

Cerebral Visual Impairment and Dysgenesis of Corpus Callosum in Multidisabled Children Aged 1 to 9 Years Old

Roxana CZIKER^{1*}, Tiberiu GUTTMAN², Benoit DELORME³, Andreea SECELEANU⁴, Adela JOANTA¹, Adriana MUREȘAN¹

¹ Department of Physiology, “Iuliu Hatieganu” University of Medicine and Pharmacy, 13 Emil Isac, Cluj-Napoca, Romania.

² Department of Medical Imaging, Private Clinic “Hiperdia” SA Imaging, 105A Republicii, Cluj-Napoca, Romania.

³ Department of Medical Imaging, Centre Hospitalier Universitaire d’Angers, 4 Larrey, France.

⁴ Ophthalmologist, Ophthalmologic Clinic, 3-5 Clinicilor, Cluj-Napoca, Romania.

E-mail(s): rcziker@yahoo.com; tguttman@yahoo.com; BeDelorme@chu-angers.fr; andreeaseceleanu@yahoo.com; adelaelena@yahoo.com. muresanadriana@yahoo.com

* Author to whom correspondence should be addressed; Roxana Cziker. Observatorului no. 13/37, Tel.: +4-0722-532576; Fax: +4-0264-431346.

Abstract: *Aims:* To emphasize the functional vision characteristics in visually impaired multiple disabled children (MDVI) aged 2 to 9 years old related to brain damages on magnetic resonance imaging in different cortical and subcortical areas and in the corpus callosum region. *Material and Method:* 12 MDVI children with severe and mild neurological disorders were medically and neuropsychological assessed. The clinical - psychological, neurological and ophthalmological – and paraclinical methods – visual evoked potential (VEP) and magnetic resonance imaging (MRI) were carried out in order to outline the complete profile of each child. The assessment was completed by morphometric measurement of corpus callosum and brain. *Results:* 10 of infants with severe neurological disorders showed ocular disorders such as ocular motility and visual function abnormalities. Severe cognitive and psychomotor retardation were associated in visual disorders in MDVI children. Significant correlation between neurological disorders, neuropsychological [$\tau(12) = 0.783$, $p = 0.001$] evaluation and visual acuity [$\tau(12) = 0.783$, $p = 0.001$] were found in multiple disabled children. The significant difference of diameter [$t(22) = -4.858$, $p = 0.000$] and surface of corpus callosum [$t(22) = -6.254$, $p = 0.000$] in multiple disabled children compared with control group was found. *Conclusion:* The structured assessment of visually impaired children due to neurological disorders, as early as possible, is the remarkably key which reveals the functionality of child and outlines the appropriate developmental and educational rehabilitation.

Keywords: Visual function; Cerebral visual impairment; Children with developmental delay; Dysgenesis of corpus callosum; Morphometry.

Introduction

The corpus callosum (CC) is the largest commissure in the brain and it is the major communication pathway between the two cerebral hemispheres. It consists of a thick plate of dense myelinated fibres that interconnect broad regions of cortex in all lobes, including the occipital lobes through the splenium. It is responsible of signals conduction between the homologous and heterotopic cortical regions and it is an essential component for brain lateralization and interhemispheric communication [1].

Structural changes were analyzed in several researches concerning its implication on a variety of neurological diseases such as dyslexia, schizophrenia, autism and unipolar and bipolar disorders. Few researches and results concerning the role of CC related to cerebral visual impairment (CVI) within the integration of visual-spatial and visual-perception information were achieved. The

implication of CC in visual brain function is still a matter of debate. Some researches on corpus callosum mentioned its role in information transfer within the occipital areas and into the visual and spatial perception [2-4]. Magnetic resonance imaging (MRI) is regarded as the best method to obtain cross-sectional area and shape information of CC, especially in the midsagittal sections.

One of the most common brain abnormalities in CC is the thinning, especially in the splenium area, which can be explained by the vulnerability of the developing of CC to hypoxic-ischemic damage, hemorrhage and hydrocephaly.

Due to the late development of the splenium and posterior area of CC, it could be explained why there is a particular susceptibility to damage in the second trimester and in the perinatal period. In general, injuries or problems which could be developed in the third trimester of foetus development or in the perinatal period could be an association of white matter damage, CC dysgenesis and increasing of lateral ventricular size area [5].

The research aim is to evaluate the relation between brain damage, dysgenesis of CC and visual functions in children with neurological disorders associated to CVI.

Materials and Method

Twelve multiple disabled visually impaired children (MDVI), 5 boys and 7 girls, aged between February 2000 and July 2005, with neurological disorders and different brain damages demonstrated on MRI were selected. Children with neurological disorders with risk of CVI, as severe and mild cerebral palsy and hydrocephaly were included, according with the criteria mentioned in the previous literature. Additional disorders and deficit functions such as motor disability, feeding difficulties, cognitive and language disorders were accepted.

A two years complete assessment programme was designed for all children. The program was *clinical* – neurological, ophthalmological, psychological and *para-clinical* – visual evoked potential (VEP) and magnetic resonance imaging (MRI) in order to emphasize the ocular features and the visual functions related to brain damages, especially dysgenesis of CC.

