- The high prevalence of OSA warrants rigorous attempts to identify children at higher risk and manage them appropriately.
- Adenotonsillectomy is a highly efficacious therapy for paediatric OSA.
- A current major issue is to improve ways of distinguishing mild from severe OSA before a child undergoes adenotonsillectomy, as those with more severe disease are at increased risk of postoperative complications and should undergo adenotonsillectomy in a tertiary centre.
- Children with obesity and other comorbid conditions are at increased risk of persisting OSA despite adenotonsillectomy.
- Topical (nasal) steroids and/or anti-inflammatory agents have a role in the non-surgical treatment of mild OSA.
- Continuous positive airway pressure and orthodontic interventions are treatment options for treatment of persisting OSA in children.

Behavioural sleep disorders

Extremely common sleep problems in children include a child not getting into bed, having difficulty or requiring undue help to settle to sleep, frequent waking in the night and/or getting out of bed, and very early morning awakenings. They are often grouped as behavioural sleep disorders because of the perception that the problem lies with how the child behaves. These problems may lead to insufficient sleep and considerable family disruption. Children with developmental disorders, attention deficit hyperactivity disorder, depression and anxiety have higher incidence of these types of sleep disturbances than other children.¹¹

Management of behavioural sleep disorders and parasomnias

Key to reducing the frequency and severity of behavioural sleep disorders is the provision to parents of preventive information, best provided opportunistically in primary care and by maternal child health nurses. Treatment interventions should then be evidence-based and developmentally appropriate. Parasomnias are usually benign and most decrease in frequency in later childhood. Education and reassurance of parents may be all that is required in less severe cases. Behavioural strategies for management of parasomnias include anxiety-relaxation techniques for poor sleep initiation, and sleep hygiene measures.¹¹ These

1 Examples of non-respiratory sleep disorders in childhood, by most common age at presentation⁸

Age group	Non-respiratory sleep disorder
Infant/toddler (0–2 years)	Behavioural insomnia of childhood: eg, excessive night waking, sleep associations (aids to sleep onset, such as rocking, dummy, milk) Rhythmic movement disorders: eg, body rocking
Preschool (3–5 years)	Behavioural insomnia of childhood: eg, excessive night waking, bedtime refusal Rhythmic movement disorders: eg, head banging Night terrors
Primary school (6–12 years)	Inadequate sleep: eg, due to social pressures such as evening activities and/or poor sleep habits such as watching television in bed Sleep walking
Adolescent (13–18 years)	Inadequate sleep: eg, due to delayed sleep phase syndrome Narcolepsy Periodic limb movements

include limit-setting — for example, gradually removing parents' attendance at the child's bedside, so they are not present at the time of sleep onset — and moving bedtime closer to the usual time of sleep onset, to avoid periods of lying in bed awake before sleep onset. These measures help to eliminate the need for parents to attend to the child at each night-time awakening, and encourage a pattern of prompt sleep onset after going to bed. Together, they avoid prolonged periods of wakefulness during the night.

The core principle of preventing and managing bedtime (settling) issues and frequent night waking is to promote independence in settling to sleep. Infants and children who depend on a parent or other sleep association (music, dummy, rocking) at the start of the night are likely to require the same attention to resume sleep after what are otherwise normal, brief awakenings during the night. Consistency is the most important factor, but the rate of possible change is family-specific and sometimes needs to occur in slow, small steps to be sustained. Maternal mental health is an important factor in managing paediatric sleep disorders; children's sleep problems and poor maternal sleep can contribute to mental health disorders, as well as being an aetiological factor for the inconsistent maintenance of the infant's sleep routines. In a small group of toddlers with difficulty initiating or maintaining sleep, melatonin could be used to entrain their sleep routine. The interventions are also safe, with no negative long-term outcomes and many benefits to child and family health and functioning.¹²

Parasomnias can occur very frequently, cause distress and/or disrupt family life. Management strategies should ensure the safety of the child; for example, by placing the mattress on the floor rather than on a bed frame, and by adding locks to doors to prevent the child opening simple latches while sleep walking. Simple strategies to minimise the frequency of events are often effective for managing parasomnias in otherwise normal children and include:

Extending sleep: insufficient sleep increases the frequency of parasomnias. As little as 30 minutes of additional sleep can reduce the frequency of parasomnias. Work towards earlier bedtime and/or later rise times. Making bedtime

earlier should occur in small steps of 10–15 minutes, to avoid increasing bedtime struggles.

