Sleep in Cerebral Palsy

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Michelle Sobremonte-King

Introduction

Cerebral palsy (CP) is a group of nonprogressive developmental disorders that affects the development of movement and posture [1]. It is the most common motor disability of childhood occurring at a rate of 1.5–4 per 1000 live births [2]. Children afflicted by CP often have muscle spasms, musculoskeletal pain, contractures, epilepsy, antiepileptic medications, and behavioral abnormalities that predispose them to a wide range of sleep disorders (Table 13.1) [3]. The handful of studies looking at the prevalence of sleep disorders in children with CP use different criteria but collectively demonstrate a higher prevalence as compared to the typically developing pediatric population, with a prevalence ranging from 19% to 44% [3–6]. Sleep disorders in patients with CP can have a significant impact on a child's physical, emotional, and cognitive development with far-reaching consequences that also affect caregivers and families who in turn also become sleep deprived [7]. Despite the substantial health consequences of sleep disorders in CP patients and their families, there is a lack of both awareness and applied clinical research geared to CP-related sleep disorders [8]. In a study involving 286 children with mild to profound intellectual delay (including patients with CP), only 19% of the parents of children with a current sleep problem received any advice about their child's sleep disorder from a healthcare professional [9]. As the multidisciplinary care and longevity of children with CP improve, recognizing and addressing sleep disorders in these patients becomes an important component in improving and maintaining their quality of life.

M. Sobremonte-King (✉)
Department of Pediatric Pulmonary and Sleep Medicine, The Breathing Institute, Children's Hospital Colorado, Aurora, CO, USA
e-mail: michelle.sobremonte-king@seattlechildrens.org

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Table 13.1 Common sleep disorders in cerebral palsy

Sleep disorder	Specific disorder	Possible etiologies	Management
Insomnia	Primary insomnia (inadequate sleep hygiene and behavioral insomnia of childhood) Secondary insomnia (insomnia due to medical condition)	Pain Abnormal muscle tone Inability to reposition Poor sleep hygiene and environment (i.e., bed-sharing, etc.)	Pain/spasticity control (i.e., baclofen pump) Behavioral intervention (i.e., graduated extinction, faded bedtime, bedtime routines, etc.) Melatonin
Sleep-related breathing disorder	Obstructive sleep apnea Hypoventilation	Maxillofacial abnormalities (i.e., maxillary hypoplasia, palatal hypotonia, glossoptosis, retrognathia, etc.) Abnormal neuromuscular control of upper airway Kyphoscoliosis Gastroesophageal reflux disease	Diagnostic polysomnogram Tonsillectomy and adenoidectomy Tongue base suspension Noninvasive ventilation (CPAP) Secretion management and airway clearance
Circadian rhythm disorder	Free-running circadian disorder Delayed sleep phase	Visual impairment Poor sleep hygiene (i.e., exposure to blue-spectrum light)	Melatonin Light therapy Sleep hygiene
Hypersomnia	Hypersomnia due to a medical condition or drug effect	Epilepsy Seizure medications (i.e., benzodiazepines, phenobarbital, phenytoin, valproic acid, etc.)	Seizure control Avoiding sedating anticonvulsants Avoiding anticonvulsants that disrupt sleep architecture

CPAP continuous positive airway pressure

Spectrum of Sleep Disorders in Children with CP

An integrative review by Lelis et al. reported 21 factors associated with the development of sleep disorders in children with CP [10]. These risk factors were divided into intrinsic and extrinsic risk factors. Intrinsic factors were related to physiological features, and extrinsic factors were related to environmental issues. The succeeding section will summarize (Table 13.2) and detail what we think are the most important factors according to the framework suggested by Lelis et al. Among the sleep disorders described are sleep hyperhidrosis, disorders of arousal, sleep anxiety, difficult morning awakening, bruxism, insomnia, nightmares, parasomnias, difficulties in initiating and maintaining nighttime sleep, sleep-wake transition disorders, and sleep-disordered breathing [5, 10]. In general, these can be grouped into six broad categories: sleep-related breathing disorder, insomnia, circadian rhythm disorders, parasomnias, hypersomnia, and sleep-related movement disorders.

