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REVIEW PAPER

Psychological and functional problems of children and adolescents with cerebral palsy from the neurodevelopmental perspective

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ABSTRACT

The main aim of this article was to review some psychological and psychiatric problems in children and adolescents with cerebral palsy (CP) in the context of individual development. The problems in gaining the appropriate experiences during sensitive periods of development were also considered. The risk factors were described and it was revealed that physical activity, sleep problems and pain accounted for the incidence of depression and other mental health problems in patients with CP. Reviewed studies showed that children with CP are at greater risk of having other neurodevelopment disorders, which means that they should be carefully diagnosed for a wider range of problems. The need of expected experiences and proper stimulations, obligatory for brain and mental development during sensitive periods, should also be taken into consideration as CP may restrict or limit the opportunity to receive them.

KEY WORDS:

children, cerebral palsy, development, adolescents, psychological problems.

INTRODUCTION

Cerebral palsy (CP) is one of the most common causes of physical disability in children, originating in prenatal and perinatal disturbances in the development of the central nervous system, concerning impairments of movement and postural control with heterogeneity of clinical outcome. The prevalence of CP is about 2.1 per 1000 live births with higher incidence in lower income areas [1]. This disorder was described for the first time in 1862 by the orthopedic surgeon William John Little as "cerebro-spinal disorder" [2, 3]. The muscle stiffness and limb deformity observed in those patients were attributed to "congenital distortions of their limbs" and were related to the "cerebrospinal disorder" due to the presence of post-mortem abnormalities such as capillary congestion on the surface of the brain or spinal cord and blood in cerebrospinal fluids. Little [2, 3] also introduced a novel hypothesis about the etiology of CP as the outcome of brain anoxia secondary to difficult labor in contrast to previous beliefs about mechanical origins of physical deformities. The term "cerebral palsy" was popularized after a series of lectures titled *The Cerebral Palsies of Children* given by Sir William Osler [4]. Furthermore, Sigmund Freud was the first to introduced the classification system of CP based on the timing of injury but he ultimately abandoned the research in that field because of the lack of definitively described neuropathological abnormalities of the brain for this disorder [5].

The recent definition of CP is described as the group of permanent disorders of the developement *the group of permanent disorders of the development of movement and posture causing activity limitation that are attributed to non-progressive disturbances occurred in the developing*

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fetal or infant brain [6]. It is worth noting that this definition still lacks the notion that symptoms of CP are also related to spinal cord distortions. The main symptoms of CP include:

- spasticity and hyper-reflexes due to loss of descending inhibition of the motor neurons, expressed as stiffness of muscles and exaggerated reflexes,
- dyskinesia (dystonia, athetosis, chorea): unwanted, sustained involuntary movements due to pathology of deep brain structures,
- ataxia: incoordination of movement due to cerebellar injury or maldevelopment,
- hypotonia: decreased muscle tone, associated with muscle or peripheral nerve disorders [7, 8].

Neurology nomenclature distinguishes four types of CP. The most common (approximately 80%) is the spastic type with walking abnormalities, muscle weakness and paralysis, affecting both or only one side of the body. The next is a dyskinesia type, characterized as difficulties with controlling body motility as a consequence of involuntary, abnormally rapid or slow movements of the arms, hands, legs, face or tongue, which significantly affects walking, sitting, swallowing or talking. The hypotonic type is characterized by diminished muscle tone, poor reflexes, and weakness of the muscles. Children with this type of CP present "floppy posture", have problems with head and gait control, breathing, speaking, and walking. The least common type of CP is the ataxic type characterized by disorganized, clumsy or jerky but voluntary muscle movements. Children with this kind of disorder have problems with balance, coordination, walking, and fine motor functions such as grasping objects or writing. Finally, a mixed type of CP is characterized by complex spastic and dyskinetic symptoms [9].

The underlying causes of CP have not been fully explained but several risk factors during prenatal and postnatal development have been identified. The preterm delivery has been identified as the strongest risk factor among such factors as multiple fetuses, atypical malformations, placental pathology, intrauterine infections, and perinatal hemorrhages. The genetic background of CP is also complex. Genetic factors may underlie phenotypically different disorders and different genetic factors may lead to phenotypically similar clinical outcomes. One of the studies confirming a shared genetic or environmental background for CP and other neurodevelopment disorders is a Norwegian study which was conducted on the population of children with CP and their biological siblings [10]. Higher incidence of other developmental abnormalities has been identified in patients with CP in comparison to the healthy control group. The differences among children with CP and those without CP were, respectively, as follows: epilepsy – 29.5% vs. 0.8%; intellectual disability – 5.8% vs. 0.5%; attention deficit hyperactivity disorder (ADHD) – 1% vs. 1.8%; autism spectrum disorders – 3% vs. 0.4%; deafness – 3.5% vs. 0.7%, and blindness – 2.1% vs. 0.06%. Higher comorbidity of CP or any other neurodevelopment disorders was also demonstrated among siblings of CP patients. Twice higher incidence of intellectual disability [odds ratio (OR) = 2.3] and epilepsy (OR = 1.8) was revealed along with other higher occurrence of other disorders such as autism (OR = 1.6), attention deficit/hyperactivity disorder (OR = 1.3), blindness (OR = 2.4), and schizophrenia (OR = 2). It is also worth noting the lack of increased risk for bipolar disorder but higher risk for losing another child by stillbirth or neonatal death in families with CP. The shared genetic or environmental background for those neurodevelopment impairments was also confirmed by stronger associations of neurodevelopment aberrations within sets of twins [10]. In conclusion, the above relations proved the complexity of CP etiology and its biological heterogeneity.

