Cognitive functioning in children with cerebral palsy

KRISTINE STADSKLEIV [1,2]

1 Department of Clinical Neurosciences for Children, Oslo University Hospital, Oslo; **2** Department of Educational Science, University of South-Eastern Norway, Vestfold, Norway.

Correspondence to Kristine Stadskleiv, Oslo University Hospital, Department of Clinical Neurosciences for Children, Post Box 4950 Nydalen, N-0424 Oslo, Norway.
E-mail: kristine.stadskleiv@oslo-universitetssykehus.no

PUBLICATION DATA
Accepted for publication 6th December 2019.
Published online

ABBREVIATION
PVL Periventricular leukomalacia

Children with cerebral palsy (CP) have an increased risk of cognitive impairments. This narrative review of the literature discusses assessment of cognition in children with CP, presents the most salient characteristics of cognitive functioning pertaining to each subtype, and discusses the relationships between brain injury, functioning, and intervention from a developmental perspective. A search for original studies of cognitive functioning in children with different subtypes of CP was performed. The search resulted in 81 unique hits. There were few studies with a representative sample of children with CP where all participants were individually assessed. Cognitive functioning in children with the most severe motor impairments were often assumed and not assessed. Furthermore, there was a confounding of IQ below 70 and intellectual disability, possibly leading to an overestimation of the prevalence of intellectual disability. Longitudinal neuropsychological studies, including also very young children and those with the most severe speech and motor impairments, as well as intervention studies, are called for.

The motor impairments of children with cerebral palsy (CP), caused by an inborn or early acquired brain lesion,[1] are often accompanied by impaired functioning in other areas, such as cognition. The scope of cognitive impairments varies between and within the spastic, dyskinetic, and ataxic subtypes.[2] The identification of cognitive impairments depends upon the quality of the assessments, and the first aim of this study is to review challenges in that regard. In this review, cognitive functioning in children with CP will be discussed from a developmental perspective, on the basis of a review of the literature on cognitive functioning in children with CP.

Assessment of cognition

Two aspects of assessment of cognition need to be addressed: the paucity of studies where a representative sample of all subtypes have been assessed and the challenges involved in reliably assessing cognition in children with impairments that makes test performance difficult. The latter is partly responsible for the former. The Gross Motor Function Classification System (GMFCS),[3] a 5-point ordinal scale where level I indicates the least impairment, is typically used to classify motor impairment in the literature discussing cognitive functioning. However, classification of fine motor functioning would have been even more relevant.

There are some epidemiological studies based on data from CP registries[4–6] and geographical cohorts,[7–10] but few where a representative sample is individually assessed.[11,12] Without assessment, IQ was estimated on the basis of clinical judgement,[4,5] school placement,[13] degree of gross motor impairment,[10] or interview with parents.[8]

Assessing cognition in children with motor impairments, including those who are able to perform the tasks of a standardized test of intelligence, is not straightforward. Even small fine motor impairments might influence test scores negatively and lead to an underestimation of IQ.[14] Results from timed tests must therefore be interpreted with care, even for children in GMFCS level I. It is even more challenging to assess cognition in children with severe speech and motor impairments.[15] The result is that one-third of children in GMFCS levels IV and V are assessed, also in studies aiming to assess a representative sample of children with CP.[12,16]

The challenges of assessing cognition in the severely affected group leads to cognitive functioning being assumed. Although a correlation between severity of motor and cognitive impairments exists, there is no absolute correspondence;[9,11] it is therefore not possible to draw conclusions about cognition from functioning in other areas. Furthermore, it is not necessary as cognition can be assessed using tests with a multiple-choice format and allowing for other means of responding than pointing with a finger. Tests of verbal comprehension, such as the Peabody Picture Vocabulary Test[17] and the Test for Reception of Grammar,[18] and non-verbal reasoning, such as Raven's matrices,[19] are suitable for adaptation. Alternative response modes include gaze pointing and scanning (Table S1, online supporting information). Comparing standard and alternative response modes, partner-assisted

 DOI: 10.1111/dmcn.14463

scanning,[20] scanning with switches on a computer,[21] the use of frames for gaze pointing,[22,23] and gaze pointing on a computer[20,24–26] have not been found to influence test results. Despite this, children with the most severe motor impairments are described as non-assessible, or test results are provided without information about how tests were adapted.[27]

METHOD

A systematic search of the databases PsycINFO, ERIC, and MEDLINE (Ovid) was performed on 20th to 22nd February 2019. The terms (cognition OR intelligence) were sequentially combined with AND (hemipleg* OR unilateral); AND (diplegi*); AND (quadriplegi*); AND (dyskinetic OR dyskinesia); AND (ataxi*). The search was limited to human children (0–18y) and papers published in English. All abstracts of the 525 hits were browsed. Duplicates, papers not reporting on CP or cognitive functioning, papers published before 1990 (when the International Classification of Diseases, 10th Revision was published), papers where the full text was not available, literature reviews, and case studies were removed, resulting in 111 hits (35 papers on hemiplegia, 31 on diplegia, 16 on quadriplegia, 21 on dyskinesia, and eight on ataxia). Some included information about more than one subtype, leaving 75 unique hits. Two papers, about adults, were removed. Eight papers, which turned up browsing reference lists, were added. From the remaining 81 papers, information about age and subtypes, cognitive areas assessed, and main findings were extracted (Table S2 and Figure S1, online supporting information).