Table 13.2 Factors associated with the development of sleep disorders in patients with cerebral palsy

Intrinsic factors	Extrinsic factors
Structural abnormalities	Medications (antiepileptics)
Airway obstruction	Posturing devices
Obesity	Sleep practices (bed-sharing)
Postural limitations/spasticity	Socioeconomic
Epilepsy	Maternal unemployment
Visual disturbances	
Cognitive impairment	
Pain	

Sleep-Related Breathing Disorders

Sleep-related breathing disorders (SRBD) are a group of respiratory disorders that are observed or become more severe during sleep and include obstructive sleep apnea (OSA), central apnea, and hypoventilation. These disorders occur more frequently in children with CP as compared to normally developing children at a prevalence of 15–18% [5, 11]. In particular, children with CP are at increased risk for developing OSA. This occurs when pharyngeal dilator muscles are unable to maintain the patency of the airway against the subatmospheric pressure generated during inspiration. Pathophysiologic mechanisms for the development of OSA can be broadly divided into anatomic factors that reduce the caliber of the upper airway, such as craniofacial abnormalities, obesity, and adenotonsillar hypertrophy, and factors that increase upper airway collapsibility, such as neurologically based alterations in upper airway muscle tone [12]. The latter are of particular concern in CP as these children have a wide range of structural and functional abnormalities that compromise upper airway muscle tone and predispose to OSA. These include maxillary hypoplasia, palatal hypotonia, glossoptosis, retrognathia, laryngomalacia, and laryngeal dystonia [13–15]. They can also have abnormal neuromuscular control of their upper airways that further complicate structural abnormalities [11]. Untreated OSA is associated with diminished quality of life and neurocognitive detriment and can also result in cardiorespiratory failure and death in severe cases [16–18]. Unfortunately, airway obstruction is underappreciated in children with CP, and the potential for OSA is often only identified from a lengthy history of snoring [10]. In a small retrospective case series of eight children with severe spastic quadriplegic CP presenting with upper airway obstruction, seven of the children required admission to the intensive care unit and two required placement of a tracheostomy. Interestingly, despite the relatively severe symptoms and presentation, only two of these children had previously been referred for sleep studies [15].

Identifying children with sleep disorders can be particularly challenging as they can present with a myriad of subtle symptoms that can be difficult to distinguish from symptoms related to other comorbidities. These symptoms can include behavioral changes, cognitive impairment, failure to thrive, decreased

appetite, and developmental delay [19]. As such, a structured evaluation with screening questionnaires can be an effective strategy to evaluate for OSA and other SRDBs in patients with CP. In a study by Garcia et al., the Pediatric Sleep Questionnaire (PSQ) and Gross Motor Function Classification System (GMFCS) were used to evaluate symptoms of OSA in 215 children, 18 of which had CP. The investigators found increased PSQ scores (indicating increased risk of OSA) for children with CP (58%) and CP with epilepsy (67%) as compared to normal controls (27%) [20]. Whether this risk for OSA increases with the severity of CP is unclear with some studies showing a relationship and others showing no relationship [11, 20]. In a prospective study evaluating parent responses to the Sleep Disturbance Scale for Children (SDSC) in 173 patients with CP, Newman et al. reported that 14.5% of patients had a pathologic score for disorders of sleep-related breathing on the SDSC [5]. These studies highlight the utility of a structured approach to screening children with CP for sleep-disordered breathing.

For children with CP, an overnight polysomnogram (PSG) remains the gold standard for the diagnosis of OSA. The PSG can also reveal the underlying presence of seizure disorders and other movement disorders that can complicate SRBDs and compromise quality of sleep (Figs. 13.1 and 13.2). Although other diagnostic techniques such as nocturnal pulse oximetry have promising utility in detecting moderate-to-severe OSA, this method has not been validated in patients with neurodevelopmental disorders [21].