The main aim of this article is to review some psychological and psychiatric problems in children and adults with CP in the context of three questions:

1. What is the comorbidity of cerebral palsy and mental disorders? Are there any differences in mental comorbidity between patients with different kinds of cerebral palsy?
2. What are the risk factors for emotional problems or mental disorders in children and adults with cerebral palsy?
3. How might cerebral palsy influence the gaining of experiences during sensitive periods of human development?

WHAT IS THE COMORBIDITY OF CEREBRAL PALSY AND MENTAL DISORDERS? ARE THERE ANY DIFFERENCES IN MENTAL COMORBIDITY BETWEEN PATIENTS WITH DIFFERENT KINDS OF CEREBRAL PALSY?

A cross-sectional European study, as part of a larger project named SPARCLE, was conducted in 2008 to characterize the psychological problems, environmental influences, and quality of life in children with CP [11]. The recruited group of 1174 children aged 8–12 years old, living in Western Europe, was diagnosed according to gross motor functioning, bimanual use of arms and hands, their intellectual abilities or presence of severe visual or hearing impairments [12]. The caregivers were also interviewed about the behavioral and emotional functioning of the patients in the context of conduct, hyperactivity, emotions, and peer relations. Some predictors of further psychopathology were revealed. The risk factors for problematic functioning defined in psychological questionnaires as abnormal scarring were described in groups of patients:

- with moderate impairment of gross motor functioning, indicating that the children with no limitation of motor ability and those who needed total assistance with moving about (Gross Motor Function Classification System

[GMFCS] level more than IV) were less likely to have severe emotional impairments,
- with intellectual disability (IQ < 70),
- suffering from chronic pain,
- having hearing impairments; the children who needed hearing aids showed a three-fold higher risk of symptomatology in comparison to the children without hearing aids,
- presenting impaired communication skills: children with normal or alternative formal methods of speech did not present elevated risk in contrast to the group with difficulties with but still using speech.

Moreover, the analysis of family factors demonstrated that having at least one chronically disabled or ill sibling was associated with an increased level of emotional and functioning difficulties. Interestingly, the lack of siblings was also associated with emotional or functional problems among patients with CP. In conclusion, the results showed that 38.5% of children with CP struggled with emotional problems and a similar group presented enhanced hyperactivity. The highest prevalence was noted for "peer problems" – almost 47.8%, whereas conduct problems were revealed to occur only in 15.1% of patients with CP. The influence of CP subtypes on the psychological condition of children was also analyzed and only slightly higher risk of psychopathology was found in the group of children with ataxic CP. The other familial and environmental factors, such as attending special units or a special school, having single/separated parents or living in a small town, as well as a lack of qualifications or low qualifications of the parents, were associated with an increased incidence of psychological problems. In summary, the incidence of psychopathology in children with CP was high, with 23.9% of children showing an abnormal level of problems. The results also showed that children with milder forms of neurological impairments (better motor and communicative functioning) were struggling with more social demands and stresses as they were more similar to the able-bodied peers but the differences in their functioning were more pronounced. In contrast, more severely handicapped children with lower expectancies demonstrated a lower level of conduct or hyperactivity symptomatology. Furthermore, greater intellectual impairments or pain complaints were associated with higher risk of psychological problems [11]. The above results demonstrated that not only functional disabilities or chronic pain afflictions but also deficits in social competencies were an important area of emotional burden and psychological distress in children and adolescents with CP.