Reducing bedtime anxiety and struggles/conflict: going to bed in an aroused state (anxious, angry or upset) can intensify parasomnias. Aim for a gentle and predictable bedtime routine. Avoid stimulating activities like television or computer games for an hour before bed. If necessary, match bedtime to the usual sleep onset time (even if this is late), then slowly bring bedtime earlier, as above. Medication is rarely indicated.^{11,13} If the problem is very severe, very frequent or atypical, raising the possibility of a seizure disorder, then referral to a sleep specialist is indicated, with polysomnography and/or electroencephalography indicated depending on the clinical scenario.

Investigating excessive daytime sleepiness and circadian rhythm disorders: excessive sleepiness requires systematic evaluation. Possible causes include inadequate sleep, sleep disruption from conditions such as restless leg syndrome and obstructive sleep apnoea (OSA), and circadian rhythm disorders. In children with an apparently sufficient duration of sleep, marked daytime sleepiness may be the only manifestation of narcolepsy, which has an estimated prevalence of 1 in 4000 to 1 in 2000, and a peak of onset at 14 years of age.¹⁴ Recognition of narcolepsy onset in childhood and appropriate treatment is likely to improve learning and daytime functioning.

Disruption to normal circadian rhythmicity, such as very late bed and rise times, can have substantial effects on the ability of a child to participate in school and other activities. Circadian sleep problems are especially common in children with pervasive developmental disorders such as autism spectrum disorder, and also occur in adolescents, where many factors impact on a tendency for the sleep phase to be delayed into the night, making socially imperative morning rise times difficult to achieve. The main focus of therapy is to establish and maintain good sleep hygiene including settling strategies (eg, avoiding screen time and caffeine-containing drinks before bedtime) and consistent timing of sleep throughout the 7-day week. Specialist referral is advised if there is concern about accuracy of diagnosis, or need for additional medical therapy including use of medications such as melatonin. Use of such medications may be indicated but must be in the context of awareness of the high need for ongoing surveillance of short- and long-term side effects.

Respiratory disorders

Snoring and OSA

Snoring and OSA are common, affecting 3%–15% of children, with peak prevalence in the preschool years when lymphoid tissue size in the upper airway is largest relative to the size of the facial skeleton.¹⁵ OSA affects up to 5.7% of children,¹⁶ and so potentially affects one child in every classroom in the country. Although the highest incidence of OSA is in preschoolers (3–5 years of age) with large tonsils, 9% prevalence of snoring has been documented in infants aged 0–3 months.^{15,17}

Identification of severe OSA is important because it is linked to increased risk for postoperative respiratory com-

2 Sleep-state distribution of sleep-related symptoms and parasomnias in childhood that do not require treatment unless they are very frequent or severe*

Sleep state	Diagnosis	Prevalence	Presentation
Non-rapid eye movement-related	Hypnagogic imagery (awake or lucid dreaming)	51%	Vivid visual dreams while in transition to sleep
	Sleep starts	33%	Sudden involuntary "jumps" at sleep onset
	Confusional arousals	17%	Child appears to wake, often distressed, but does not respond normally
	Night terrors	17%	Out of slow-wave sleep, so most often in first third of the night. Child appears to wake and be terrified, but remains unaware of surroundings; attempts to comfort can prolong the event
	Sleep walking	14%	Out of slow-wave sleep, so most often in first third of the night. Child performs apparently coordinated activity (walking, opening doors) but electroencephalography and behaviour retain some characteristics of sleep
Rapid eye movement-related	Dreams	na	Semi-coherent images and sensations recalled after sleep
	Sleep paralysis	7.6%, general population	Seconds to minutes of being unable to perform voluntary movement at sleep onset or awakening
	Nightmare	5.2%, one per week; 10%–50%, 3–5 year olds	Dreams with frightening content
Sleep-state independent	Bruxism	28%	Sounds of grinding and/or evidence of tooth wear
	Rhythmic movement disorder	17%	Body rocking or head banging mainly at sleep onset and/or following night awakenings
	Sleep talking	55%	Semi-coherent speech while apparently asleep
	Periodic limb movements	8.4%–11.9%	Repetitive, brief limb movements during sleep that can cause sleep disturbance, daytime sleepiness and leg discomfort. Associated with reduced iron stores