RESULTS

Cognitive impairments can be global (expressed as a low IQ score) or specific (pertaining to only one cognitive domain).

Global cognitive impairment

There is wide variability in estimates on the proportion of children with CP having an IQ less than 70, but Western countries with national registries report about 30% to 40%.[4,5,9] Spastic quadriplegia, epilepsy, severe motor impairment, and brain malformations are associated with more severe cognitive impairments.[7,9,11] Disorders of intellectual development can be considered with an IQ less than 70, but the diagnostic criteria[2] specifies that there should also be significant impairment in mastery of everyday activities. Difficulties with the latter should not be solely attributable to motor impairment, which complicates the diagnostic process for children in GMFCS levels III to V as there is a lack of appropriate tools for assessing adaptive functioning.[28] Further, although a full-scale IQ score might be below 70, it would not be advisable to diagnose intellectual disability if the profile is skewed and functioning is as expected for age in one or more areas. In the only study differentiating between IQ less than 70 and intellectual disability, it was found that although 33% had an IQ less than 70, only 25% qualified for a diagnosis of intellectual disability.[11] Reporting only IQ and making assumptions about proportion with intellectual disability on this basis may lead to an overestimation of the prevalence of intellectual disability in the population with CP.

What this paper adds

- Few studies have assessed cognition in a representative sample of children with cerebral palsy.
- Cognition in children with severe motor impairment is often assumed, not assessed.
- Lack of assessment may lead to overestimating the prevalence of intellectual disability.
- Lowered cognitive functioning in older children highlights the need for longitudinal studies.

Giftedness

Children with CP have an increased risk of cognitive impairments compared with peers, but reporting only this leaves out an important part of the picture. The finding[11,29] that children with CP can obtain IQ scores above 120 indicates that some are gifted and need follow-up accordingly.

Unilateral spastic CP

Most children with unilateral CP have normal cognition: 81% to 89% are reported to have an IQ greater than 70.[4,9,11] IQ is not significantly different in children with left- and right-sided paresis, or in children born preterm and at term.[30–32] Epilepsy is associated with lower IQ.[32,33] One-third have specific learning impairments,[34] including impairment in visual–spatial cognition,[35] acquisition of visual imagery,[36] and executive functioning.[37] The language functioning in children with right-sided paresis illustrates the plasticity of the developing brain. Contrary to what would happen if adults sustained similar focal brain injuries in the left hemisphere, language is often spared[34] and there is no difference in verbal IQ between children with left versus right unilateral brain lesions.[30] However, this right hemispheric reorganization of language comes with a cost, as it is associated with lower performance IQ.[38]

Bilateral spastic CP: diplegia

Typically, 67% to 78% of children with diplegia are reported to have an IQ greater than 70.[9,11] When the cause of diplegia is periventricular leukomalacia (PVL), IQ is reported to be similar in children born preterm and at term.[39] However, in children born at term and with varied aetiology, as few as 39% have an IQ greater than 70.[40]

Typically, children with diplegia have a skewed profile with normal verbal comprehension and impaired visual–spatial reasoning and non-verbal intelligence.[9,41–44] Even though this profile can be observed in 3-year-olds,[42] the difference becomes more pronounced as children enter school.[45] PVL affects the brain connectivity in the temporal–parietal cortex,[46] is particularly frequent in children born preterm,[47] and leads to visual–perceptual impairments.[48]

The relationships between ophthalmological impairments, the extent of white matter injury, visual–perceptual impairment, and non-verbal reasoning are difficult to disentangle, and lack of consensus on definition of core concepts and variability in measurement methods makes comparison across studies challenging. Different concepts, such as cerebral visual impairment (defined as damage to or dysfunction of the retrochiasmatic visual pathways[49]) and visual–perceptual impairment (defined as an impairment in the ability to analyse and process visual information[50]), are sometimes used interchangeably, confounding neurological and functional levels of description.[48]

In children with diplegia, visual–perceptual impairments have been found to be particularly pronounced in those born preterm.[39] For this group, the degree of visual–perceptual impairment is related to white matter thinning.[51] However, PVL has been found not necessarily to lead to visual–perceptual impairment.[52] Performance IQ (which also includes visual–spatial perception, in addition to other abilities such as non-verbal reasoning and processing speed) in those born preterm correlates with white matter integrity.[53] In children born at term, full-scale IQ is related to white matter integrity and severity of PVL;[39,54] possibly because severe cognitive impairments are more common and thus full-scale IQ correlates with the extent of the brain lesions. The presence of ophthalmological impairments has been reported both to be[39] and not to be associated with or fully explain visual–perceptual impairment[41,55] or constructional dyspraxia.[43]

Visual–spatial impairments and performance IQ are by far the most studied functions in children with diplegia, but specific impairments in attention and executive function have also been reported,[44,56] especially if there is damage to the anterior corpus callosum in addition to other white matter lesions.[41] Verbal cognition and memory for verbally presented materials are reported to be as expected for age.[41]

Bilateral spastic CP: quadriplegia

In the subgroup with spasticity there is a correlation between degree of motor and cognitive impairment, and up to 90% to 100% with quadriplegia are reported to have an IQ less than 70.[5,57] However, as formal testing is often reported to be inaccessible to children with quadriplegia,[12,58] this might represent an overestimate of impairment. Studies in which all children were individually assessed report lower frequencies, around 65%.[9,11] This implies that there could be more children with quadriplegia who have an IQ in the normal range.[9,11,59] Documenting the capabilities of children with quadriplegia requires adapted testing. It has been found that 20% had an IQ greater than 85,[9] illustrating that severe motor impairments can mask cognitive skills.