The other valuable research conducted by Downs *et al.* [13] from the Cerebral Palsy Mental Health Group was the analysis of the prevalence of mental health problems in patients with CP, defined in line with the DSM-5 or ICD-10 criteria. In contrast to previous studies, neurodevelopment disorders such as attention-deficit-hyperactivity disorder or autism spectrum disorder (ASD) were excluded from the research. Eight papers involving 1771 children were analyzed, with the majority of studies providing a description of the GMFCS levels, while three studies provided intellectual functioning with the proportion of intellectual disability in the range 21–55%. The majority of studies used the Strengths and Difficulties Questionnaire (SDQ) or the Child Behavioral Checklist (CBCL) [14–18]. The authors demonstrated that 19.6% of children suffered from mental disorders, with the majority having more than one diagnosis. The pooled prevalence of abnormal scores in the SDQ was 35% with significant heterogeneity and higher occurrence of peer (32–87%) and emotional problems (17–30%) than hyperactivity (5–28%) and conduct disorders (0–34%). The prevalence of mental health symptoms measured in the CBCL was estimated at 28% but almost 46% of children scored positively for any CBCL symptoms. Only one study [19] included the Kiddie Schedule for Affective Disorders and Schizophrenia

(K-SADS) psychiatric interview and revealed the prevalence of psychiatric diagnoses as high as 57% in the group with CP. Interestingly, oppositional defiant disorder was the most common diagnosis, next to obsessive-compulsive disorder, generalized anxiety disorder, and separation anxiety disorder. Other diagnoses such as affective, phobic, and panic disorders occurred with lower frequency whereas psychosis, substance abuse, and post-traumatic stress disorders or even conduct disorders were not detected [13]. The authors also confirmed that the presence and severity of psychiatric disorders were determined by the type and severity of CP [19]. Next, the relationship between clinical outcome of CP such as motor functioning or intellectual disability and prevalence of mental health problems was analyzed [17]. It was confirmed that the prevalence of mental health symptoms in children with CP was associated with the range of their motor impairments. In relation to the GMFCS level (I–V), the prevalence of mental health problems was as follows: 24% of children with GMFCS level I (low impairment), 40% in groups with levels II and III and 34% of patients with the highest motor difficulties (level IV or V in the GMFCS). For intellectual disability, the results showed that mental problems were detected in 24% of CP children with the developmental quotient of greater than 85, 42% with the developmental quotient of 50–85, and 38% of CP patients with the developmental quotient of less than 50 [17]. None of the studies conducted an analysis of any relationship between emotional disorders and CP topography [13].

Sleep difficulties are another clinically significant problem in patients with CP. The comorbidity and severity of sleep disorders have not been studied extensively in this group of patients. The recent systemic review by Horwood *et al.* [20] demonstrated that the prevalence of sleep disorders in children with CP was 23.4% for any sleep abnormalities and 26.9% for sleep initiation

and maintenance disorders (DIMS). Among the most common sleep problems were: sleep-wake transition disorders (18.1%), sleep breathing disorders (12.5%), disorders of excessive somnolence (9.6%), disorders of arousal (9.0%), sleep hyperhidrosis (8.3%), parasomnias (7.8%), and non-restorative sleep (3.4%). This meta-analysis also revealed higher rates of sleep problems in the group of older children. The severity of motor impairments or the number of comorbidities was revealed to correlate with higher rate of sleep problems. Moreover, the research of Munyumu *et al.* [21] confirmed and characterized sleep differences in children with various kinds of CP and motor impairments. The total scoring measured in the Sleep Disturbance Scale for Children (SDSC) differs slightly between children with bilateral spastic CP (spastic diplegia/quadriplegia), unilateral spastic CP (spastic hemiplegia), dyskinesia and ataxia/non-classified and measured 41.2%, 24.0%, 30.8%, and 17.4% respectively [21]. Furthermore, the severity of sleep problems was revealed to be related to the level of motor functioning. According to GMFCS levels I–V, the incidence of sleep disturbances was as follows: 15.4%, 20.0%, 15.4%, 38.5%, and 48.9% respectively. In addition, the analysis conducted in a group of pre-school children also proved the relationship between scoring of the SDSC and GMFCS levels I–V. The results showed growing incidence of sleep difficulties in line with GMFCS levels I to V as follows: 7.5%, 4.3%, 13.3%, 16.7%, and 37.% respectively [22]. Also rates of sleep disturbance in school-aged children with CP were revealed to be significantly higher in comparison to the healthy control group and concerned 12.9% of children with GMFCS levels I–IV and 36.6% of children with GMFCS level V [23].

In conclusion, it is worth emphasizing that sleep problems in children with CP might often be unrecognized despite their broad prevalence. Their treatment should be the priority goal with the behavioral interventions as a first-line treatment (Table 1).