The neurodevelopmental screening tool was Oregon Project Screening Tests⁶. The real function of each child and possible delays on general development on different areas such as: cognitive, language, vision, compensation, fine and gross motor, social aspects and independency were assessed.

The ophthalmological assessment was based on functional and ocular objective exams: refractometry to determine the refractive errors of the eye, stereoscopic vision tests and ophthalmoscopy for the eye fundus. The assessment was completed by oculo-orbital ultrasonography. All those methods were used in order to exclude the possible vision disorders due to the anterior pole of the eye and to demonstrate the implication and the impact of brain damage into the visual information interpretation.

The electrical activity on visual system with *VEP paraclinical method* was revealed. The registration tool was Neurospectrum 5 with certificate ISO 9001, based on three channels: O1-Fz, O2-Fz și Oz-Fz. The registration band was between 0.5 and 100 Hz, with rectangular flash stimuli. The stimulation period was established at 30ms, at the frequency of 1Hz. A number of 50 stimuli/each stimulation was used.

The brain damage on *MRI paraclinical method* was put in evidence. The brain MRI studies were performed using 1.5 Tesla Siemens systems. After a marking section of T1_SE midsagittal, the exam consisted on: T1_SE_midsagittal, T2_TSE axial, T2 coronal dark fluid, T2_TSE midsagittal and T2 3D trufi transversal on sections of 0.70mm, 2mm and 5mm. The brain MRI studies were performed under sedation with DCI. The anaesthetics dosage was accordingly adjusted within the particularities of small children, in order to obtain accurate images and taking into consideration the severity of disability. The MRI exams were interpreted by two radiologists in order to have accurate results.

The dysgenesis of CC was put in evidence with *morphometrical method*. The morphometrical results of CC in disabled children were compared with the average of CC morphometry in 126 healthy children age 12 months to 9 years. They were selected in the Radiology Department of University Hospital Centre Angers, France. The size, thinning and the surface area of the CC for

both, disabled and children within the normal range, were analyzed on midsagittal T1-weighted section of 5mm, on T2-weighted dark-fluid section of 2mm. The "BioImage Suite 260" computer soft was used for brain and CC morphometrical analysis.

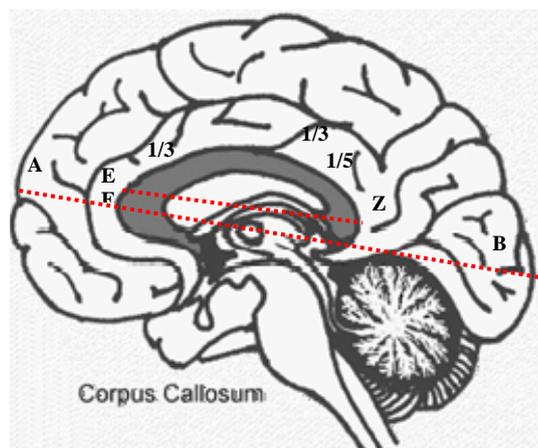


Figure 1. Diagram of the CC and different regions [2]

- AB** - the longitudinal diameter of the brain (frontal-occipital diameter);
- EZ** - the longitudinal diameter of corpus callosum;
- EZ/3** - the longitudinal diameter of the third portion of the CC;
- EZ/5** - the splenium longitudinal size
- EZ/AB** - the ratio of the frontal-occipital diameter and the longitudinal diameter of CC

The midsagittal image was defined as the image that clearly demonstrated a pituitary infundibulum, the cerebral aqueduct, the pineal gland and the CC in a single image. The CC was depicted by snake contour on midsagittal sections, by moving each point on the boundary between the cingulate gyrus, the cingulate sulcus and the lateral ventricles below. The measurements results were done on midsagittal slice and were identified using standard midline landmarks: callosal sulcus, cerebral aqueduct, pineal gland, the roof of the fourth ventricle and minimal gray matter in the interhemispheric fissure [7]. All measurements were obtained on the CC and excluded the fornix and pituitary rod. The ration of internal skull surface area and the surface area of CC (S-B/S-CC) were calculated (Figure 1). The ratio of 0.4 between the longitudinal diameter of CC and the frontal-occipital diameter was considered as reference value.

Statistical analysis was performed using SPSS software versions 15.00. The Kendal tau-b correlation between neuroradiological findings, results on neuropsychological test and visual functions parameters was proceed.

The difference between the morphometrical values of CC and brain in disabled children compared with normal children was performed with statistical "t" test for independent samples.

Results

Clinical Data - Population

Severe psychomotor and neurological disorders in association with visual disorders were revealed in 10 children (84%); the other two children (16%) have mild neurological disorders. The chronological age was bounded between 17 and 108 months (the average of 43.75 ± 25.08 months) and the gestational age between 24 and 42 weeks with an average of 37.92 ± 5.53 weeks.

Neuropsychological Assessment

Characteristics Related to Developmental Areas

Ten children out of 12 have severe motor problems and abnormal neurological examination, language and cognitive disorders. The other two children had normal results in motor, cognitive

and language screening, without severe neurological disorders. The psychological age on different developmental areas for 10 children was 2.3 months in average (Figure 2 and 3).