Dyskinesia

In this second largest subtype, constituting 6% to 15% of the total population with CP,[4,12] wide variability in standardized scores on tests of non-verbal reasoning (20–129) and verbal comprehension (55–119) is reported.[60,61] Often 50% to 60% are reported to have an IQ less than 70,[4,5,9] but it varies between as few as 25%[11,62] and as many as 70% to 80%.[8,63] As in spastic CP, normal cognition is also found in those with the most severe motor impairments.[64] In children with dyskinetic CP this is to be expected, as magnetic resonance imaging (MRI) studies show that lesions in subcortical areas, affecting extrapyramidal pathways, are common.[65]

Studies of cognition including only children with dyskinetic CP are rare, but show that visual perception, language, memory, and executive functions often are as expected for age.[60,66] Non-vocal children (i.e. children who are not able to use speech as mode of communication) struggle with literacy despite normal cognition.[62] Recently, executive functioning has been the focus in studies of the dyskinetic subtype,[29,61] but it has not been confirmed that they struggle more in this area than those in the subgroup with spasticity.[11,66]

Ataxia

Ataxic CP constitutes around 5% to 6% of the CP group,[4,9] and 42% to 67% are reported to have an IQ less than 70.[4,5,8,9] No studies have focused solely on this group, and knowledge about specific impairments is therefore lacking.

DISCUSSION

Brain lesions and cognitive functioning

Recent MRI studies, particularly those using diffusion tensor imaging which allows more precise descriptions of white matter tracts, have advanced our understanding of the complex relationships between brain lesions and functioning.[27] An acute and severe intrapartum hypoxic–ischaemic insult at term has been linked to dyskinetic CP, while diplegia is often seen in children born preterm with PVL.[67] However, a lesion is visible on MRI in only 85% to 90% of children with CP.[47,68] All types of lesion (brain malformations and white and grey matter lesions) are found in all subtypes.[47] In children with the same type of lesion, cognition has been found to vary between normal and severe intellectual disability.[69] Studies are mixed in their reporting of correlations between MRI findings and IQ,[39,40,54] implying that cognitive functioning cannot be confidently predicted from these scanning technologies. Acute severe perinatal hypoxia–ischaemia can lead to cognitive deficits and no motor impairments.[67] It is well established that in children with unilateral left hemispheric lesions, language might be reorganized in the right hemisphere and might be their strongest skill.[34,38] However, the developing brain is vulnerable if the lesion is more extensive. In a study of non-vocal children with bilateral CP, the language tracts were visualized using diffusion tensor imaging in the right hemisphere for all five patients regardless of their level of verbal comprehension, but was not visible in the left hemisphere for one child without any

comprehension of spoken language.[70] Not only the localization and extent of the brain lesion, but also the aetiology, must be taken into consideration. When the cause is cerebral dysgenesis, more have severe cognitive impairments.[11,71] This might possibly be linked to epilepsy: more children with a cerebral dysgenesis have epilepsy[71] and epileptiform activity is negatively correlated with intellectual functioning.[11,30]

Preterm birth, especially if resulting in infarction, is associated with CP and a risk of cognitive impairment.[72] However, in children with CP earlier gestational age does not necessarily imply more cognitive challenges. In bilateral spastic CP, the proportion of individuals with severe cognitive impairment increased with increasing gestational age,[73] and also no relation between cognition and gestational age has been reported.[11]

Together, this implies that cognitive functioning cannot be inferred from MRI findings alone, nor from information about brain lesion, epilepsy, gestational age at birth, and motor functioning. Instead, the initial brain lesion can be viewed as a constraint on development. Cognitive impairments are the result of reciprocal and continuing interactions between the child and their environment, influenced by the child's opportunities for active exploration and participation.[74] Knowledge about the risk factors is important because it can lead to awareness about the need for assessment and interventions, and aid in developing follow-up programmes.[75]

Developmental trends

The panorama of CP has changed over recent decades, with a lower prevalence, proportion of children developing bilateral spastic CP, and incidence of intellectual disability.[76] However, knowledge about the developmental trajectories of cognition in children with CP is less clear, as there are few longitudinal studies. Of the 81 identified studies included in this review, only nine had a longitudinal design.[12,13,30,39,42,45,77–79]