TABLE 1. Prevalence of psychological and functional problems in children with cerebral palsy – summary

Developmental abnormalities in children with CP in comparison to a group without diagnosis of CP were respectively as follows [10]	Epilepsy – 29.5% vs. 0.8% Intellectual disability – 5.8% vs. 0.5% ADHD – 1% vs. 1.8% ASD – 3% vs. 0.4% Deafness – 3.5% vs. 0.7% Blindness – 2.1% vs. 0.06%
The prevalence of CP and other disorders in siblings of the patients with CP in comparison to siblings of the healthy control children was as follows [10]	Intellectual disability [OR= 2.3] Blindness [OR=2.4] Schizophrenia [OR=2] Epilepsy [OR=1.8] ASD [OR=1.6] ADHD [OR=1.3]
Emotional problems among children with CP in Europe [11]	38.5% of children with CP struggled with emotional problems Among them "peer problems" (47.5%) were noted for the highest prevalence, with 23.9% of children showing an abnormal level of problems
The prevalence of mental health problems defined with the DSM-5 or ICD-10 criteria, excluding neurodevelopment disorders (ASD and ADHD) described by Downs [13]	19.6% of children with CP suffered from mental disorders 35% of patients presented abnormal level of the SDQ scoring with the highest occurrence of peer (32–87%) and emotional problems (17–30%) 28% of patients fulfilled the criteria of mental health symptoms in the CBCL Almost 46% of children had positive scoring for CBCL symptoms
Prevalence of psychiatric diagnoses confirmed by the K-SADS interview [19]	Prevalence of psychiatric diagnoses was as high as 57%, with oppositional defiant disorder as the most common diagnosis
Prevalence of neurodevelopmental (ASD, ADHD) disorders among children with CP [41]	35% of children fulfilled the criteria for ASD 56% of children fulfilled the criteria for ADHD The overlap of both diagnoses was as high as 29%
Sleep difficulties [20]	Prevalence of sleep disorders in children with CP was 23% for any sleep abnormalities 26.9% for disorders of sleep initiation and maintenance 18.1% for sleep-wake transition disorders 12.5% for sleep breathing disorders 9.6% for disorders of excessive somnolence 9.0% for disorders of arousal 8.3% for sleep hyperhidrosis 7.8% for parasomnias 3.4% for non-restorative sleep

ADHD – attention deficit/hyperactivity disorder, ASD – autism spectrum disorders, CBCL – Child Behavioral Checklist, CP – cerebral palsy, K-SADS – Kiddie Schedule for Affective Disorders and Schizophrenia, SDQ – Strengths and Difficulties Questionnaire

WHAT ARE THE RISK FACTORS FOR MENTAL HEALTH DISORDERS IN CHILDREN WITH CEREBRAL PALSY?

The majority of patients with CP suffer from many accompanying disorders including epilepsy, intellectual impairment, autism, chronic pain and several physical health problems such as scoliosis, gastrological or pulmonary dysfunctions. Almost half of the patients with CP fulfil the criteria for mental health disorders. Recent studies confirmed that adults with CP were more likely to suffer from at least one psychiatric disorder (with the exception of addiction-related disorders) in comparison to the general population [24]. This might pose a question about the reason for such high comorbidity. One answer is the fact that children with any neurodevelopmental disorders more often present problems in other domains of functioning and are more prone to psychiatric disorders later in adulthood [25–27]. It might be presumed that the severity of motor impairments, intellectual disability, epilepsy or other neurological dysfunctions or autism spectrum disorders may significantly impair the mental and physical functioning of patients with CP. Recently, a new comorbidity index for adults was introduced to clinical practice – the Whitney Comorbidity Index – as an answer to the complexity of problems in this group of patients. This tool has been designed to include and assess clinically relevant comorbidity in order to better discriminate the increased risk of mortality for adults with CP [27].

As mentioned abov, the severity of accompanying dysfunctions and disorders has prognostic value for further life quality of patients with CP. From the therapeutic perspective, the knowledge about early risk factors and the relationship between their incidence and development have vital importance for prophylaxis and individualization of treatment, particularly in the younger population of patients. The physical ability and the mental health quality of children with CP were studied by Whitney *et al.* [24]. The authors compared the patients with CP and the healthy control group in terms of occurrence of mental problems such as depression, anxiety, behavioral/conduct problems or hyperactivity disorder. They also tried to estimate the influence of some general factors such as physical activity, sleep duration, and pain intensity on the functional outcome of patients with CP, in order to differentiate their contribution to health quality from the burden of physical disability. Physical activity was defined as the number of days with sport activity lasting for 60 minutes, sleep duration was determined according to age of the child (9–12 hours/24 hours for children aged 9–12; 8–10 hours/24 hours for adolescents aged 13–18). Finally, pain symptoms were rated as present when the child experienced frequent or chronic difficulties related to repeated or chronic head, back or body pain. The results showed that 56.6% of the patients with CP had moderate or severe disabilities not related to age, sex, household poverty status, or sleep duration, and 22.3% of them suffered from multiple mental disorders with the overall incidence significantly higher even after adjusting for physical activity, sleep duration, and pain in comparison to the control group. Lower prevalence of physical activity and higher prevalence of pain were demonstrated in children with CP. Physical activity was associated with the level of depression, whereas anxiety, behavior/conduct problems, and multimorbidity were directly related to the diagnosis of CP. The occurrence and the level of depression, anxiety, behavior/conduct problems, and multimorbidity in the group with CP were not fully related to the sleep duration variable but were increased in the group with CP independently. On the other hand, pain was revealed to fully explain occurrence of depression in children with CP, which confirmed that pain accounts for the comorbidity of CP and depression. The main conclusion from this study is that children with CP have higher incidence of mental health problems in comparison to children without CP, even after adjusting for several population risk factors. The limitation of this study is the cross-sectional design, which made it unable to explore causality or directionality of exposures and outcomes. It is also worth mentioning the recent paper showing the interrelation between pain, fatigue, and mental problems in individuals with CP [28]. It demonstrated that individuals with GMFCS level I suffered from less pain, fatigue, and depressive symptoms in comparison to individuals with higher GMFCS levels. Moreover, pain and fatigue were more interrelated and more strongly associated with global mental health in individuals with CP [28]. In conclusion, limited physical activity, sleep problems, and pain accounted for the incidence of depression and other mental health problems in this group of patients, which should be taken into consideration in rehabilitation and treatment strategies.