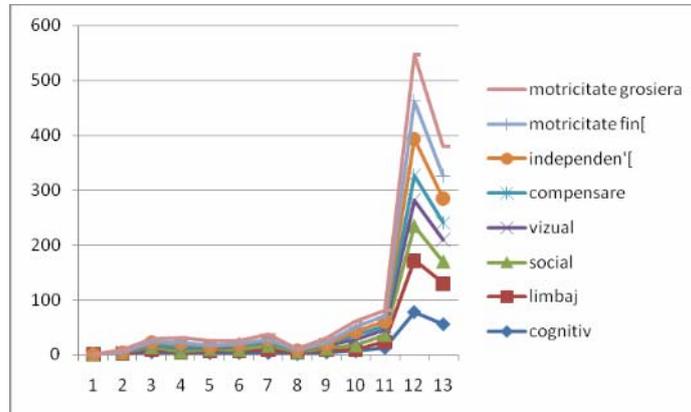


Figure 2. Graphic representation of children development related to different developmental areas

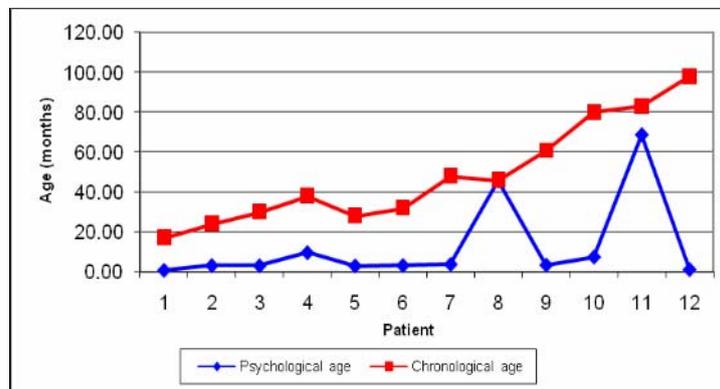


Figure 3. Graphic representation of psychological age in average related to chronological age on the Oregon Project Screening

A positive significant correlation between neurological disorders and neuropsychological development of children was found: $\tau(12) = 0.575, p = 0.029$ ($p < 0.05$). Each developmental area and neurological disorders were compared. Significant negative correlation was obtained among neurological disorders and cognitive development, socialization, visual, compensation, independency and find and gross motor function except language development (Table 1).

Table 1. Correlation between Oregon screening test and neurological disorders

	Oregon project results								
	Cog	Lang	Social	Vision	Compen	Indep	Fine mot	Gross mot	Global score
Neurological disorders – correlation coefficient	-0.677	-0.426	-0.562	-0.749	-0.868	-0.773	-0.711	-0.657	0.575
Neurological disorders – Sig. (2-tailed)	0.004	0.061	0.015	0.001	0.000	0.001	0.002	0.004	0.029

Characteristics of Visual Functions and Functional Vision

Ten children with severe neurological disorders demonstrated severe papillary constriction, ocular motility (visual fixation and pursuit), voluntary visual attention and visual contact dysfunctions and mild pale optic nerve papilla (Figure 4). Significant correlation between neurological disorders and optic nerve dysfunction was revealed for the value of $\tau(12) = 0.586, p =$

0.023 ($p < 0.05$). Two children have mild ocular pursuit and visual attention disorders. The capacity of convergence is absent in ten children and eight of them have strabismus and nystagmus. Neurological disorders is not a condition for production of strabismus and nystagmus in severe impaired children ($\tau(12) = -0.137$, $p = 0.588$ ($p > 0.05$)).

The visual acuity (VA) was poor for 10 out of 12 children. For 2 patients (28.6%) the VA was between the limit of 0.50 and 0.33, for 2 patients (14.3%) between 0.33 and 0.1, for 1 patient (7.1%) a value of 0.50, for 3 patients (21.4%) between 0.05 and 0.02 and for the rest of 4 patients between 0.02 and light perception. A strong positive significant correlation between neurological disorders and the value of VA at distance was obtained [$\tau(12) = 0.783$, $p = 0.001$ ($p < 0.01$)].

The contrast sensitivity values are very low with a medium of distance between 20 cm and 50 cm for the contrast of 5% for ten out of 12 children. The other two children had a value of contrast acuity in range of 5% at 1m. A positive correlation among contrast sensitivity and cortical-subcortical atrophy of white matter [$\tau(12) = 0.659$, $p = 0.009$ ($p < 0.01$)]. and cognitive development [$\tau(12) = 0.591$, $p = 0.017$ ($p < 0.05$)] was found.

Paraclinical Assessment – VEP, MRI and Morphometry of CC

The brain damage on MRI in areas involved into the transfer, integration and interpretation of visual information, in particularly CC related to visual data, such as: oculomotor dysfunctions, visual functions (visual acuity, grating acuity, contrast sensitivity) was revealed.