These studies show that measures of cognitive functioning at 12 and 18 months of age correlate,[77] that the skewed cognitive profile of school-aged children with spastic bilateral CP is observable from 3 years of age,[42] and that after entering school there is an increase in verbal IQ so that it becomes age-average while performance IQ continues to be in the low range.[45,79] There is a differential development of non-verbal reasoning capacity in children in different GMFCS levels: children in level V not only show initial lower functioning but also increase less with development.[12] For children with unilateral CP, IQ was stable from 3 to 5 years of age,[30] while children with the most severe speech and motor impairments did not show the expected increase in non-verbal reasoning from 6 to 12 years of age despite normal cognition.[78] The development of expressive communication is related to the type of motor impairment, whereas receptive communication is related to IQ.[13] Following children born preterm and at term, significantly more children with spastic diplegia born preterm were found to develop visual–perceptual impairments.[39]

Developmental model

The most troubling finding is the lack of age-expected increases in cognitive development in children with severe speech and motor impairments.[78] This might be explained by the brain lesion, but children with motor impairments also have different experiential backgrounds compared with those who are not restricted in their locomotion. In children restricted in their locomotion, both localization of a brain lesion and restricted upper-limb functioning explain why action-based visual perception is more demanding than object-based perception.[80] In children expressing themselves using aided communication, their instructions on a construction task included little information about sizes and spatial relations,[81] further suggesting that allocentric strategies are particularly challenging for severely motor-impaired children. An interaction between the brain lesion and lack of appropriate experiences seems likely.[74] Applying an embodied cognition framework, the 'mind must be understood in the context of its relationship to a physical body that interacts with the world'.[82] Findings supporting this position are that: (1) action planning, which implies that we consider the end point of a movement from the start, is challenging for children with CP and does not improve with age as would be expected;[83] (2) finger gnosis is important for early numeracy skills, implying that the use of hands, the understanding of numbers, and the perception of space are related;[84] and (3) constructional dyspraxia in children with CP cannot be related to visual and visual–perceptual impairments.[43] Thus, the consequences of an initial impairment might be 'wide reaching with cascading developmental effect on other abilities',[85] if measures are not put in place to counteract and minimize the developmental consequences.

Interventions for children with cognitive impairments

It is well established that early interventions are beneficial for alleviating motor impairments in children with CP, but the effect of cognitive training has scarcely been studied.[86] Also, most studies aim at improving literacy, despite visual–spatial and attentional impairments being more frequent than language impairments.[27] In a study of executive functioning, no effect of training was found.[87] This does not imply that one should do nothing. Children with CP attending mainstream school had significantly better progress in mathematics and reading than those attending a special school, despite identical verbal IQ. The finding that the groups differed in the amount of the teaching received, with the children in the mainstream school receiving 1.7 times more, emphasizes the importance of interventions.[88,89] For the non-vocal group, it is imperative to provide augmentative and alternative communication as early as possible. Otherwise they might be seriously hampered in their development of communication and language and

have severely restricted opportunities for interaction and participation,[90] which in turn might have negative cascading effects on their social, academic, emotional, and cognitive development.

CONCLUSION

There is a wealth of studies on motor functioning in children with CP. However, even though parents report that learning difficulties are at least as challenging and cognition plays a greater role for communication, academic functioning, participation, and social functioning, it has been less focused upon.[59,91]

This review illustrates that there are gaps to be filled; few studies have assessed cognitive profiles in a large representative population of children with CP, including also very young children and those with the most severe speech and motor impairments, and there is a need for longitudinal and intervention studies. Some areas, such as visual–spatial abilities and language, are much more focused on, while others, such as memory, are less well researched. This might be because the few studies on memory have not reported specific challenges;[41,44] however, as these studies only include children with milder motor impairments, further research seems warranted.

There seems to be an increasing focus on finding associations between extent and localization of brain lesions and cognitive functions; however, given the evidence of early plasticity as well as the heterogeneity of cognitive functioning in children with similar MRI lesions, it might be questioned whether this is the most useful path forwards. The interplay between brain lesions, sensory deficits, experiential opportunities, and cognitive functioning is complex. It has been investigated whether developmental disregard in children with hemiplegia, a neglect-like disregard of their affected upper limb, is the result of injury to neural networks involved in spatial attention, which are connected to areas involved in motor planning, or the result of lack of use of affected hands during important developmental periods. Monitoring event-related potential during task performance, specific impairments in executive functioning were not found, but general difficulties with performing were. This implied that the executive control processes preceding the motor response were affected, requiring an enhanced cognitive effort in goal-directed behaviour and a developmental delay of executive control mechanisms.[92] The same complexity is found when investigating the relationships between visual perception and cognition. Visual–perceptual impairment has been reported to be associated with lower cognitive functioning, found in children with normal cognition, and unrelated to non-verbal cognitive functioning.[52,93,94]

Future studies of cognitive development and the effect of interventions should therefore take the complex interplay over time between body, brain, and mind into account. Tests need to be adapted, for example using eye-gaze technologies, so that cognitive functioning can be reliably assessed, and not only assumed, in the most severely motor-impaired children.[20,24–26] In the future, brain–computer interfaces might gain importance both for assessment and interventions.[95] Furthermore, it might be that not only traditional neuropsychological tests and computerized training tasks, but more naturalistic tests and tasks increasing real-life abilities, such as goal-setting and planning abilities, are needed.