Finally, the role of mental executive functions in the psychological and social functioning of patients with unilateral CP has been taken into consideration. The influence of cognitive functioning on mental health quality in the general population is a well-known fact [29, 30]. The executive functioning is related to cognitive functioning and its role has been demonstrated in many neurodevelopment disorders such as ADHD, Asperger syndrome, nonverbal learning disorders, and fetal alcohol syndrome. The research conducted by Tajik-Parvinchi *et al.* [31] revealed the relationship between cognitive control processes and internalizing/externalizing symptoms in children with neurodevelopment disorders. The authors demonstrated the association of emotion regulation with cognitive functioning such as attention shifting, inhibitory control, and working memory which in consequence mediates the occurrence of internalizing and externalizing symptoms in children with neurodevelopment difficulties.

Cognitive and emotional functioning is also important for emotional-behavioral regulation and social competencies. A study by Whittingham *et al.* [29] conducted in a population with CP analyzed the role of cognitive executive functioning in behavioral, psychological, and social functioning of children with unilateral palsy. The authors confirmed that cognitive executive functioning correlated with psychological or social functioning of patients with CP but they did not find any differences between groups with left or right unilateral palsy. In conclusion, problems with cognitive executive functions proved to be a risk factor for behavioral disorders but not for emotional or peer problems in children with CP.

The higher prevalence of difficulties in cognitive control and executive functioning in children with CP was also described by Coceski *et al.* [32], who revealed that cognitive abilities of adolescents with CP were significantly below the abilities of the healthy group and were characterized by an uneven cognitive profile with higher verbal functions. Particularly, severity of motor impairment, insufficient weight for gestational age, and seizures should be recognized as risk factors for cognitive impairment and taken into consideration by health professionals during interview or treatment planning. In conclusion, executive functioning disabilities tend to substantially increase the risk of mental or behavioral problems/disorders among patients with CP [33].

HOW MAY CEREBRAL PALSY AFFECT THE SENSITIVE PERIODS OF INDIVIDUAL HUMAN DEVELOPMENT?

The sensitive period is usually described as a particular time window during which specific experiences or lack of them plays an essential role in an individual's development. Such specific experiences alter the structure and function of the brain during ontogenesis (Table 2). According to Bornstein [34], sensitive periods influence the physiological, physical, and psychological aspects of human brain functioning, as the modulation of the central nervous system by experiences is the last and by far the most subtle strategy of development. Hebb [35] and Hubel *et al.* [36] proved the existence of critical period plasticity during development of the visual system in the widely known experiment with visual deprivation in rats and cats. In their description of the fundamental principle of neurodevelopment, the occurrence of the appropriate experience/stimuli is obligatory to provide the nervous system with the necessary information to create the subset of synaptic connections. The underlying neural mechanisms are based on the overproduction of synaptic connections and subsequent activity-dependent selective elimination or preservation of neural circuits. In human mental development, Rutter *et al.* [37] described the unique sensitive time to form the attachment system.