Visual Evoked Potential Results

The VEP results underlined partial atrophy of optic nerve in ten of severe disabled children. Optic nerve, visual pathways in the retrochiasmatic segment or on the left occipital cortex level dysfunctions were underlined for ten out of 12 children, all with severe neurological disorders. For two severe disabled children it was identified retrobulbar damage due to hydrocephaly (Figure 4).

The MRI Findings

The MRI findings and morphometry methods in case of MDVI children revealed the following aspects: *median brain region*: eleven of children (91%) had dysgenesis of CC, with reduction, in different degree, of the thinning of the total body; *ventricular system*: eleven of children (91%) with different degree of obstructive or non-obstructive ventricular dilatation; *white and gray matter*: cortico-subcortical atrophy (three cases – 25%), occipito-temporal atrophy (one case – 8%), left hemisphere atrophy (one case – 8%), fronto-parietal atrophy (one case – 8%) and gliosis with focal fronto-parietal cortical dysplasia (one case – 8%); *posterior fossa*: severe ponto – cerebellar atrophy (one cases – 8%), dysgenesis of cerebellar vermis (one case – 8%) and brain stem lesion (one case – 8%); *central gray nuclei*: atrophy of the central gray nuclei (four cases – 34%).

The Morphometry of CC

The longitudinal diameter, the thinning and surface area of CC and the fronto-occipital diameter and the surface area of the brain were measured for each child within the normal range (Figure 4). The average of each parameter was calculated according with each group (Table 2). The measured were achieved in order to have some bench-marks concerning the values within the normal population.