SUPPORTING INFORMATION

The following additional material may be found online:

Table S1: Review of studies of adapted assessment of cognition.

Table S2: Eighty-one studies of cognitive functioning in children with cerebral palsy.

Figure S1: PRISMA 2009 flow diagram.

REFERENCES

1. Rosenbaum P, Paneth N, Leviton A, et al. A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol* 2007; **49**(Suppl 109): 8–14.
2. World Health Organization. International Classification of Diseases 11th Revision Geneva. Switzerland: World Health Organization, 2018.
3. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. *Dev Med Child Neurol* 1997; **39**: 214–23.
4. Andersen GL, Irgens LM, Haagaas I, Skranes JS, Meberg AE, Vik T. Cerebral palsy in Norway: prevalence, subtypes and severity. *Eur J Paediatr Neurol* 2008; **12**: 4–13.
5. Himmelmann K, Beckung E, Hagberg G, Uvebrant P. Gross and fine motor function and accompanying impairments in cerebral palsy. *Dev Med Child Neurol* 2006; **48**: 417–23.
6. Hammal D, Jarvis SN, Colver AF. Participation of children with cerebral palsy is influenced by where they live. *Dev Med Child Neurol* 2004; **46**: 292–8.
7. Murphy CC, Yeargin-Allsopp M, Decouflé P, Drews CD. Prevalence of cerebral palsy among ten-year-old children in metropolitan Atlanta, 1985 through 1987. *J Pediatr* 1993; **123**: S13–20.
8. Türkoğlu G, Türkoğlu S, Çelik C, Uçan H. Intelligence, functioning, and related factors in children with cerebral palsy. *Arch Neuropsychiatry* 2017; **54**: 33–7.
9. Sigurdardottir S, Eiriksdottir A, Gunnarsdottir E, Meintema M, Arnadottir U, Vik T. Cognitive profile in young Icelandic children with cerebral palsy. *Dev Med Child Neurol* 2008; **50**: 357–62.
10. Hutton J, Pharoah P. Effects of cognitive, motor, and sensory disabilities on survival in cerebral palsy. *Arch Dis Child* 2002; **86**: 84–9.
11. Stadskleiv K, Jahnsen R, Andersen GL, von Tetzchner S. Neuropsychological profiles of children with cerebral palsy. *Dev Neurorehabil* 2018; **21**: 108–20.
12. Smits D, Ketelaar M, Gorter J, et al. Development of non-verbal intellectual capacity in school-age children with cerebral palsy. *J Intellect Disabil Res* 2011; **55**: 550–62.
13. Vos RC, Dallmeijer AJ, Verhoef M, et al. Developmental trajectories of receptive and expressive communication in children and young adults with cerebral palsy. *Dev Med Child Neurol* 2014; **56**: 951–9.
14. Sherwell S, Reid SM, Reddihough DS, Wrennall J, Ong B, Stargatt R. Measuring intellectual ability in children with cerebral palsy: Can we do better? *Res Dev Disabil* 2014; **35**: 2558–67.
15. Foo RY, Guppy M, Johnston LM. Intelligence assessment for children with cerebral palsy: a systematic review. *Dev Med Child Neurol* 2013; **56**: 911–8.