* The major differential diagnosis of parasomnias, which needs to be excluded in frequent or severe cases, is frontal lobe epilepsy. ◆

promise, including emergency reintubation and unplanned admissions to intensive care. It is a major challenge to identify the children who require perioperative management in tertiary paediatric centres. Box 3 highlights cases where referral for polysomnography is warranted, rather than direct referral for adenotonsillectomy.

OSA is associated with sleep fragmentation and repeated episodes of hypoxia. Polysomnography is superior to other testing methods for determining disease severity and also permits diagnosis of comorbid disorders (eg, periodic limb movements). The thresholds for severity of OSA are lower than in adults, with OSA defined as ≥ 1 obstructive event per hour of sleep on polysomnography. Treatment is generally recommended if the frequency of obstructive respiratory events is > 1.5 per hour. Severity is usually defined as mild for 1–5 events per hour, moderate for 5–10 events per hour and severe for ≥ 10 events per hour. However, no threshold has been established for disease severity with regard to the development of complications. Even mild disease is associated with adverse neurocognitive, behavioural and cardiovascular outcomes, such that even chronic partial obstruction causing snoring without gas exchange abnormalities or evident sleep disruption is associated with adverse effects.

Despite the fact that no clinical assessment method other than polysomnography has proven discriminatory for OSA in children who snore, the number of paediatric sleep units in Australia is inadequate to provide polysomnography to screen all snoring children. The presence of snoring and large tonsils is a sensitive but not specific marker. Helpful clinical indicators include increased work of breathing, parental concern, and frequent daytime mouth breathing.¹⁸ Markers that are specific but not sensitive (helpful when positive, but unable to rule out disease) include excessive daytime somnolence and observed OSA.¹⁹ Almost all screening tools are also specific but not

sensitive, including overnight oximetry (most useful if positive, but most children have a negative study that does not rule out OSA²⁰), video recordings and nap studies, so the search for an ideal screening tool continues. Overnight oximetry is helpful in identifying cases with marked hypoxia, but those using it need to be familiar with the technical aspects and diagnostic limitations of the tool.²¹ All screening tools, including oximetry, are best used in combination with clinical indicators such as young age (under 3 years) and comorbidities (syndromes, obesity, etc), to help evaluate the likelihood of postoperative respiratory complications.²¹

Among the major sequelae of untreated OSA, cardiovascular risks include systemic hypertension, increased sympathetic activation and ventricular hypertrophy, while pulmonary hypertension and right heart failure still occur occasionally in infants and children with severe OSA.²² Even mild OSA is linked to daytime neurocognitive dysfunction that translates into decrements of intelligence quotient, and a randomised controlled study has now been published regarding assessment of neuropsychological development in school-age children with OSA after tonsillectomy.²³ Plausible mechanisms for this association include sleep fragmentation, repetitive hypoxia, and reduced cerebral blood flow and oxygenation. Behavioural improvements follow adenotonsillectomy,^{24–26} but responses in neurocognitive function are variable.²⁷ A review of 25 studies investigating behavioural and neurocognitive outcomes following adenotonsillectomy found that all studies reported improvement in one or more measures including quality of life, behavioural problems including hyperactivity and aggression, and neurocognitive skills including memory, attention and school performance.²⁶ Improvement or resolution of OSA has also been linked to concomitant improvements in systemic and pulmonary blood pressures, heart rate and pulse variability, cardiac morphology and cardiac function.²²