TABLE 2. Sensitive periods in human development – their onset and characteristics

Functional development of the brain	Description
6th month of gestation	Cortical responses to sound and light are detected. Prior exposure to sounds in utero leads to an indexed attention preference for a native compared to a foreign language [45]
At birth time	Auditory and emotional systems comprising the limbic and paralimbic structures are well developed to form an attachment and social adaptation
Until 5 years old	Strabismus; patching the healthy eye until this age of life restores functionality of the squinting eye due to prevention of elimination of the non-functioning synapses in the visual cortex area of the weaker eye [46]
2–4 months postnatally until adolescence	Synaptogenesis in auditory and visual cortex takes place
Age of 2–5 years old	Vulnerability to adversity during cognitive development
Up to 8 years of age	Until this time, the function of language might be recovered following destruction of speech areas in the dominant hemisphere [47]
Adolescence	Myelination and synaptic selection "pruning" in the prefrontal cortex. The phase of myelination signals the end of the developmental stage and allows functional stabilization within the established pathways [48, 49]
Early adulthood	Myelination in the frontal cortex concludes following sexual differentiation [48]
Attachment formation	
Up to 3 months postnatally	A child is emotionally less vulnerable to stressful separations from caregivers
1–2 years of age	Infant's ability to differentiate others from themselves. The time of peak synaptic density in the neocortex [49]
From 3 months postnatally up to 6–9 years of age	Sensitive period for stressful separations from parents but also window to develop new intimate and close relationship with new caregivers (children adopted at age 4–7 were able to develop good and close ties with their parents)

They revealed that the attachments were formed as late as seven years of age and children were most vulnerable to stressful separation from their parents before the middle years (6–9) but not before the age of 3 months. They also stated that failure to form an attachment in those first years of life was more significant than in a later period. On the other hand, cognitive development is more influenced by adversity between the ages of 2 and 5 than before this period. The sensitive period also takes place during the development of cortical responses to sound and light. Preterm children show regional differentiation to sensory input and functional connections in their brains because prior exposure to sounds in utero led to sound preferences to native language as a prosodic but not a phonetic pattern penetrates the womb [38]. The sensitive period during development of language lasts to 8 years of age and until this time, language may be recovered completely following destruction of speech areas in the dominant hemisphere. It is worth remembering that the early unilateral hemispherical lesions usually lead to a mild decrease in IQ but do not impair language development [39].

Children with CP gaining the appropriate experiences during sensitive periods may be restricted and disturbed by many functional and morphological distortions. Cerebral palsy concerns not only motor impairment but also problems related to cognition, communication, behavior, and sensation. There is also growing evidence that neurodevelopmental disorders such as autism spectrum disorders or attention-deficit/hyperactivity disorders are more prevalent in children with CP in comparison to the general population [40]. The study conducted by Påhlman *et al.* [41] demonstrated that one third (35%) of children with CP fulfilled the criteria of ASD and half of them (56%) fulfilled the criteria of ADHD (attention hyperactivity disorder). The overlap of the two diagnoses is common and was found in 29% of children. Another screening study conducted in a group of 7-year-old children with CP demonstrated the prevalence of ADHD as high as 42% [42]. Problems with verbal communication, social skills, attention maintenance, and motor abilities may influence the quantity and quality of experiences acquired by patients with CP. The adolescence and transition into adulthood is, in addition to the early period of life, also an important sensitive period during development. This period is characterized by changes in a maturing brain under the influences of physical, psychosocial, and cognitive factors. One study showed functional differences during that time in patients with CP in comparison to a healthy control group [43]. Adolescents with CP usually engage in more passive, solitary activities, which as a result affects structural and functional brain development, in particular self-concept and emotional functioning. Moreover, the analysis using resting-state functional images revealed that higher well-being scores (less motor impairment, low pain level) and better everyday functioning were associated with higher levels of functional connectivity between the medial prefrontal cortex and the right lateral parietal region, the areas related to social and emotional regulation skills [44]. These results suggest that symptoms such as pain or gross motor impairment experienced by patients with CP may lead to disturbances in the brain functional connectivity in areas important for emotional regulation and deterioration of their quality of life. These observations also support the conclusion that the clinical progression of the disorder is not only limited to perinatal or postnatal stages of life, but may be continued into further developmental stages, as the symptoms of CP reduce the opportunity to gain the expected quality and amount of experiences during the sensitive periods of mental development.

CONCLUSIONS

The studies described in this paper show that children with CP are at greater risk of having other neurodevelopment disorders, which means that these patients should be carefully diagnosed for a wider range of problems to enhance their early therapeutic interventions and achieve a better prognosis for their further outcome. The most frequent comorbidity in patients with CP is oppositional defiant disorder, and almost every third patient with CP suffers from peer and emotional problems. That is why therapeutic efforts should take into consideration emotional support in the area of communication and interpersonal skills. Other aspects such as sleep disturbances and pain also negatively affect everyday functioning and quality of life in patients with CP. Pain proved to be a particular factor for comorbidity and depression. Caregivers and professionals should also keep in mind the need for proper and expected experiences and stimulation – obligatory for brain and mental development during sensitive periods – and the fact that CP symptoms may restrict or limit the opportunity to receive them. Recent studies have also shown that CP, aside from higher incidence of metabolic abnormalities, cardiovascular diseases, and depression, is associated with higher risk of dementia, which might be mainly explained by co-occurring intellectual deficits, epilepsy, and sensory impairments [45].