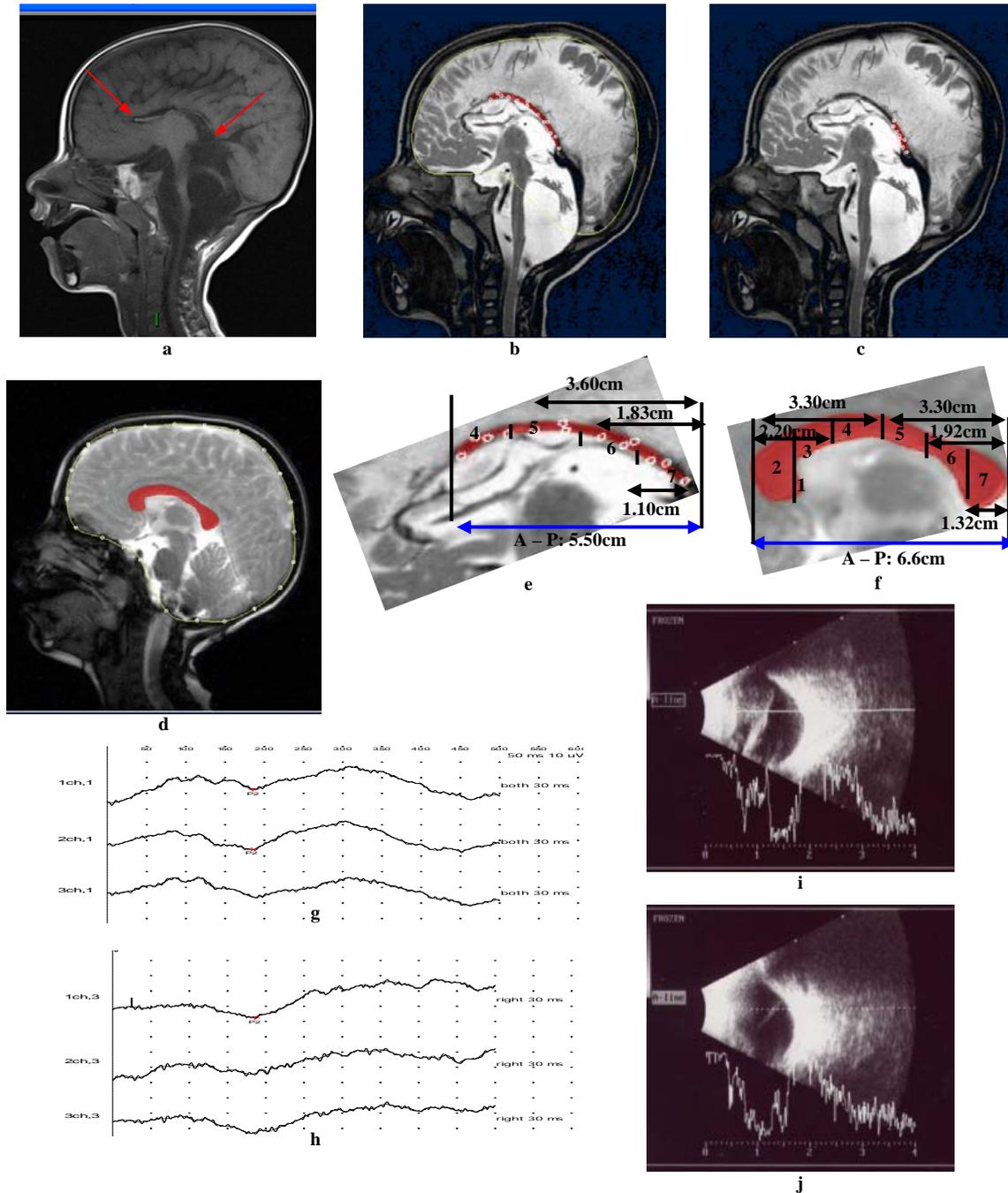


Figure 4. Case report. – clinical and paraclinical examinations: MRI, VEP, ocular ultrasonography and morphometry of CC of 2 years and 10 months old child with hydrocephaly, retinopathy of prematurity and detached retina, dysgenesis of CC, with lack of the genu and CVI. The psychological age determined with Oregon project is 3 months, with 21 months delayed against the chronological age. **a.** MRI T1-weighted, image at 5mm, midsagittal section – dysgenesis of CC and cerebellar hemispheres; **b.** MRI T2_tse_midsagittal image - the total surface area of CC; **c.** MRI T2_tse_midsagittal image – the surface of splenium is 78.1mm²; **d.** MRI T2-weighted image of the healthy CC of a 3 years old child **e.** the morphometry and segmentation of CC of disabled child; surface area of CC: 174.5 mm²; the surface area of the brain: 18567 mm²; the longitudinal diameter of CC: 5 cm; CC thinning: 0.17 cm; the frontal-occipital diameter: 13.59 cm; **f.** the average value of normal CC in a group of 3 years of age: surface area of CC: 651.32±68.80 mm²; the longitudinal diameter of CC: 6.29±0.44cm; thinning of CC: 0.60 cm; the frontal-occipital diameter: 15.36±0.80cm; the brain surface: 19219.24±2029.98mm²; **g.** VEP left eye – lack of visual information transfer through the optic nerve; **h.** PEV both eyes – lack of visual information transfer through the optic nerve; **i.** ocular ultrasonography left eye- total detached retina; **j.** ocular ultrasonography right eye – funnel retina aspect.

Table 2. The average values of the longitudinal diameter and surface of CC, the frontal-occipital diameter and the brain surface in children within the normal range

No.	Group of age (years)	AB (cm)	EZ (cm)	EZ/3 (cm)	EZ/5 (cm)	Ratio EZ/AB	Surface of CC (mm ²)	Brain surface (mm ²)	Ratio S-CC/S-B
1.	1	14.24	5.91	1.97	1.18	0.42	488.0	16599.1	0.026
2.	1.6	13.90	5.90	1.97	1.18	0.42	555.4	17424.7	0.031
3.	2	14.95	6.13	2.05	1.23	0.41	658.6	18944.1	0.035
4.	3	15.36	6.29	2.10	1.26	0.41	651.3	19219.2	0.034
5.	4	15.54	6.47	2.16	1.29	0.42	723.7	19829.9	0.038
6.	5	15.94	6.74	2.24	1.35	0.42	723.7	19829.9	0.038
7.	6	15.56	6.39	2.13	1.28	0.40	820.6	19626.8	0.042
8.	7	14.92	6.35	2.12	1.27	0.42	889.2	22107.6	0.040
9.	8	16.21	6.79	2.27	1.36	0.41	810.7	21110.8	0.038
10.	9	16.38	7.18	2.40	1.44	0.44	762.7	20286.9	0.042

AB – the frontal-occipital diameter;

EZ – the longitudinal diameter of CC;

EZ/3 – the third part of CC; EZ/5 – the fifth part of CC, the splenium;

EZ/AB – the ration between the longitudinal diameter of CC and the frontal-occipital diameter;

S-CC/S-B – ratio between the surface of CC and the surface of brain

The frontal-occipital diameter in MDVI children is in average less (14.22 ± 0.70 cm versus 15.70 ± 0.51 cm) compared with the value in children within the normal range [$t(22) = -4.144$, $p = 0.000$] (Table 3).

Decreasing of frontal-occipital diameter (8.59%) in MDVI children compared with normal range value was registered. The longitudinal diameter of CC in children with brain damage is more likely inferior in average (5.38 ± 0.59 cm versus 6.52 ± 0.31 cm) with 15.15% compared with the normal value in the control group [$t(22) = -4.858$, $p = 0.000$].

Table 3. Results of the morphometry of CC, brain and the ratio between the CC and brain in children with dysgenesis of CC

Patient	AB (cm)	BZ (cm)	EZ (cm)	EZ/3 (cm)	EZ/5 (cm)	Thinning of CC (cm)	Ratio EZ/AB	Surface of CC (mm ²)	Brain surface (mm ²)	Ratio S-B/S-CC
1.	14.44	4.67	5.37	1.79	1.07	0.4	0.37	264.4	17860.6	0.015
2.	13.59	4.51	5.00	1.67	1.00	0.4	0.36	174.5	18567.9	0.009
3.	13.87	4.64	5.64	1.89	1.14	0.3	0.40	392.3	16543.3	0.024
4.	12.92	4.23	4.69	1.57	0.94	0.3	0.36	213.6	14104.3	0.015
5.	13.99	4.87	5.70	1.90	1.14	0.6	0.40	520.3	20715.6	0.025
6.	14.47	5.97	4.91	1.64	0.98	0.4	0.33	389.3	17469.0	0.022
7.	13.80	5.00	5.10	1.70	1.02	0.5	0.36	450.6	16668.7	0.027
8.	16.42	5.81	6.63	2.21	1.33	0.6	0.40	638.3	20859.3	0.030
9.	14.60	5.12	5.50	1.84	1.10	0.3	0.35	545.2	21818.1	0.025
10.	15.50	5.10	6.50	2.17	1.30	0.3	0.41	502.6	18995.4	0.026
11.	14.51	4.59	6.09	2.03	1.22	0.3	0.41	625.9	21003.4	0.026
12.	13.68	4.90	4.63	1.54	0.93	0.5	0.34	355.9	16011.2	0.022

