Sleep disorders in children

Sleep problems, including problems at bedtime and frequent night waking, affect 30%–40% of infants and children before school age. 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. 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. 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. 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.

Non-respiratory disorders

Sleep phenomena or parasomnias in children

Parasomnias are undesirable motor, autonomic or experiential phenomena that occur exclusively or predominantly during sleep. Parasomnias are common in childhood — examples include bruxism (teeth grinding, 6%–10%), sleep terrors (0.7%–2%) and somnambulism (sleep walking, up to 7%). A simplified summary of parasomnias with their prevalence rates is provided in Box 2. 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).
1 Examples of non-respiratory sleep disorders in childhood, by most common age at presentation

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

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 rhythm, 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-
### Sleep disorders

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

<table>
<thead>
<tr>
<th>Sleep state</th>
<th>Diagnosis</th>
<th>Prevalence</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-rapid eye movement-related</td>
<td>Hypnagogic imagery (awake or lucid dreaming)</td>
<td>51%</td>
<td>Vivid visual dreams while in transition to sleep</td>
</tr>
<tr>
<td></td>
<td>Sleep starts</td>
<td>33%</td>
<td>Sudden involuntary “jumps” at sleep onset</td>
</tr>
<tr>
<td></td>
<td>Confusional arousals</td>
<td>17%</td>
<td>Child appears to wake, often distressed, but does not respond normally</td>
</tr>
<tr>
<td></td>
<td>Night terrors</td>
<td>17%</td>
<td>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</td>
</tr>
<tr>
<td></td>
<td>Sleep walking</td>
<td>14%</td>
<td>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</td>
</tr>
<tr>
<td>Rapid eye movement-related</td>
<td>Dreams</td>
<td>na</td>
<td>Semi-coherent images and sensations recalled after sleep</td>
</tr>
<tr>
<td></td>
<td>Sleep paralysis</td>
<td>7.6%, general population</td>
<td>Seconds to minutes of being unable to perform voluntary movement at sleep onset or awakening</td>
</tr>
<tr>
<td></td>
<td>Nightmare</td>
<td>5.2%, one per week; 10%–50%, 3–5 year olds</td>
<td>Dreams with frightening content</td>
</tr>
<tr>
<td>Sleep-state independent</td>
<td>Bruxism</td>
<td>28%</td>
<td>Sounds of grinding and/or evidence of tooth wear</td>
</tr>
<tr>
<td></td>
<td>Rhythmic movement disorder</td>
<td>17%</td>
<td>Body rocking or head banging mainly at sleep onset and/or following night awakenings</td>
</tr>
<tr>
<td></td>
<td>Sleep talking</td>
<td>55%</td>
<td>Semi-coherent speech while apparently asleep</td>
</tr>
<tr>
<td></td>
<td>Periodic limb movements</td>
<td>8.4%–11.9%</td>
<td>Repetitive, brief limb movements during sleep that can cause sleep disturbance, daytime sleepiness and leg discomfort. Associated with reduced iron stores</td>
</tr>
</tbody>
</table>

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

Promising, 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. Markers that are specific but not sensitive (helpful when positive, but unable to rule out disease) include excessive daytime somnolence and observed OSA. 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 OSA), 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. 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.

Among the major sequela 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. 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. Plausible mechanisms for this association include sleep fragmentation, repetitive hypoxia, and reduced cerebral blood flow and oxygenation. Behavioural improvements follow adenotonsillectomy, but responses in neurocognitive function are variable. 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. 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.
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.\textsuperscript{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.\textsuperscript{29} The highest peak of new symptoms occurred between the ages of 1.5 and 2.5 years.\textsuperscript{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%.\textsuperscript{30}

Preschool children generally respond to adenotonsillectomy; meta-analysis shows cure rates of 82% in otherwise normal children.\textsuperscript{31} Success rates for adenotonsillectomy are lower in obese\textsuperscript{32} and older\textsuperscript{33} 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.\textsuperscript{32,34} Nasal corticosteroid sprays\textsuperscript{35-37} and leukotriene-receptor antagonists (eg, monteleukast)\textsuperscript{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.\textsuperscript{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 \(\text{SaO}_2 < 80\%\), obesity with body mass index \(> 95\%\) tile for age and sex, and children aged \(> 7\) years at the time of surgery (whether obese or non-obese).\textsuperscript{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.\textsuperscript{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.\textsuperscript{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 hypotrophy of the lingual tonsils.\textsuperscript{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.\textsuperscript{45}

Children with neuromuscular diseases have increased incidence of OSA in the first decade.\textsuperscript{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 scoliois surgery, and pragmatic consideration of screening versus full polysomnographic studies.\textsuperscript{37} 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.\textsuperscript{48}

Congenital central hypoventilation syndrome is a rare but highly treatable condition (incidence, 1 in 50 000 live births).\textsuperscript{49} This usually presents during the neonatal period with frequent apnoeas or colour change during sleep, but milder forms can present in older children.\textsuperscript{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-
Retrieved reimbursement for expenses relating to speaking at a conference sponsored by effective, early intervention in sleep disorders.