DISCLOSURE

The authors declare no conflict of interest.

REFERENCES

1. Odding E, Roebroeck ME, Stam HJ. The epidemiology of cerebral palsy: incidence, impairments and risk factors. Disabil Rehabil 2006; 28: 183-191.
2. Little WJ. On the influence of abnormal parturition, difficult labours, premature birth and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities. Trans Obstet Soc Lond 1862; 3: 243-344.

3. Little WJ. On the influence of abnormal parturition, difficult labors, premature birth and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities. Clin Orthop Relat Res 1966; 46: 7-22.
4. Osler W. The cerebral palsies of childhood, Lewis, London (UK) 1989.
5. Freud S. Les diplegies cerebrates infantiles [Infantile cerebral diplegia]. Rev neurol (Paris) 1893; 1 : 178-183.
6. Brandenburg JE, Fogarty MJ, Sieck GC. A critical evaluation of current concepts in cerebral palsy. Physiology 2019; 34: 226-229.
7. Shevell M, Dagenais L, Oskoudi M. The epidemiology of cerebral palsy: new perspectives from a Canadian registry. Semin Pediatric Neurol 2013; 20: 60-64.
8. Novak I, Morgan C, Adde L, Blackman J, et al. Early, accurate diagnosis and early intervention in cerebral palsy: advances in diagnosis and treatment. JAMA Pediatr 2017; 171: 897-907.
9. Michałowicz M, Jóźwiak S (eds.). Neurologia dziecięca [Pediatric neurology]. Elsevier Urban & Partner, Wrocław (Poland) 2000, 1st ed.
10. Tollånes MC, Wilcox AJ, Stoltenberg C, et al. Neurodevelopmental disorders or early death in siblings of children with cerebral palsy. Pediatrics 2016; 138: e20160269.
11. Parkes J, White-Koning M, Dickinson HO, et al. Psychological problems in children with cerebral palsy: a cross-sectional European study. J Child Psychol Psychiatry 2008; 49: 405-413.
12. Colver A, SPARCLE Group. Study protocol: SPARCLE – a multicentre European study of the relationship of environment to participation and quality of life in children with cerebral palsy. BMC Public Health 2006; 6: 105.
13. Downs J, Blackmore AM, Epstein A, et al. Cerebral Palsy Mental Health Group. The prevalence of mental health disorders and symptoms in children and adolescents with cerebral palsy: a systematic review and meta-analysis. Dev Med Child Neurol 2018; 60: 30-38.
14. Brossard-Racine M, Hall N, Majnemer A, et al. Behavioral problems in school age children with cerebral palsy. Eur J Paediatr Neurol 2012; 16: 35-41.
15. Brossard-Racine M, Waknin J, Shikako-Thomas K, et al. Behavioral difficulties in adolescents with cerebral palsy. J Child Neurol 2013; 28: 27-33.
16. Romeo DM, Brogna C, Quintiliani M, et al. Sleep disorders in children with cerebral palsy: neurodevelopmental and behavioral correlates. Sleep Med 2014; 15: 213-218.
17. Rackauskaite G, Bilenberg N, Bech BH, et al. Screening for psychopathology in a national cohort of 8- to 15-year-old children with cerebral palsy. Res Dev Disabil 2016; 49-50: 171-180.
18. Weber P, Bolli P, Heimgartner N, et al. Behavioral and emotional problems in children and adults with cerebral palsy. Eur J Paediatr Neurol 2016; 20: 270-274.
19. Bjorgaas HM, Hysing M, Elgen I. Psychiatric disorders among children with cerebral palsy at school starting age. Res Dev Disabil 2012; 33: 1287-1293.
20. Horwood L, Li P, Mok E, et al. A systematic review and meta-analysis of the prevalence of sleep problems in children with cerebral palsy: how do children with cerebral palsy differ from each other and from typically developing children? Sleep Health 2019; 5: 555-571.
21. Munyumu K, Idro R, Abbo C, et al. Prevalence and factors associated with sleep disorders among children with cerebral palsy in Uganda; a cross-sectional study. BMC Pediatr 2018; 18: 26.
22. Romeo DM, Brogna C, Musto E, et al. Sleep disturbances in preschool age children with cerebral palsy: a questionnaire study. Sleep Med 2014; 15: 1089-1093.
23. Romeo DM, Brogna C, Quintiliani M, et al. Sleep disorders in children with cerebral palsy: neurodevelopmental and behavioral correlates. Sleep Med 2014; 15: 213-218.
24. Whitney DG, Warschausky SA, Peterson MD. Mental health disorders and physical risk factors in children with cerebral palsy: a cross-sectional study. Dev Med Child Neurol 2019; 61: 579-585.