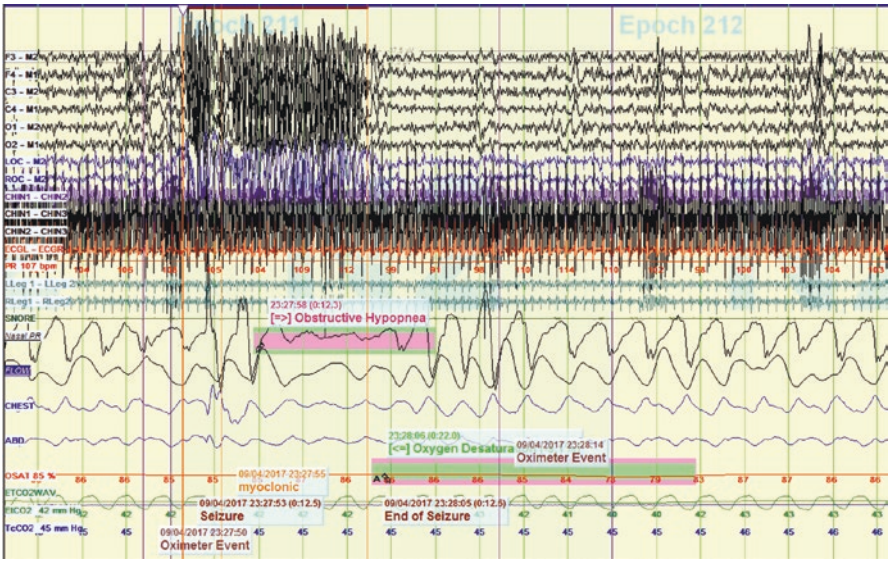

Fig. 13.1 Polysomnogram epoch demonstrating myoclonic seizure associated with obstructive hypopnea in a 17-year-old with spastic quadriplegic cerebral palsy

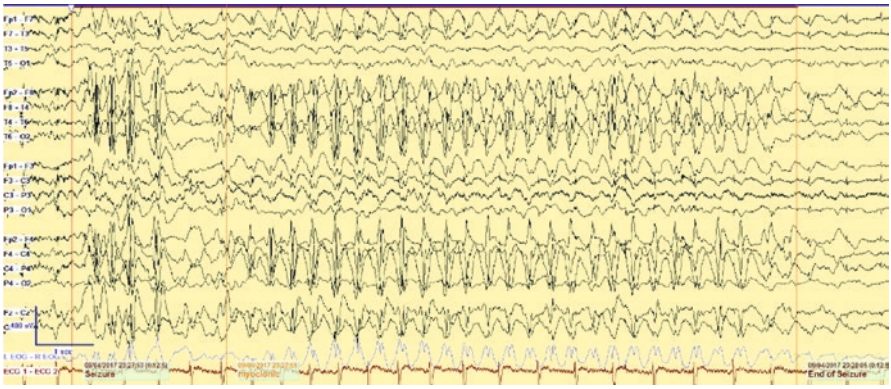

Fig. 13.2 Myoclonic absence seizure, right posterior predominant shown in a 21-channel electroencephalogram (EEG) obtained during polysomnography in a 17-year-old with spastic quadriplegic cerebral palsy

Insomnia

Disorders of initiation and maintenance of sleep are common in children with CP. In a study of 100 children with CP divided into 2 groups (preschool group, 52 patients, and school-aged group, 48 patients), Elsayed et al. found that up to 46.2% of preschool children and 25% of school-aged children had evidence of early insomnia based on results from surveys with sleep questionnaires. Insomnia was also found to be more common in GMFSC grade IV and V [22]. In the study by Newman et al., disorders of initiation and maintenance of sleep were found mostly in patients with spastic quadriplegia and dyskinetic CP [5]. These findings support the concept that children with CP have problems with initiation and maintenance of sleep due predominantly to physical factors that stem from their primary neuromuscular disorder. Factors such as abnormal muscle tone and the inability to alter posture to alleviate pain make it difficult for the child with CP to initiate and maintain sleep. Ironically, caregivers can be distressed by observing these difficulties and decide to co-sleep with their child which in turn has the potential to exacerbate insomnia [5, 10].

Circadian Rhythm Disorders

Establishing and maintaining normal sleep patterns for children with CP can be difficult. As will be evident in subsequent section, children with CP have a multitude of factors that can affect sleep quality and consistency, and addressing all or even some of these factors can be challenging. Perhaps the most significant factor contributing to circadian rhythm disorders in patients with CP is visual deficits. Light is the central regulator of the circadian rhythm in humans via its stimulation of the retinal-hypothalamic tract and suppression of melatonin secretion. Cortical blindness and other visual disturbances either nullify or diminish the ability of these patients to develop a circadian rhythm. In fact, patients with CP often have free-running sleep-wake cycles. For patients who are not blind, neuromuscular

immobility can still play a role in children who are exposed to blue-spectrum light and poor sleep hygiene. Careful consideration frequency and timing of blue-spectrum light can be important in children with CP who may not have the same opportunities and abilities to participate in outdoor activities that provide sufficient daytime light exposure [7].