3 Indications for polysomnography in a child suspected to have obstructive sleep apnoea (OSA)

Indications

Conditions with increased surgical risk that should have documentation of disease severity	Complex medical conditions such as Down syndrome, neuromuscular disorders and craniofacial syndromes Age < 3 years
Discordance between history and examination	For example, strong history of OSA with small tonsils and no apparent nasal obstruction
Potential alternative explanations for sleep disturbance	Possible combination of central apnoea/hypoventilation (eg, spina bifida) Need to differentiate nocturnal epilepsy (eg, from parasomnias)
Persistence of symptoms after adenotonsillectomy	High-risk groups for persisting OSA: severe initial disease; history of prematurity; congenital syndrome/malformation; obesity; atopy; age > 7 years

The natural history of symptoms of OSA (eg, snoring, mouth breathing and apnoea) is for around 50% of pre-school children to move (bidirectionally) among severity groups over a 2-year follow-up period.²⁸ In a cohort of 12 447 children studied across seven time points between ages 6 months and 6.75 years, the prevalence of OSA symptoms was highest between 3.5 and 4.8 years of age.²⁹ The highest peak of new symptoms occurred between the ages of 1.5 and 2.5 years.²⁹ Another study undertook polysomnography on 45 children with mild OSA at baseline; at follow-up 4 years later, disease had worsened in 37% and resolved in 26%.³⁰

Preschool children generally respond to adenotonsillectomy; meta-analysis shows cure rates of 82% in otherwise normal children.³¹ Success rates for adenotonsillectomy are lower in obese³² and older³³ children, and adenoidectomy and/or tonsillectomy is usually not appropriate for infants. Although adenotonsillectomy reduces the severity of OSA in obese children, such children have more severe initial disease, and obesity increases the risk for persisting disease.^{32,34} Nasal corticosteroid sprays³⁵⁻³⁷ and leukotriene-receptor antagonists (eg, montelukast)³⁸ are helpful in children with mild OSA and for some with persistent disease after adenotonsillectomy, and a treatment trial is appropriate before pursuing other interventions.³⁹ Specific airway problems, especially infants with Pierre Robin sequence, may respond to mandibular distraction, continuous positive airway pressure, nasopharyngeal tube, and/or oral tongue positioning devices, but may necessitate tracheostomy.

Factors that indicate a higher risk for persisting OSA despite adenotonsillectomy include more severe initial disease (respiratory disturbance index > 10/h or minimum SaO₂ < 80%), obesity with body mass index > 95th percentile for age and sex, and children aged > 7 years at the time of surgery (whether obese or non-obese).³³ There is interplay between obesity and atopy, in that for non-obese children, comorbid asthma increases the risk of persisting disease whereas allergic rhinitis is only significant when both obese and non-obese groups are considered together.⁴⁰ These groups need follow-up after surgery to establish whether snoring has or has not resolved.

Older children and adolescents may respond to adenotonsillectomy or require other treatments including continuous positive airway pressure, orthodontic and other surgical or dental procedures (rapid maxillary expansion,

or mid-face advancement). Evidence of efficacy and safety in children is limited for orthodontic options such as mandibular advancement splints.⁴¹⁻⁴⁴ These interventions aim to affect growth of the face and oropharyngeal airway to produce long-term structural changes, irrespective of whether the initial airway problem is primary, or secondary to OSA.

