Visual Navigation in Adolescents with Early Periventricular Lesions: Knowing Where, but Not Getting There

Visual navigation in familiar and unfamiliar surroundings is an essential ingredient of adaptive daily life behavior. Recent brain imaging work helps to recognize that establishing connectivity between brain regions is of importance for successful navigation. Here, we ask whether the ability to navigate is impaired in adolescents who were born premature and suffer congenital bilateral periventricular brain damage that might affect the pathways interconnecting subcortical structures with cortex. Performance on a set of visual labyrinth tasks was significantly worse in patients with periventricular leukomalacia (PVL) as compared with premature-born controls without lesions and term-born adolescents. The ability for visual navigation inversely relates to the severity of motor disability, leg-dominated bilateral spastic cerebral palsy. This agrees with the view that navigation ability substantially improves with practice and might be compromised in individuals with restrictions in active spatial exploration. Visual navigation is negatively linked to the volumetric extent of lesions over the right parietal and frontal periventricular regions. Whereas impairments of visual processing of point-light biological motion are associated in patients with PVL with bilateral parietal periventricular lesions, navigation ability is specifically linked to the frontal lesions in the right hemisphere. We suggest that more anterior periventricular lesions impair the interrelations between the right hippocampus and cortical areas leading to disintegration of neural networks engaged in visual navigation. For the first time, we show that the severity of right frontal periventricular damage and leg-dominated motor disorders can serve as independent predictors of the visual navigation disability.

Keywords: active locomotion, periventricular lesions, visual spatial navigation, volumetric structural MRI

Introduction

Visual navigation in familiar and unfamiliar surroundings is an essential ingredient of adaptive daily life behavior. Brain imaging in humans along with neuropsychological evidence in lesional patients reveals a specific cortico-subcortical brain network engaged in visual navigation. Positron emission tomography and functional magnetic resonance imaging (fMRI) repeatedly point to the right prefrontal lobe, right hippocampus, right medial temporal lobe, parahippocampal gyri, retrosplenial cortex, medial and lateral superior parietal lobules, and posterior cingulated and intraparietal cortex as parts of this distributed network (Maguire and others 1998; Aquivre and D’Esposito 1999; Grön and others 2000; Shelton and Gabrieli 2002; Hartley and others 2003). In a series of structural volumetric magnetic resonance imaging (MRI) studies, Maguire and others (2000) showed that hippocampal volume positively correlates with the amount of time spent as a taxi driver in London city. They suggested that visual experience in navigation plays a crucial role leading to enlargement of the posterior hippocampus. In healthy adolescents and adults, similar pattern of activation involving the right frontal and right anterior medial temporal lobe was found during visually guided navigation in the virtual reality (Pine and others 2002).

Recent work suggests that connectivity between brain structures engaged in visual navigation, rather than integrity of each structure per se, is of importance for successful way finding (Holscher 2003). Here, we examined visual navigation in adolescents with periventricular leukomalacia (PVL), the dominant form of brain injury in individuals who were born premature (about 30–50% of all premature births). PVL is a result of necrosis of myelinated fibers around the lateral ventricles in the peritrigonal area. This lesion pattern is characterized by gliosis in the white matter (WM) and tissue loss with secondary ventricular dilatation, thereby impinging on the pathways interconnecting subcortical structures with cortex. PVL represents a bilateral lesion pattern of early origin (third trimester of pregnancy) and relatively high homogeneity in terms of timing, pathogenesis, and topography and, therefore, serves a proper model for addressing the issue of how topography and extent of WM lesions of similar timing relate to functional abnormalities (Pavlova and others 2003; Krägeloh-Mann 2004).

Patients suffering bilateral damage to the periventricular regions are often reported by their care providers and clinicians to have specific difficulties in way finding and easily getting lost in unfamiliar surroundings (Jacobson and others 1996, 1998). However, these behavioral deficits and underlying brain functional neuropathology have not been systematically investigated. Current progress in the neonatal intensive care leads to an increase in the survival rate of even very premature infants. Quality of survival, therefore, has become a major concern (Volpe 2001; Peterson and others 2003).

In the present work, within the context of a basic study aimed at assessing perceptual deficiencies in adolescents who were born premature (Pavlova and others 2003, 2005; Pavlova, Marconato, and others 2006; Pavlova, Sokolov, and others 2006), we ask 1) how, if at all, visual navigation is impaired in patients with PVL, 2) whether these deficits are specifically related to the topography and extent of periventricular lesions, and 3) whether the visual navigation ability is related to motor disability. Because up to date it is well established that the ability to navigate substantially improves with practice (Winocur and others 2005), we expect that it might be compromised in individuals with restrictions in active locomotion leading to difficulties in active spatial exploration of the environment.
Methods