The surface of CC in disabled children is also less in average with 40.52% (403.15 ± 143.41 mm² versus 718.86 ± 70.79 mm²) compared with value in the group of children without brain damage [$t(22) = -6.254$, $p = 0.000$].

A insignificant difference between the brain surface in disabled children and control group was found [$t(22) = -1.985$, $p = 0.068$]. Thus, the brain surface average in disabled children is nearby the average in children within the control group (18159.77 ± 2352.29 mm² versus 19892.52 ± 777.58 mm²).

Discussion

Visual functions in most MDVI children with cerebral visual impairment (CVI), in particularly visual acuity (VA), is measurable using large, black and white gratings (stripes) presented using preferential looking tests [8,19,10] or using cortical visually evoked potentials [11,12].

In the study of Gronqvist et al. [13] 31% out of 30 evaluated children have abnormal radiological findings and 40% have ocular disorders. He mentioned in his study that many of children with abnormal cerebral morphology had additional neurological symptoms, such as epilepsy, cerebral palsy, attention deficit disorder, and ataxia which reduce the possibility of assessment [14,15]. This study revealed 26% of the patients with ocular disorders associated with abnormal neuroradiological findings. This reflects the high association between ocular and cerebral abnormalities.

In the study of Waugh et al. [16], 51% of the children with congenital disorders on peripheral visual system, i.e. disorders of the anterogeniculate visual pathways and the globe, had brain anomalies confirmed by neuroradiology. In the study of Gronqvist, eight (26%) of the 30 children with ocular disorders had brain anomalies confirmed by neuroradiology. This is a smaller proportion that reported by Waugh et al., however, when the eight children with ocular disorders and impaired mental development (a sign of brain dysfunction despite normal neuroradiology) are included, the total proportions become comparable. It should be stressed that ten of the children with ocular disorders in his study had not been examined by neuroradiology.

In the present research, more than 50% of children with abnormal radiological findings have associated morphological and functional ocular disorders. Eighteen percent of children in the study demonstrated to have cerebral palsy, which is responsible in proportion of 60% of the cerebral visual impairment, according with the previous researches [17-20]. They have associated problems, such as visual functioning problems, oculomotor dysfunctions, language and cognitive retardation, fine and gross motor deficiencies due to the brain injuries, responsible of the transmission and integration of information perceived by different sensorial modalities. Those neurological disorders and brain damages are responsible of ocular motility and visual functions difficulties.

As consequence of neurological disorders, the *psychological development* of each child is very poor for more than 80% (10 children). The psychological age is delayed in MDVI children with severe neurological disorders (around the age of 6 month). According with the developmental stages of Piaget they are within the sensorial-motor stage. The psychological age of the other two children is within the normal range. The results are in concordance with the study of Gronqvist et al. [13] that mentioned 66% of 45 children with cerebral functional abnormalities such as impaired mental development and/or neurological symptoms. [21,22,23,24].

These aspects impaired the children's abilities to explore, to perceive the environment and to capture proper information, which is the base for the further process of transmission and interpretation on the superior level. The exploring capacity, perception, visual attention, representation and memory are psychological aspects, which contribute to the integration of visual information processes, are severe damaged in case of all eight MDVI children [13]. This aspect is also linked with the component of voluntary attention which is deficient [26]. The only component of attention which remains intact is the involuntary attention which is an unconditioning orientation reflex, specific for the first weeks of life till 3-4 years old. The responsible brain regions on attention are the brain stem and the central gray nuclei (the thalamus), morphological structures which are damaged in case of some MDVI children.

Severe oculomotor problems, visual acuity, contrast sensitivity and cognitive visual problems were revealed in MDVI children. The more the brain lesions and both, the cortical and subcortical areas are involved the more the visual functions are affected. This is well explained by the difference in VA and visual behaviours in children with severe disability compared with children with mild disability in our study.

According with the visual functions results, all ten children with severe disorders have difficulties to fixate objects and images for a long period of time, due in majority of case to the subcortical injuries or atrophy. These motility problems could be explained by lesions demonstrated on MRI that proved structural damages on cortical and/or subcortical atrophy. The disruption of normal fixation was associated with nystagmus and saccadic eye movements in five cases (35%)

[25-27]. The visual pursuit is delayed according with the children's chronological age, having difficulties to be oriented, to fixate, to gaze their vision to objects situated in the periphery of vision and difficulty of spontaneity ocular movement [25-27].