Hypersomnias, Parasomnias, and Sleep-Related Movement Disorders

A study by Newman et al. showed that the principal factor associated with sleep disturbance was epilepsy, which was primarily associated with excessive daytime somnolence [5]. A similar observation was seen in a study of 293 children and adults with learning disabilities where the presence of active epilepsy was the strongest independent predictor of excessive daytime sleepiness [23]. As such, hypersomnia appears to be more of a manifestation of an underlying disease process such as epilepsy, rather than being a primary sleep disorder. Further complicating matters is that some medications used to treat epilepsy can be sedating and cause hypersomnolence. In particular, benzodiazepines which can be used to treat either seizures or muscle spasms can be particularly sedating and can also increase the risk of developing SRBDs [24].

With regard to parasomnias and sleep-related movement disorders, Elsayed et al. observed the following frequencies in a cohort of 100 children with CP: sleepwalking in 30%, sleep bruxism in 38%, and periodic limb movement disorder/restless legs syndrome in 46% [22]. Although attribution of underlying factors to the development of parasomnias and sleep-related movement disorders is difficult to specify, CP children with pain had a higher prevalence of parasomnias [7].

Risk Factors Associated with the Development of Sleep Disorders in Children with CP

Intrinsic Factors

Structural Abnormalities

Children with CP have a wide range of structural and functional abnormalities that predispose them to a variety of sleep disorders. These abnormalities can be grouped anatomically and divided into abnormalities affecting the upper airway or the thoracic cage. Among the reported anatomic abnormalities that lead to dynamic airway obstruction during sleep are maxillary hypoplasia, palatal hypotonia, glossoptosis, retrognathia, and laryngomalacia [13–15]. In addition, there can be abnormal neuromuscular control of their upper airways that further complicates structural abnormalities [11]. For instance, in older children, laryngomalacia is thought to develop due to repetitive collapse of supraglottic structures into the airway due to poor supraglottic muscle tone. This results in reduction of the caliber of the airway and consequent stridor from airway compromise during inspiration [14, 15].

With regard to thoracic abnormalities, kyphoscoliosis is common in children with CP occurring in as much as 39% [14]. It is generally thought that thoracic skeletal abnormalities such as kyphoscoliosis can affect sleep and exacerbate SRBDs by reducing the child's ability to expectorate which contributes to recurrent aspiration, impaired mucociliary clearance, and lower airway obstruction and respiratory failure [14]. Severe kyphoscoliosis has also been associated with hypoventilation and hypoxemia during sleep assuming a supine position [10, 25].

Another anatomic, but also functional, abnormality that can contribute to pulmonary disorders which can lead to sleep disorders is gastroesophageal reflux disease (GERD). Up to 80–90% of children with CP have a chronic gastrointestinal problem of which GERD is the most common [26]. GERD results from reflux of gastric acid into the esophagus through an either anatomically incompetent (hiatal hernia) or functionally incompetent gastroesophageal sphincter [27]. The presence of GERD in patients with CP puts them at significant risk for recurrent aspiration which leads to the development or exacerbation of SRBDs and chronic lung disease [5, 14]. This is particularly important for children with CP since abnormal tone and neuromuscular disorders prevent some children to optimally position themselves to prevent recurrent aspiration. In severe cases, anti-reflux surgery with Nissen fundoplication has been reported to improve symptoms with caregiver reported improvement in sleep in 32% of patients [28].

Epilepsy

The presence of an epilepsy in patients with CP is strongly associated with the presence of a sleep disorder. Epilepsy is present in approximately 20–40% of patients with CP and is most common in children with hemiplegic or quadriplegic CP [29]. Newman et al. conducted one of the first studies to determine the prevalence of sleep disorders in CP patients and found that the presence of active epilepsy was associated with the presence of a sleep disorder (odds ratio [OR] = 17.1) [5]. Another equally large cohort of CP patients demonstrated a similar association where investigators found an abnormal SDSC total score in 34% of children with CP plus active epilepsy as compared to 18% in children with CP and controlled epilepsy and 15% in children with CP without epilepsy [3]. The similar rates of abnormal SDSC scores in patients with controlled epilepsy and normal control underscore the importance of effective antiepileptic treatment in treating sleep disorders in patients with CP. Although the precise nature of the relationship between epilepsy and sleep disorders in patients with neurodevelopmental abnormalities is complex, it is generally viewed that uncontrolled seizure activity can perpetuate a vicious cycle of fragmented sleep architecture that results in sleep deprivation which in turn results in lowering of the seizure threshold [30]. A case-controlled study of 31 patients with drug-resistant epilepsy showed that compared to normal controls, these patients had a significant reduction of time in bed, total sleep time, rapid eye movement (REM) sleep, sleep stage N2, sleep efficiency, and a significant increase in wake after sleep onset [30]. Antiepileptic drugs add to the complexity as they themselves can influence sleep architecture and will be discussed later.