25. Gillberg C. The ESSENCE in child psychiatry: Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations. Res Dev Disabil 2010; 31: 1543-1551.
26. Fernell E, Gillberg C. Preterm birth, ADHD and the ESSENCE in adult psychiatry. Acta Paediatr 2012; 101: e568-569.
27. Whitney DG, Kamdar NS. Development of a new comorbidity index for adults with cerebral palsy and comparative assessment with common comorbidity indices. Dev Med Child Neurol 2021; 63: 313-319.
28. Van Gorp M, Dallmeijer AJ, van Wely L, et al. PERRIN DECADE Study Group. Pain, fatigue, depressive symptoms and sleep disturbance in young adults with cerebral palsy. Disabil Rehabil 2021; 43: 2164-2171.
29. Whittingham K, Bodimeade HL, Lloyd O, et al. Everyday psychological functioning in children with unilateral cerebral palsy: does executive functioning play a role? Dev Med Child Neurol 2014; 56: 572-579.
30. Goodman R, Yude C. IQ and its predictors in childhood hemiplegia. Dev Med Child Neurol 1996; 38: 881-890.
31. Tajik-Parvinchi D, Farmus L, Tablon Modica P, et al. The role of cognitive control and emotion regulation in predicting mental health problems in children with neurodevelopmental disorders. Child Care Health Dev 2021; 47: 608-617.
32. Coceski M, Hocking DR, Abu-Rayya HM, et al. WISC-V motor-free cognitive profile and predictive factors in adolescents with cerebral palsy. Res Dev Disabil 2021; 113: 103934.
33. McMorris CA, Lake J, Dobranowski K, et al. Psychiatric disorders in adults with cerebral palsy. Res Dev Disabil 2021; 111: 103859.
34. Bornstein MH. Sensitive periods in development: structural characteristics and causal interpretations. Psychol Bull 1989; 105: 179-197.
35. Hebb DO. The innate organization of visual activity: I. Perception of figures by rats reared in total darkness. J Gen Physiology 1937; 51: 101-126.
36. Hubel DH, Weisel TN. The period of susceptibility to physiological effects of unilateral eye closure in kittens. J Physiol (London) 1970; 206: 419-436.
37. Rutter M, Rutter M (eds.). Developing minds: challenge and continuity across the lifespan. Penguin Books, London 1992, 69-71.
38. DeCasper A, Spence MJ. Prenatal maternal speech influences newborns' perception of speech sounds. Infant Behavior Develop 1986; 9: 133-150.
39. Woods BT, Teuber HL. Changing patterns of childhood aphasia. Ann Neurol 1978; 3: 273-280.
40. Craig F, Savino R, Trabacca A. A systematic review of comorbidity between cerebral palsy, autism spectrum disorders and Attention Deficit Hyperactivity Disorder. Eur J Paediatr Neurol 2019; 23: 31-42.
41. Påhlman M, Gillberg C, Wentz E, et al. Autism spectrum disorder and attention-deficit/hyperactivity disorder in children with cerebral palsy: results from screening in a population-based group. Eur Child Adolesc Psychiatry 2020; 29: 1569-1579.
42. Bjorgaas HM, Hysing M, Elgen I. Psychiatric disorders among children with cerebral palsy at school starting age. Res Dev Disabil 2012; 33: 1287-1293.
43. Tajik-Parvinchi D, Davis A, Roth S, et al. My Story Study Group. Functional connectivity and quality of life in young adults with cerebral palsy: a feasibility study. BMC Neurol 2020; 20: 388.
44. Smith KJ, Peterson MD, Victor C, et al. Risk of dementia in adults with cerebral palsy: a matched cohort study using general practice data. BMJ Open 2021; 11: e042652.

45. Vaughan G, Kutzberg D. Electrophysiological indices of human brain maturation and cognitive development. In: Gunnar M, Nelson C (eds.). Developmental behavioral neuroscience: the Minnesota symposia on child psychology. Erlbaum, Hillsdale, New York 1992, 24, 1-36.
46. Assaf AA. The sensitive period: transfer of fixation after occlusion for strabismic amblyopia. Br J Ophthalmol 1982; 66: 64-70.
47. Woods BT, Teuber HL. Changing patterns of childhood aphasia. Ann Neurol 1978; 3: 273-280.
48. Yakovlev P, Lecours A. The myelogenetic cycles of regional maturation of the brain. In: Minkowski A (ed.). Regional development of the brain in early life, Blackwell, Oxford 1967, 3-64.
49. Huttenlocher PR. Synaptogenesis, synapsę elimination, and neural plasticity in the human cerebral cortex. In: nelson CA (ed.). Threats to optimal development: Integrating biological, psychosocial and social risk factors: the Minnesota symposia on child psychology. Erlbaum, Hilsdale, New York 1994; 27: 35-54.