16. Romeo DM, Cioni M, Battaglia LR, Palermo F, Mazzone D. Spectrum of gross motor and cognitive functions in children with cerebral palsy: gender differences. *Eur J Paediatr Neurol* 2011; **15**: 53–8.
17. Dunn LM, Dunn DM. Peabody Picture Vocabulary Test (PPVT-4). Minneapolis, MN: Pearson Assessment, 2007.
18. Bishop D. Test for Reception of Grammar (2nd edition) (TROG-2). London, UK: Pearson Assessment, 2003.
19. Raven JC. Raven's Coloured Progressive Matrices. London, UK: Pearson Assessment, 1998.
20. Kurmanaviciute R, Stadskleiv K. Assessment of verbal comprehension and non-verbal reasoning when standard response mode is challenging: a comparison of different response modes and an exploration of their clinical usefulness. *Cog Psychol* 2017; **4**: 1275416.
21. Miller P. Use of the Peabody Picture Vocabulary Test - Revised (PPVT-R) with individuals with severe speech and motor impairment: effect of response mode on test results. Ann Arbor, MI: University Microfilms International: University of Kansas, 1990.
22. Spillane MM, Ross KK, Vasa SF. A comparison of eye-gaze and standard response mode on the PPVT-R. *Psychol School* 1996; **33**: 265–71.
23. Casey M, Tonsing KM, Alant E. Comparison of a non-spoken response mode and a spoken response mode in a test of phonological awareness. *South African J Occup Ther* 2007; **37**: 25–8.
24. Warschausky S, Van Tubbergen M, Asbell S, Kaufman J, Ayyangar R, Donders JJA. Modified test administration using assistive technology: preliminary psychometric findings. *Assessment* 2012; **19**: 472–9.
25. Thurén R. Assessment Tools for Eye Tracker: Developing a Prototype of a Test of Reception of Language Using Eye Tracker. Stockholm, Sweden: Royal Institute of Technology, 2010.
26. Geytenbeek JJ, Mokkink LB, Knol DL, Vermeulen RJ, Oostrom KJ. Reliability and validity of the C-BiLLT: a new instrument to assess comprehension of spoken language in young children with cerebral palsy and complex communication needs. *Augment Altern Commun* 2014; **30**: 252–66.
27. Gosling AS. Recent advances in the neuroimaging and neuropsychology of cerebral palsy. *J Appl Neuropsychol Child* 2017; **6**: 55–63.
28. James S, Ziviani J, Boyd R. A systematic review of activities of daily living measures for children and adolescents with cerebral palsy. *Dev Med Child Neurol* 2014; **56**: 233–44.
29. Laporta-Hoyos O, Pannek K, Ballester-Plané J, et al. White matter integrity in dyskinetic cerebral palsy: relationship with intelligence quotient and executive function. *NeuroImage Clini* 2017; **15**: 789–800.
30. Muter V, Taylor S, Vargha-Khadem F. A longitudinal study of early intellectual development in hemiplegic children. *Neuropsychologia* 1997; **35**: 289–98.
31. Riva D, Franceschetti S, Erbetta A, Baranello G, Esposito S, Bulgheroni S. Congenital brain damage: cognitive development correlates with lesion and electroencephalographic features. *J Child Neurol* 2013; **28**: 446–54.
32. Zelnik N, Lahat E, Heyman E, et al. The role of prematurity in patients with hemiplegic cerebral palsy. *J Child Neurol* 2016; **31**: 678–82.
33. Vargha-Khadem F, Isaacs E, van der Werf S, Robb S, Wilson J. Development of intelligence and memory in children with hemiplegic cerebral palsy: the deleterious consequences of early seizures. *Brain* 1992; **115**: 315–29.
34. Frampton I, Yude C, Goodman R. The prevalence and correlates of specific learning difficulties in a representative sample of children with hemiplegia. *Br J Educ Psychol* 1998; **68**: 39–51.
35. Carlsson G, Uvebrant P, Hugdahl K, Arvidsson J, Wiklund LM, von Wendt L. Verbal and non-verbal function of children with right-versus left-hemiplegic cerebral palsy of pre-and perinatal origin. *Dev Med Child Neurol* 1994; **36**: 503–12.
36. Carlsson G. Memory for words and drawings in children with hemiplegic cerebral palsy. *Scand J Psychol* 1997; **38**: 265–73.
37. Bodimeade HL, Whittingham K, Lloyd O, Boyd RN. Executive function in children and adolescents with unilateral cerebral palsy. *Dev Med Child Neurol* 2013; **55**: 926–33.
38. Lidzba K, Staudt M, Wilke M, Krägeloh-Mann I. Visuospatial deficits in patients with early left-hemispheric lesions and functional reorganization of language: consequence of lesion or reorganization? *Neuropsychologia* 2006; **44**: 1088–94.
39. Pagliano E, Fedrizzi E, Erbetta A, et al. Cognitive profiles and visuoperceptual abilities in preterm and term spastic diplegic children with periventricular leukomalacia. *J Child Neurol* 2007; **22**: 282–8.