Children with underlying medical disorders

Underlying medical disorders work to both increase risk for OSA and to reduce the effectiveness of surgical treatment (Box 3). In particular, congenital abnormalities that affect craniofacial or thoracic growth, such as achondroplasia and Down syndrome, will predispose to sleep-disordered breathing. In Down syndrome, there appears to be particular risk for hypertrophy of the lingual tonsils.³³ It is also known that children with multiple disabilities have increased risk for other sleep disturbances such as difficulties with sleep initiation and maintenance, insomnia and other sleep pattern abnormalities.⁴⁵

Children with neuromuscular diseases have increased incidence of OSA in the first decade.⁴⁶ Congenital cardiothoracic abnormalities or restrictive lung disorders, often linked to neuromuscular disorders or neurodevelopmental disability such as cerebral palsy, also predispose to nocturnal respiratory failure. Symptoms suggestive of nocturnal hypoventilation include increased frequency or severity of lower respiratory tract infections, and progression of scoliosis. Screening should include pulmonary function testing, with sleep studies for children with vital capacity < 60% of that predicted and for non-ambulant children before scoliosis surgery, and pragmatic consideration of screening versus full polysomnographic studies.⁴⁷ Early identification and treatment of impaired pulmonary function can prevent or reduce the frequency and duration of admissions to intensive care units, as well as improving quality and duration of life.⁴⁸

Congenital central hypoventilation syndrome is a rare but highly treatable condition (incidence, 1 in 50 000 live births).⁴⁹ This usually presents during the neonatal period with frequent apnoeas or colour change during sleep, but milder forms can present in older children.⁵⁰

Conclusion

Sleep disorders are common in childhood and are associated with significant consequences for children and parents. Behavioural disorders include sleep onset delay, sleep interruptions, early morning waking and combinations of these elements. Parasomnias are very common and can be frequent and severe enough to warrant specialist referral. Access to tertiary and specialist assessment services is limited, so good triage of sleep disorders by primary care services and general paediatricians is essential. Identification and treatment of OSA is important in children. Immediate risk for respiratory compromise can be identified before adenotonsillectomy, and there are high rates of cure after surgery. Untreated, OSA is associated with risk of cardiovascular, neurodevelopmental and ongoing respiratory health problems. For triage purposes, Box 3 highlights situations where referral for specialist services with access to polysomnography is suggested in cases of sus-

pected OSA. Finally, children with persisting symptoms despite surgery will often benefit from polysomnography and specialist evaluation to determine the severity of ongoing disease, identification of cause, and need (or not) for ongoing treatment. Childhood presents an opportunity for effective, early intervention in sleep disorders.