Visual Impairment

Between 20% and 50% of children with CP have cortical visual impairment which significantly increases the risk of developing sleep disorders through dysregulation of the circadian rhythm [8]. Bright light is the major stimulus for the human circadian pacemaker located in the suprachiasmatic nuclei which regulates wakefulness by suppressing melatonin secretion by the pineal gland. Without light, melatonin secretion is disinhibited and the diurnal variation is lost. Consequently, the prevalence of sleep disorders in blind patients is considerably high, and one study showed that up to 83% of patients in a combined pediatric and adult cohort disclosed at least one sleep problem, as compared to 57% in the control population [31]. More specifically, 17% of these patients reported a free-running sleep-wake cycle which is a phenomenon that is described in children with congenital blindness [31, 32].

Cognitive Disability

Cognitive impairment frequently occurs in children with CP and has been increasingly reported to be associated with sleep disorders in children with CP [33]. Some studies demonstrate that the degree of cognitive impairment can be a predictor for the presence of an underlying sleep problem [3, 9]. In contrast, other studies do not find an association between the presence of cognitive impairment making the precise nature of the relationship yet to be fully determined [34, 35].

Pain and Muscle Spasticity

Pain and discomfort during sleep is a common occurrence in children with physical disabilities and has been suggested by some to be the strongest contributing factor for sleep disorders to develop in these children [36, 37]. Pain is particularly common in children with CP and is reported in as high as 67–84% of patients [38]. Many factors such as skin breakdown, pressure ulcers, involuntary movements, abnormal postures, and abnormal muscle tone in the form of spasticity contribute to pain in children with CP and have the ability to decrease the quality of sleep [7]. In a study of 123 children with CP, patients who had pain also had significantly more sleep problems overall, more night awaking, parasomnia, sleep-disordered breathing, and shorter sleep duration [39].

In particular, pain may be associated to the underlying disease-related mechanisms in CP. Lesions in the motor areas of the brain and descending corticospinal tracts result in muscle spasticity, characterized by increased tone, hyperreflexia, clonus, and resistance to stretching. The resulting musculoskeletal pain and primary motor impairment decrease the ability to change body position during the night and may contribute to sleep disturbances [5]. Intervention to decrease pain and discomfort from muscle spasticity has been demonstrated to improve sleep quality in children with CP. A study evaluating the use of an intrathecal baclofen pump in 35 children with bilateral cerebral palsy and severe spasticity improved sleep quality with a reduction in nighttime awakening within 6 months of implantation [40]. As such, pain and abnormal muscle tone are significant factors that interfere with sleep in children with CP and can be addressed with recognition and intervention. Unfortunately, although this area is a major contributor to sleep disorders, it is underappreciated and research activity is sparse [7].

Extrinsic Factors

Antiepileptic Medications

Antiepileptic medications have been described to influence the sleep architecture and the sleep quality of patients who take them [41]. Since many patients with CP with epilepsy will need antiepileptic medications to control their seizure disorder, it is generally viewed that these medications can predispose them to have sleep disorders. However, the interaction is complex since different types of antiepileptics have different effects on sleep, and some CP patients with epilepsy will be on multiple antiepileptic medications. For instance, drugs like phenobarbital, phenytoin, valproic acid, and higher-dose levetiracetam aggravate daytime sleepiness, whereas the antiepileptics topiramate and zonisamide do not [41, 42]. In contrast, drugs like gabapentin, pregabalin, tiagabine, clobazam, and carbamazepine were found to reduce sleep latency and/or actually improve sleep efficiency [42]. Alternatively, other antiepileptic drugs such as benzodiazepines not only affect sleep architecture but also promote somnolence and decrease upper airway muscle tone leading to aggravation of sleep-disordered breathing and OSA [10].