40. Numata Y, Onuma A, Kobayashi Y, et al. Brain magnetic resonance imaging and motor and intellectual functioning in 86 patients born at term with spastic diplegia. *Dev Med Child Neurol* 2013; **55**: 167–72.
41. Di Lieto MC, Brovedani P, Pecini C, et al. Spastic diplegia in preterm-born children: executive function impairment and neuroanatomical correlates. *Res Dev Disabil* 2017; **61**: 116–26.
42. Fedrizzi E, Inverno M, Botteon G, Anderloni A, Filippini G, Farinotti M. The cognitive development of children born preterm and affected by spastic diplegia. *Brain Dev* 1993; **15**: 428–32.
43. Koeda T, Inoue M, Takeshita K. Constructional dyspraxia in preterm diplegia: isolation from visual and visual perceptual impairments. *Acta Paediatrica* 1997; **86**: 1068–73.
44. Pirila S, van der Meere J, Korhonen P, et al. A retrospective neurocognitive study in children with spastic diplegia. *Dev Neuropsychol* 2004; **26**: 679–90.
45. Ito J-I, Araki A, Tanaka H, Tasaki T, Cho K. Intellectual status of children with cerebral palsy after elementary education. *Pediatr Rehabil* 1997; **1**: 199–206.
46. Pavlova M, Lutzenberger W, Sokolov AN, Birbaumer N, Krägeloh-Mann I. Oscillatory MEG response to human locomotion is modulated by periventricular lesions. *Neuroimage* 2007; **35**: 1256–63.
47. Arnfield E, Guzzetta A, Boyd R. Relationship between brain structure on magnetic resonance imaging and motor outcomes in children with cerebral palsy: a systematic review. *Res Dev Disabil* 2013; **34**: 2234–50.
48. Ego A, Lidzba K, Brovedani P, et al. Visual–perceptual impairment in children with cerebral palsy: a systematic review. *Dev Med Child Neurol* 2015; **57**: 46–51.
49. Fazzi E, Bova S, Giovenzana A, Signorini S, Uggetti C, Bianchi P. Cognitive visual dysfunctions in preterm children with periventricular leukomalacia. *Dev Med Child Neurol* 2009; **51**: 974–81.
50. Dutton GN. Terminology for brain injury-related vision loss: the debate continues. *J Vis Impair Blind* 2011; **105**: 133–4.
51. Koeda T, Takeshita K. Visuo-perceptual impairment and cerebral lesions in spastic diplegia with preterm birth. *Brain Dev* 1992; **14**: 239–44.
52. Ortibus E, Lagae L, Casteels I, Demaerel P, Stiers P. Assessment of cerebral visual impairment with the L94 visual perceptual battery: clinical value and correlation with MRI findings. *Dev Med Child Neurol* 2009; **51**: 209–17.
53. Fedrizzi E, Inverno M, Bruzzone MG, Botteon G, Saletti V, Farinotti M. MRI features of cerebral lesions and cognitive functions in preterm spastic diplegic children. *Pediatr Neurol* 1996; **15**: 207–12.
54. Rai Y, Chaturvedi S, Paliwal VK, et al. DTI correlates of cognition in term children with spastic diplegic cerebral palsy. *Eur J Paediatr Neurol* 2013; **17**: 294–301.
55. Fedrizzi E, Anderloni A, Bono R, et al. Eye-movement disorders and visual-perceptual impairment in diplegia children born preterm: a clinical evaluation. *Dev Med Child Neurol* 1998; **40**: 682–8.
56. Al-Nemr A, Abdelazeim F. Relationship of cognitive functions and gross motor abilities in children with spastic diplegic cerebral palsy. *Appl Neuropsychol Child* 2018; **7**: 268–76.
57. Dalvand H, Dehghan L, Hadian MR, Feizy A, Hosseini SA. Relationship between gross motor and intellectual function in children with cerebral palsy: a cross-sectional study. *Arch Phys Med Rehabil* 2012; **93**: 480–4.
58. Majnemer A, Shevell M, Hall N, Poulin C, Law M. Developmental and functional abilities in children with cerebral palsy as related to pattern and level of motor function. *J Child Neurol* 2010; **25**: 1236–41.
59. Majnemer A, Shevell M, Law M, Poulin C, Rosenbaum P. Level of motivation in mastering challenging tasks in children with cerebral palsy. *Dev Med Child Neurol* 2010; **52**: 1120–6.
60. Ballester-Plane J, Laporta-Hoyos O, et al. Measuring intellectual ability in cerebral palsy: the comparison of three tests and their neuroimaging correlates. *Res Dev Disabil* 2016; **56**: 83–98.
61. Laporta-Hoyos O, Ballester-Plané J, Póo P, et al. Proxy-reported quality of life in adolescents and adults with dyskinetic cerebral palsy is associated with executive functions and cortical thickness. *Qual Life Res* 2017; **26**: 1209–22.
62. Beal S, Zeitz H, Connell T, Zschorn M. Athetoid quadriplegia and literacy. *J Paediatr Child Health* 2000; **36**: 389–91.
63. Himmelmann K, Hagberg G, Wiklund L, Eek M, Uvebrant P. Dyskinetic cerebral palsy: a population-based