Ten severe MDVI children have disengaged attention difficulties to move their eyes and implicit their attention from one target to another situated at the periphery of visual field. This aspect is probably due in part to the problems on the *superior colliculus*, which are responsible to organize the eye movements toward the peripheral targets and to the capacity of concentration [25-27].

According with WHO classification, visual impairment was defined as an optotype acuity ≤ 0.3 with the best correction. In our study the VA value is ≤ 0.3 for ten children (71%) and > 0.3 for the other two children (between 0.5 and 0.7). This aspect is responsible on reducing or obstructing the capacity to explore / to scan the visual stimuli elements, the capacity to separate the image from it background, to differentiate and identify the shape, to extract the elements with maximum information value and to compare them with other elements which already existing in the memory and try to make the categorical identification.

The contrast sensitivity is poor for all 12 children, but more evident in children with severe disability in the limit mostly at 5% at 20-50 cm [28].

The children have difficulties to recognize and interpret human faces, characteristics, mimics and other elements, to recognize objects or images, depict characteristics and detailed elements and integrated them at high level in order to identify the objects or images [29,30]. Those aspects could be explained by brain gliosis and cortical displasy or cortical-subcortical atrophy in the temporal lobe responsible by the recognition of human faces and facial expressions.

The MRI examinations revealed different brain damages of CC, ventricular system, cortical-subcortical atrophy or gliosis of white matter and dysgenesis of cerebellar vermis and brain stem. All these brain lesions could explain the visual behavior and dysfunction of visual information transfer, integration and interpretation. [31]

Values framed between 175.5 mm² and 638.3 mm² was revealed by the CC morphometry in MDVI children. The CC surface area in children with brain damage is with 11.80% till 73.21% less compared with the value in children within the control group. The most reduced surface of CC was observed in children with cortical-subcortical atrophy and modification of ventricular system. Thus, the CC dysgenesis could be one morphological and functional area responsible of visual-motor information transfer, visual perception and recognition of objects and faces dysfunctions in case of MDVI children. The splenium subregion, with a smaller surface and a reduced thinning in case of ten out of twelve children, could be associated with partial lack of inter-hemispheric transfer at the occipital level and interemispheric integration of sensory and motor information. This could have a possible consequence on *decreasing the amount of visual informational transfer within the posterior part of the callosum*. In this respect, ten out of 12 children with severe and mild disabilities and dysgenesis of CC have limited cognitive abilities related to superior functional vision [32,33].

Conclusions

The results of complete assessment through psychological and medical approaching, clinical and paraclinical followed by morphometrical evaluation, revealed the visual and neurocognitive profile of visually impaired multiple disabled children (MDVI).

These methods could provide important information not only about the maturation of visual function, but also on the brain development both morphological and functional. This process could be a very good possibility to find out appropriated rehabilitation programmes in the benefit of MDVI children.

Acknowledgements

The authors thank to the Logistic MRI department of CHU Angers, France for the collaboration, delivering and providing the MRI-results; to the Hiperdia SA Clinic and to the

District Clinical Hospital, Imagistic Department in Cluj-Napoca, Romania for making and providing the MRI examination results of MDVI children; to Mrs. Laura Marginean from the “Aviator Badescu” Centre from Cluj-Napoca, Romania for providing the neurological diagnosis; to Mr. Ciprian Rociu from the Neurological Centre “EpiCenter”, Cluj-Napoca, Romania for making and providing the visual evoked potential results.

The study was possible to the grant “PN II Idei”, *number of project 437/2007*, supported by the Ministry of Education and Research in Romania and to the Socrates – Erasmus fellowship, contract no. 2 from 04 September 2009.