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- 1 Mindell JA, Kuhn B, Lewin DS, et al. Behavioral treatment of bedtime problems and night wakings in infants and young children. *Sleep* 2006; 29: 1263-1276.
- 2 Martin J, Hiscock H, Hardy P, et al. Adverse associations of infant and child sleep problems and parent health: an Australian population study. *Pediatrics* 2007; 119: 947-955.
- 3 Blunden S, Lushington K, Lorenzen B, et al. Are sleep problems under-recognised in general practice? *Arch Dis Child* 2004; 89: 708-712.
- 4 Schreck KA, Richdale AL. Knowledge of childhood sleep: a possible variable in under- or misdiagnosis of childhood sleep problems. *J Sleep Res* 2011; 20: 589-597.
- 5 Galland BC, Taylor BJ, Elder DE, et al. Normal sleep patterns in infants and children: a systematic review of observational studies. *Sleep Med Rev* 2012; 16: 213-222.
- 6 Blair PS, Humphreys JS, Gringras P, et al. Childhood sleep duration and associated demographic characteristics in an English cohort. *Sleep* 2012; 35: 353-360.
- 7 Owens JA, Dalzell V. Use of the 'BEARS' sleep screening tool in a pediatric residents' continuity clinic: a pilot study. *Sleep Med* 2005; 6: 63-69.
- 8 Moore M, Allison D, Rosen CL. A review of pediatric nonrespiratory sleep disorders. *Chest* 2006; 130: 1252-1262.
- 9 American Academy of Sleep Medicine. International classification of sleep disorders: diagnostic and coding manual. 2nd edition. Westchester, Ill: American Academy of Sleep Medicine, 2005.
- 10 Agargun MY, Cilli AS, Sener S, et al. The prevalence of parasomnias in preadolescent school-aged children: a Turkish sample. *Sleep* 2004; 27: 701-705.
- 11 Heussler H, Chan P, Price AM, et al. Pharmacological and non-pharmacological management of sleep disturbance in children: an Australian Paediatric Research Network survey. *Sleep Med* 2013; 14: 189-194.
- 12 Hiscock H, Davey MJ. Sleep disorders in infants and children. *J Paediatr Child Health* 2012; 21 Dec [Epub ahead of print].
- 13 Kotagal S, Chopra A. Pediatric sleep-wake disorders. *Neurol Clin* 2012; 30: 1193-1212.
- 14 Peterson PC, Husain AM. Pediatric narcolepsy. *Brain Dev* 2008; 30: 609-623.
- 15 Raynes-Greenow CH, Hadfield RM, Cistulli PA, et al. Sleep apnea in early childhood associated with preterm birth but not small for gestational age: a population-based record linkage study. *Sleep* 2012; 35: 1475-1480.
- 16 Marcus CL, Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2012; 130: e714-e755.
- 17 Piteo AM, Lushington K, Roberts RM, et al. Prevalence of snoring and associated factors in infancy. *Sleep Med* 2011; 12: 787-792.
- 18 Carroll JL, McColley SA, Marcus CL, et al. Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. *Chest* 1995; 108: 610-618.
- 19 Certal V, Catumbela E, Winck JC, et al. Clinical assessment of pediatric obstructive sleep apnea: a systematic review and meta-analysis. *Laryngoscope* 2012; 122: 2105-2114.
- 20 Brouillette RT, Morielli A, Leimanis A, et al. Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. *Pediatrics* 2000; 105: 405-412.
- 21 Nixon GM, Kermack AS, Davis GM, et al. Planning adenotonsillectomy in children with obstructive sleep apnea: the role of overnight oximetry. *Pediatrics* 2004; 113 (1 Pt 1): e19-e25.
- 22 Teo DT, Mitchell RB. Systematic review of effects of adenotonsillectomy on cardiovascular parameters in children with obstructive sleep apnea. *Otolaryngol Head Neck Surg* 2013; 148: 21-28.
- 23 Marcus CL, Moore RH, Rosen CL, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. *N Engl J Med* 2013; 368: 2366-2376.
- 24 Mitchell RB, Kelly J. Outcome of adenotonsillectomy for severe obstructive sleep apnea in children. *Int J Pediatr Otorhinolaryngol* 2004; 68: 1375-1379.
- 25 Ali NJ, Pitson D, Stradling JR. Sleep disordered breathing: effects of adenotonsillectomy on behaviour and psychological functioning. *Eur J Pediatr* 1996; 155: 56-62.
- 26 Garetz SL. Behavior, cognition, and quality of life after adenotonsillectomy for pediatric sleep-disordered breathing: summary of the literature. *Otolaryngol Head Neck Surg* 2008; 138 (1 Suppl): S19-S26.
- 27 Giordani B, Hodges EK, Guire KE, et al. Changes in neuropsychological and behavioral functioning in children with and without obstructive sleep apnea following tonsillectomy. *J Int Neuropsychol Soc* 2012; 18: 212-222.