study of children born between 1991 and 1998. *Dev Med Child Neurol* 2007; **49**: 246–51.

64. Ballester-Plané J, Laporta-Hoyos O, Macaya A, et al. Cognitive functioning in dyskinetic cerebral palsy: its relation to motor function, communication and epilepsy. *Eur J Paediatr Neurol* 2018; **22**: 102–12.
65. Gosling AS. Recent advances in the neuroimaging and neuropsychology of cerebral palsy. *Appl Neuropsychol Child* 2017; **6**: 55–63.
66. Pueyo R, Junqué C, Vendrell P, Narberhaus A, Segarra D. Neuropsychologic impairment in bilateral cerebral palsy. *Pediatr Neurol* 2009; **40**: 19–26.
67. Rennie JM, Hagmann CF, Robertson NJ. editors. Outcome after intrapartum hypoxic ischaemia at term. *Semin Fet Neonat Med* 2007; **12**: 398–407.
68. Blair E. Epidemiology of the cerebral palsies. *Orthop Clin* 2010; **41**: 441–55.
69. Choi JY, Choi YS, Rha DW, Park ES. The clinical outcomes of deep gray matter injury in children with cerebral palsy in relation with brain magnetic resonance imaging. *Res Dev Disabil* 2016; **55**: 218–25.
70. Harlaar L, Pouwels PJ, Geytenbeek J, Oostrom K, Barkhof F, Vermeulen RJ. Language comprehension in young people with severe cerebral palsy in relation to language tracts: a diffusion tensor imaging study. *Neuropediatrics* 2013; **44**: 286–90.
71. Kitai Y, Haginoya K, Hirai S, et al. Outcome of hemiplegic cerebral palsy born at term depends on its etiology. *Brain Dev* 2016; **38**: 267–73.
72. Roze E, Van Braeckel KN, van der Veere CN, Maathuis CG, Martijn A, Bos AF. Functional outcome at school age of preterm infants with periventricular hemorrhagic infarction. *Pediatrics* 2009; **123**: 1493–500.
73. Hemming K, Colver A, Hutton JL, Kurinczuk JJ, Pharoah PO. The influence of gestational age on severity of impairment in spastic cerebral palsy. *J Pediatr* 2008; **153**: 203–8.
74. Bøttcher L. Children with spastic cerebral palsy, their cognitive functioning, and social participation: a review. *Child Neuropsychol* 2010; **16**: 209–28.
75. Bøttcher L, Stadskleiv K, Berntsen T, et al. Systematic cognitive monitoring of children with cerebral palsy–The development of an assessment and follow-up protocol. *Scand J Disabil Res* 2016; **18**: 304–15.
76. Hollung SJ, Vik T, Lydersen S, Bakken IJ, Andersen GL. Decreasing prevalence and severity of cerebral palsy in Norway among children born 1999 to 2010 concomitant with improvements in perinatal health. *Eur J Paediatr Neurol* 2018; **22**: 814–21.
77. Cioni G, Paolicelli PB, Sordi C, Vinter A. Sensorimotor development in cerebral-palsied infants assessed with the Uzgiris-Hunt scales. *Dev Med Child Neurol* 1993; **35**: 1055–66.
78. Dahlgren Sandberg A. Reading and spelling abilities in children with severe speech impairments and cerebral palsy at 6, 9, and 12 years of age in relation to cognitive development: a longitudinal study. *Dev Med Child Neurol* 2006; **48**: 629–34.
79. Pleacher MD, Vohr BR, Katz KH, Ment LR, Allan WC. An evidence-based approach to predicting low IQ in very preterm infants from the neurological examination: outcome data from the Indomethacin Intraventricular Hemorrhage Prevention Trial. *Pediatrics* 2004; **113**: 416–9.
80. Dennis M, Fletcher JM, Rogers T, Hetherington R, Francis DJ. Object-based and action-based visual perception in children with spina bifida and hydrocephalus. *J Int Neuropsychol Soc* 2002; **8**: 95–106.
81. Stadskleiv K, Batorowicz B, Massaro M, van Balkom H, von Tetzchner S. Visual-spatial cognition in children using aided communication. *Aug Alt Commun* 2018; **34**: 68–78.
82. Van Rooijen M, Verhoeven L, Steenbergen B. Early numeracy in cerebral palsy: review and future research. *Dev Med Child Neurol* 2011; **53**: 202–9.
83. Krajenbrink H, Crichton A, Steenbergen B, Hoare B. The development of anticipatory action planning in children with unilateral cerebral palsy. *Res Dev Disabil* 2019; **85**: 163–71.
84. Van Rooijen M, Verhoeven L, Steenbergen B. Working memory and fine motor skills predict early numeracy performance of children with cerebral palsy. *Child Neuropsychol* 2016; **22**: 735–47.
85. Critten V, Campbell E, Farran E, Messer D. Visual perception, visual-spatial cognition and mathematics: Associations and predictions in children with cerebral palsy. *Res Dev Disabil* 2018; **80**: 180–91.
86. Novak I, Mcintyre S, Morgan C, et al. A systematic review of interventions for children with cerebral palsy: state of the evidence. *Dev Med Child Neurol* 2013; **55**: 885–910.
87. M. Piovesana A, Ross S, Lloyd O, et al. Randomized controlled trial of a web-based multi-modal therapy program for executive functioning in children and adolescents with unilateral cerebral palsy. *Disabil Rehabil* 2017; **39**: 2021–8.
88. Jenks KM, van Lieshout EC, de Moor JM. Cognitive correlates of mathematical achievement in children with cerebral palsy and typically developing children. *Br J Educ Psychol* 2012; **82**: 120–35.
89. Jenks KM, de Moor J, van Lieshout EC, Maathuis KG, Keus I, Gorter JW. The effect of cerebral palsy on arithmetic accuracy is mediated by working memory, intelligence, early numeracy, and instruction time. *Dev Neuropsychol* 2007; **32**: 861–79.
90. von Tetzchner S. Introduction to the special issue on aided language processes, development, and use: an international perspective. *Augment Altern Commun* 2018; **34**: 1–15.
91. Schenker R, Coster WJ, Parush S. Neuroimpairments, activity performance, and participation in children with cerebral palsy mainstreamed in elementary schools. *Dev Med Child Neurol* 2005; **47**: 808–14.
92. Zielinski IM, Steenbergen B, Baas CM, Aarts PB, Jongsma ML. Neglect-like characteristics of developmental disregard in children with cerebral palsy revealed by event related potentials. *BMC Neurol* 2014; **14**: 221.
93. Schenk-Rootlieb A, Van Nieuwenhuizen O, Schiemanck N, Van der Graaf Y, Willemse J. Impact of cerebral visual impairment on the everyday life of cerebral palsied children. *Child Care Health Dev* 1993; **19**: 411–23.
94. Fazzi E, Bova SM, Uggetti C, et al. Visual–perceptual impairment in children with periventricular leukomalacia. *Brain Dev* 2004; **26**: 506–12.
95. Kosmyna N, Lécuyer A. A conceptual space for EEG-based brain-computer interfaces. *PLoS One* 2019; **14**: e0210145.