Despite the complexity of these interactions, clinicians should not lose track of the utility and benefits of these medications in controlling seizure activity which, as mentioned, is a stronger factor influencing sleep in patients with CP. It is clear that children with CP and uncontrolled seizures have a higher prevalence of sleep disorders as compared to those who have their epilepsy controlled, regardless of the number of medications they may be on [3].

Positioning and Posturing Devices

Preoccupation with nighttime positioning and the use of uncomfortable devices for improving postural problems in children with CP are common and generally viewed as having the potential to disrupt sleep [10]. Although the body of available data describing this interaction is sparse and varied, the general impression is that posturing devices do not significantly disrupt sleep and may actually improve the quality of sleep. In one study, no significant differences in PSG sleep quality measures were seen in children wearing nighttime posturing equipment as compared to children without [43]. Another study reached the same conclusion using GMFCS levels to evaluate sleep quality [35].

Familial and Cultural Effects

A variety of familial factors including bed-sharing, being a single parent, maternal unemployment, and low socioeconomic status have been associated with an increase in sleep disorders in children with CP [5]. Of note, although several studies have reported an increase in sleep problems associated with the practice of bed-sharing, drawing inferences as to cause and effect can be tricky [44, 45]. Problems during sleep may be the reason for parents to co-sleep with their child, and, expectedly, this setting ideally places them to be able to observe a number of sleep-related events which would have otherwise remained unnoticed [5]. Consequently, parents who co-sleep with their child often report more sleep problems in their children as compared to non-bed-sharing parents [10].

Behavioral Correlates

Behavioral and psychiatric problems are common in children with CP, and both can significantly impact sleep quality and exacerbate underlying sleep disorders [46]. A tool that is often used to evaluate behavioral problems in patients with neurodevelopmental disorders is the Child Behavior Checklist (CBCL), which can group scored syndromes into two "broad band" scales: internalizing problems (anxious/depressed, withdrawn-depressed, and somatic complaints scores) and externalizing problems (rule-breaking and aggressive behavior) [47]. Romeo et al. used the CBCL to estimate the incidence and patterns of behavioral problems and their association with sleep disorders in a prospective single-institution study of 165 children with CP. They found that 24% of children reported an abnormal CBCL total score, with 27% showing internalizing and 9% with externalizing problems. In addition, behavioral problems were often associated with abnormal Sleep Disturbance Scale for Children (SDSC) total scores, as well as total and internalizing CBCL scores [3].

Social Impact of Sleep Disorders on Children with Cerebral Palsy and Their Families

Unrecognized and untreated sleep disorders in patients with CP can have serious and far-reaching consequences that affect both the child and their families. It is well established that poor sleep quality in children with sleep disorders can result in negative effects on behavior and school performance [24, 48, 49]. In addition, the burden of caring for children with CP can be significant for family members especially for those with sleep disorders. Wayte et al. report that nearly 40% of children with CP required parental attention on at least one occasion every night, and 74% of parents reported that their own daytime functioning was affected by their child's sleep disorder [50].

Some parents feel the need to co-sleep due to their child's restlessness and the compulsion of needing to position them to address issues of pain related to abnormal tone. This in turn creates the potential for sleep disorders in the parents, and many studies document that they do not get enough sleep themselves [5, 40]. In a study of 40 children with CP, 40% of mothers were found to have poor sleep quality of whom 44% had a depressed mood. In addition, child and maternal sleep disturbance were significantly correlated, and maternal sleep quality predicted 50% of the variance in maternal depression [50]. Other studies also report that parents of children with sleep problems feel more stressed and irritable and express that their child's sleep problems interfere and negatively impact their own work and social lives [5, 34, 51]. As such, when placed in this broader context, it is clear that sleep disorders in children with CP need to be recognized as of being of wider concern because of the physical and psychological toll that can result for the entire family [7].