- 28 Lofstrand-Tidestrom B, Hultcrantz E. The development of snoring and sleep related breathing distress from 4 to 6 years in a cohort of Swedish children. *Int J Pediatr Otorhinolaryngol* 2007; 71: 1025-1033.
- 29 Bonuck KA, Chervin RD, Cole TJ, et al. Prevalence and persistence of sleep disordered breathing symptoms in young children: a 6-year population-based cohort study. *Sleep* 2011; 34: 875-884.
- 30 Li AM, Zhu Y, Au CT, et al. Natural history of primary snoring in school-aged children: a 4-year follow-up study. *Chest* 2013; 143: 729-735.
- 31 Brietzke SE, Gallagher D. The effectiveness of tonsillectomy and adenoidectomy in the treatment of pediatric obstructive sleep apnea/hypopnea syndrome: a meta-analysis. *Otolaryngol Head Neck Surg* 2006; 134: 979-984.
- 32 Costa DJ, Mitchell R. Adenotonsillectomy for obstructive sleep apnea in obese children: a meta-analysis. *Otolaryngol Head Neck Surg* 2009; 140: 455-460.
- 33 Shott SR. Evaluation and management of pediatric obstructive sleep apnea beyond tonsillectomy and adenoidectomy. *Curr Opin Otolaryngol Head Neck Surg* 2011; 19: 449-454.
- 34 O'Brien LM, Sitha S, Baur LA, et al. Obesity increases the risk for persisting obstructive sleep apnea after treatment in children. *Int J Pediatr Otorhinolaryngol* 2006; 70: 1555-1560.
- 35 Brouillette RT, Manoukian JJ, Ducharme FM, et al. Efficacy of fluticasone nasal spray for pediatric obstructive sleep apnea. *J Pediatr* 2001; 138: 838-844.
- 36 Esteitie R, Emani J, Sharma S, et al. Effect of fluticasone furoate on interleukin 6 secretion from adenoid tissues in children with obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 2011; 137: 576-582.
- 37 Kheirandish-Gozal L, Gozal D. Intranasal budesonide treatment for children with mild obstructive sleep apnea syndrome. *Pediatrics* 2008; 122: e149-e155.
- 38 Goldbart AD, Greenberg-Dotan S, Tal A. Montelukast for children with obstructive sleep apnea: a double-blind, placebo-controlled study. *Pediatrics* 2012; 130: e575-e580.
- 39 Kheirandish L, Goldbart AD, Gozal D. Intranasal steroids and oral leukotriene modifier therapy in residual sleep-disordered breathing after tonsillectomy and adenoidectomy in children. *Pediatrics* 2006; 117: e61-e66.
- 40 Bhattacharjee R, Kheirandish-Gozal L, Spruyt K, et al. Adenotonsillectomy outcomes in treatment of obstructive sleep apnea in children: a multicenter retrospective study. *Am J Respir Crit Care Med* 2010; 182: 676-683.
- 41 Villa MP, Malagola C, Pagani J, et al. Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. *Sleep Med* 2007; 8: 128-134.
- 42 Villa MP, Bernkopf E, Pagani J, et al. Randomized controlled study of an oral jaw-positioning appliance for the treatment of obstructive sleep apnea in children with malocclusion. *Am J Respir Crit Care Med* 2002; 165: 123-127.
- 43 Chung CH, Font B. Skeletal and dental changes in the sagittal, vertical, and transverse dimensions after rapid palatal expansion. *Am J Orthod Dentofacial Orthop* 2004; 126: 569-575.
- 44 Marino A, Ranieri R, Chiarotti F, et al. Rapid maxillary expansion in children with obstructive sleep apnoea syndrome (OSAS). *Eur J Paediatr Dent* 2012; 13: 57-63.
- 45 Tietze AL, Blankenburg M, Hechler T, et al. Sleep disturbances in children with multiple disabilities. *Sleep Med Rev* 2012; 16: 117-127.
- 46 Suresh S, Wales P, Dakin C, et al. Sleep-related breathing disorder in Duchenne muscular dystrophy: disease spectrum in the paediatric population. *J Paediatr Child Health* 2005; 41: 500-503.
- 47 Hull J, Aniapravan R, Chan E, et al. British Thoracic Society guideline for respiratory management of children with neuromuscular weakness. *Thorax* 2012; 67 Suppl 1: i1-i40.
- 48 Yates K, Festa M, Gillis J, et al. Outcome of children with neuromuscular disease admitted to paediatric intensive care. *Arch Dis Child* 2004; 89: 170-175.
- 49 Hasegawa H, Kawasaki K, Inoue H, et al. Epidemiologic survey of patients with congenital central hypoventilation syndrome in Japan. *Pediatr Int* 2012; 54: 123-126.
- 50 Parodi S, Vollono C, Baglietto MP, et al. Congenital central hypoventilation syndrome: genotype-phenotype correlation in parents of affected children carrying a PHOX2B expansion mutation. *Clin Genet* 2010; 78: 289-293. □