References

1. He Q, Duan Y, Miles J, Takahashi N. A context-sensitive active contour for 2D corpus callosum segmentation. *Int J Biomed Imaging*. 2007; <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2246007/>.
2. Anagnostopoulou S, Mourgela S, Katritsis D. Morphometry of corpus callosum: an anatomical study. *Neuroanatomy* 2008;5:20-23.
3. Parazzini C, Baldoni C, Scotti G, Triulzi F. Terminal zones of myelination/ MR evaluation of children aged 20-40 months. *Am J Neuroradiol* 2002;23:1669-1974.
4. Bennett GL, Bromley B, Benacerraf BR. Agenesis of the corpus callosum: Prenatal detection usually is not possible before 22 weeks of gestations. *J Radiology* 1996;199:447-450.
5. Paus T, Zijdenbos A, Worsley K, Collins DL, Blumenthal J, Giedd JN et al. Structural maturation of neural pathways in children and adolescents: in vivo study. *Science* 1999;283(5409):1908-1911.
6. Anderson S, Boigon S, Davis K. The Oregon Project for visually impaired and blind preschool children – Fifth Edition. Jackson County Education Service District, United States of America. 1991.
7. Plessen KJ, Gruner R, Lundervolt A, Hirsch JG, XU D, Bansai R et al. Reduced white matter connectivity in the corpus callosum of children with Tourette syndrome. *J Child Psychol Psychiatry* 2006;47(10):1013-1022.
8. Birch EE, Bane MC. Forced-choice preferential looking acuity of children with cortical visual impairment. *Dev Med Child Neurol* 1991;33(8):722-729.
9. Hertz BG, Rosenberg J, Sjo O, Warburg M. Acuity card testing of patients with cerebral visual impairment. *Dev Med Child Neurol* 1988;30(5):632-763.
10. Weiss AH, Kelly JP, Phillips JO. The infant who is visually unresponsive on a cortical basis. *Ophthalmology* 2001;108(11):2076-87.
11. Frank Y, Kurtzberg D, Kreuzer JA, Vaughan HG, Jr. Flash and pattern-reversal visual evoked potential abnormalities in infants and children with cerebral blindness. *Dev Med Child Neurol* 1992;34(4):305-15.
12. Good WV. Development of a quantitative method to measure vision in children with chronic cortical visual impairment. *Trans Am Ophthalmol Soc* 2001;99:253-69.
13. Gronqvist S, Flodmark O, Tornqvist K et al. Association between visual impairment and functional and morphological cerebral abnormalities in full-term children. *Acta Ophthalmol Scand* 2001;79:140-146.
14. Blohme´ J. On Visual Impairment In Swedish Children. Thesis, Faculty of Medicine, Lund University, 2000.
15. Rosenberg T, Flage T, Hansen E et al. Incidence of registered visual impairment in the Nordic child population. *Br J Ophthalmol* 1996;80: 49–53.
16. Waugh M, Chong WK, Sonksen P. Neuroimaging in children with congenital disorders of the peripheral visual system. *Dev Med Child Neurol* 1998;40: 812–819.
17. WIKIPEDIA, Free Encyclopedia - Cerebral Palsy, http://en.wikipedia.org/wiki/Cerebral_palsy
18. Luisa Mayer, PhD, Doctors Information Resource CVI. http://www.e-advisor.us/HighContrast/doctors_cvi.htm
19. Good W. Development of a quantitative methods to measure vision in children with chronic cortical visual impairment. *Tr Am Ophth Soc* 2001;99:253-269.

20. Demachak MA, Rickard Ch, Martz Elquist. Fact Sheet – Cortical Visual Impairment, Nevada USA, July 2002.
21. Cziker R, Seceleanu A, Guttman T, Joanta A. Relatia dintre vederea functionala, dezvoltarea neuropsihologica, rezonanta magnetica nucleara si potentialul vizual evocat in deficienta vizuala cerebrala. *Revista Oftalmologia* 2009;4:67-76.
22. Cziker R, Codreanu C. Deficienta vizuala cerebrala in interventia timpurie. Studiu de caz. Simpozion aniversar 50 de ani de la infiintarea Liceului „Transparenta si comunicare in educatia si integrarea socio-profesionala a persoanelor cu deficiente de vedere”. 16-18 octombrie 2008, Cluj-Napoca, Romania.
23. Seceleanu A, Cziker RE, Joanta AE, Matias M, Guttman T. The impact of cortical visual processing disorders on the developmental level of children aged between 0 and 6 years old. Clinical and experimental study. International Conference on Low Vision. Montreal, Canada, 2008.
24. Cziker R, Seceleanu A, Joanta AE, Guttman T, Tintea I. Complete assessment of a multiple disabled visually impaired four years old child. Case study. International Conference on Low Vision. 2008, Montreal, Canada.
25. Goldberg MC, Maurer D, Lewis TL. Influence of a central stimulus on infants visual field. *Infant Behavior and Development* 1997;20(3):359-370.
26. Danila L, Golu M. *Tratat de neuropsihologie*. Editura Medicala, Bucuresti, 2006, pp. 539-553.
27. Dutton GN, Jacobson LK. Cerebral visual impairment in children. *Semin Neonatol* 2001;6:477-485.
28. Alexander KR, Barnes CS, Fishman GA, Pokorny J, Smith VC. Contrast sensitivity deficits in inferred magnocellular and parvocellular pathways in retinitis pigmentosa. *Association for Research in Vision* 2004;45(12):4510-4519.
29. Barbeau EJ, Taylor MJ, Regis J, Marquis P, Chauval P, Liégeois-Chauvel C. Spatio temporal dynamics of face recognition. *Cerebral Cortex* 2008;18(5):997-1009.
30. Kanwisher N, Moscovitch M. The cognitive neuroscience of face processing: An introduction. *Cognitive Neuropsychology* 2000;17(1/2/3):1-11.
31. Cziker R, Joanta A. Deficientele de procesare a informatiilor vizuale la nivel cortical in cazul copiilor de la 0 la 7 ani – Evaluare complexa. Conferinta Internationala „Traditii, valori si perspective in stiintele educatiei”. Universitatea „Babes-Bolyai”. 2008, Cluj-Napoca, Romania.
32. Palmer SL, Reddick WE, Glass JO, Gajjan A, Goloubeva O, Muller RK. Decline in corpus callosum volume among pediatric patients with medulloblastoma: Longitudinal MR imaging study. *J Neuroradiol* 2002;23:1088-1094.
33. Nosarti C, Rush TM, Woodruff PR., Stewart AL, Rifkin L and Murray RM. Corpus callosum size and very preterm birth: relation to neuropsychological outcomes. *Brain* 2004;127:2080-2089.