Treatment of Sleep Disorders in Children with CP

The management of sleep disorders in children with CP can be particularly challenging due to the multitude and variety of contributing factors that often occur simultaneously. It often necessitates addressing the contributing intrinsic and extrinsic factors, many of which are social and behavioral. As such, Tremblay et al. recommend that the first step in treatment of insomnia in children with CP should always be behavioral interventions. Examples of behavioral interventions include graduated extinction, faded bedtime, parent education, and positive bedtime routines. These can be particularly effective in non-SRDB-related sleep disorders where several studies have demonstrated their efficacy [52, 53]. Modification of factors related to the environment, such as exposure to blue-spectrum light, sound, and wide swings in ambient temperature, has the potential to interfere with sleep is another noninvasive strategy that can be effective in improving sleep quality in patients with CP. For instance, excessive and untimely exposure of children to artificial blue-spectrum light, usually through electronic devices like television and smartphones, can alter the timing, duration, and amount of melatonin synthesis and, therefore, interfere with the circadian rhythm [54]. This becomes a particular issue considering that some parents and caregivers co-sleep with their children. Alternatively, bed-sharing is another modifiable factor that can be addressed to improve sleep quality in children [7].

Pharmacologic treatment may be indicated when behavioral measures and environmental modification are ineffective or do not suffice. However, robust data on pharmacologic treatment strategies are lacking, much more so in children with CP or other neurodevelopmental abnormalities. Chloral hydrate is the only medication currently labeled by the US Food and Drug Administration for the treatment of insomnia in children but is not recommended by the American Academy of Pediatrics due to the risk of hepatotoxicity [55]. The use of other medication appears to be based largely on personal clinical experience and data extrapolated in the treatment of adults [55]. A review by Owens and Mindell describes reports of antidepressants, anticonvulsants, antipsychotics, and alpha-agonists in the treatment of childhood insomnia, but data are mostly derived from small studies and case series and do not describe including patients with CP [55].

Exogenous administration of melatonin has been successfully used in typically developing children to treat circadian rhythm disorders and disorders of initiating and maintaining sleep. Of all the previously described medications, melatonin has received the most attention, and randomized data that includes patients with CP are available. A review of six randomized controlled trials ($N = 82$) of melatonin in children with intellectual disability suggests that melatonin can be effective in reducing sleep onset latency and increase total sleep time [56]. Wasdell et al. conducted a randomized double-blind placebo-controlled crossover trial to evaluate the efficacy of controlled-release melatonin in the treatment of delayed sleep phase syndrome and impaired sleep maintenance in children with neurodevelopmental disabilities. The study enrolled 51 children ages 2–18 years (26 patients had CP)

who did not respond to sleep hygiene intervention. Fifty patients completed the crossover trial and 47 completed the open-label phase. The study reported a significant improvement of total nighttime sleep and sleep latency by approximately 30 minutes as documented by direct observation and by actigraph recordings [57]. Unfortunately, the authors of this study did not provide data or a detailed description of potential toxicities of melatonin during the trial. Potential toxicities that have been described include depression, enuresis, somnolence, and one report of increase in seizure activity [24, 58]. However, a study of melatonin in children with intractable epilepsy did not report an increase in seizure activity [59].

Evidently, some sleep disorders in children with CP are structural and need to be addressed with more invasive measures. OSA is the primary example and is a common cause of sleep-disordered breathing in children with CP. The initial management of OSA in children involves identifying adenotonsillar hypertrophy as a contributing factor and tonsillectomy as the primary treatment [17]. Of note, children with CP are at an increased risk of postsurgical complications, and the need for postoperative monitoring cannot be understated [60].

Tongue base suspension (TBS) is another surgical technique that has been described to potentially benefit patients with OSA and CP in combination with other surgical techniques [61]. A study by Hartzell et al. described PSG data in CP children who received adenotonsillectomy and uvulopalatopharyngioplasty plus TBS vs. adenotonsillectomy and uvulopalatopharyngioplasty alone. Patients who received TBS had significant improvements in their arousal index leading the authors to conclude that children with CP and moderate to severe OSA may potentially benefit from TBS [60].

In children in whom OSA persists despite tonsillectomy or in whom tonsillectomy cannot be done, other measures such as nasopharyngeal airway devices, oxygen supplementation, and continuous positive airway pressure (CPAP) are implemented. A CPAP machine works by delivering an intraluminal pressure that is above the critical closing pressure of the airway that effectively stents the airway open to overcome dynamic obstruction [62]. Unfortunately, compliance with nonsurgical techniques is a common problem, and treatment of OSA in patients with neurodevelopmental abnormalities like CP can be challenging. A guiding principle in managing OSA in patients with CP is recognizing that OSA is a multifactorial disorder and that treatment should be individualized based on the underlying neurologic abnormality and site of obstruction [24].

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