Participants

Patients were 14 adolescents (aged 13-16 years) born premature between 27 and 33 weeks of gestation with MRI evidence for PVL, which is a form of a WM injury affecting the regions around the lateral ventricles in the periventricular area (Fig. 1). Two male patients did not come for neuropsychological examination, and 1 female patient with PVL was excluded from the data processing because of cortical lesions revealed on her MRI scan. This left the data sets from 11 PVL patients (mean age 14.5, standard deviation [SD] ±1.29) for the subsequent processing. Eight children who were born premature (mean age 14.5, SD ±1.2) and 8 term-born participants (mean age 14.57, SD ±0.78) had MRI scans without any identifiable signs of brain damage or other abnormalities and served as controls. Participants who were born premature were recruited on a voluntary basis from a data pool of the Department of Paediatric Neuropediatrics and Developmental Care, Children’s Hospital, University of Tübingen. Term-born controls were recruited as volunteers from the local community. All participants had normal or corrected vision. Verbal IQ (Intelligence Quotient) greater than 85 (HAWIK-III 2001; based on the WISC-III, adapted to the German population) was an inclusion criterion for all participants. For patients, verbal IQ scores were in the range from 101 to 127 (average 111.09, SD ±8.561), for premature-born controls in the range from 103 to 127 (average 112.75, SD ±8.972), and for term-born controls in the range from 97 to 144 (average score 117.875, SD ±14.827). Pairwise comparisons revealed no significant differences in the verbal IQ scores between the groups of participants. We also controlled for oculomotor dysfunctions (such as nystagmus) that could be observed in patients with PVL (Cioni and others 1997; Jacobson and Dutton 2000) and may affect performance on visual tasks. All participants underwent neurological examination. With respect to locomotion ability, the patients with PVL ranged from normal function through impairment in walking pattern to complete walking inability. More specifically, in 8 of the 11 patients with PVL, a leg-dominated bilateral spastic cerebral palsy (BS-CP) was diagnosed (for details, see section Motor Disorders Assessment below). All patients attended a mainstream school with the exception of only one male patient who attended a special school for motor-disabled children. Note that this patient’s score on verbal IQ was within the normal range (108).

Informed written consent was obtained from the participants and their care providers in accordance with the requirements of the Ethical Committee of the Faculty of Medicine at the University of Tübingen.

Structural MRI and Quantification of Lesion Extent

MRI scans were obtained as axial dual turbo spin-echo slices (35 axial slices, repetition time 4800 ms, echo time 85 ms, 4 mm slice thickness) through a 1.5-T Siemens Vision scanner (Erlangen, Germany). PVL is characterized by gliosis in the WM and tissue loss with secondary ventricular dilatation (Krägeloh-Mann and others 1999). For quantification of the volumetric extent of PVL, therefore, on each T2-weighted slice, the area of the lateral ventricle and any identifiable gliosis in the WM were manually traced on contiguous axial planes using the MRICro software. The resulting volume was divided into anterior (frontal), inferior (temporal), and posterior (parieto-occipital) sections for each hemisphere. The central sulcus served as a border between the posterior and anterior sections, and the tip of the occipital horn of the lateral ventricle was taken as a border between the superior and inferior section. In order to achieve standard dimensions and orientation, a linear normalization was performed through SPM99 (Statistical Parametric Mapping, Welcome Department of Cognitive Neurology, University College London). The normalized lesion volumes were determined in the MRICro software with 50% threshold for interpolated voxels.

Volume of Gray Matter, White Matter, and Cortico-Spinal Fluid

By using SPM99 algorithms, we automatically assessed the total volumes of gray matter (GM), WM, and cortico-spinal fluid (CSF) for each patient.

Motor Disorders Assessment

All participants underwent a standardized neurological examination. BS-CP was diagnosed if the following signs were present: abnormal pattern of posture or movement (e.g., hip adduction or internal rotation with equines of the feet or its secondary malposition), increased tone, and pathological reflexes (flexor hypertonicity or increased tendon reflexes). Motor disorders of upper and lower extremities were separately assessed and scored on four-point scales (Krägeloh-Mann and others 1993; Pavlova and others 2003; Staudt and others 2003). In brief, for lower limbs, one of the major components of spasticity is restriction of forefoot dorsal extension that leads to a characteristic abnormal walking pattern. The scores for assessment of spastic motor disorders were 1) near-to-normal walking pattern, able to walk on heels; 2) moderately abnormal walking pattern, walking on heels only with intermittent forefoot-ground contact; 3) severely abnormal walking pattern, restricted ability for unaided walk, marked slowing down of locomotion speed, inability to lift forefoot from the ground when trying to walk on heels; and 4) complete inability for unaided walking. For upper limbs, a separate scale was used: 1) sequential finger opposition not markedly impaired, 2) marked slowing down of incomplete sequential finger opposition, 3) inability to move single finger, preserved grasp function, and 4) complete inability to grasp. If one of the upper or lower extremities was more affected than the other, the greatest score was taken. For making the outcome of neurological examination suitable for further data processing, the severity of motor disability was assessed as zero (score 0), if no signs of BS-CP were detected.

In participants with normal MRI scan, neurological examination did not reveal any signs of motor disability. Among 11 patients with bilateral PVL, 3 were free from impairment of either lower or upper extremities (score 0), and in 8 of them, a leg-dominated BS-CP was diagnosed: lower limbs were more affected than upper limbs. Upper limbs were affected

Figure 1. Structural magnetic resonance images (axial T2-weighted, z = 22 mm above the bicommissural plane) (A) for patients with mild (TSA, female, left panel; and RUL, female, middle panel; light arrows point to the gliosis in the WM) and more severe (SMA, male, right panel) PVL and (B) for representative controls DHE (healthy term-born adolescent, male, left panel) and KRO (adolescent who was born premature, male, right panel) without signs of brain abnormalities. TSA, RUL, SMA, DHE, and KRO are codes for patients’ names.
only in 3 patients: in one of them with score 1 and in two with score 2. Lower limbs scores were 2 in two and 3 in four patients. Two participants were completely unable to walk autonomously (score 4). In respect to walking ability, therefore, the patients ranged from normal function over impairment in walking to complete disability for autonomous locomotion.

**Labyrinth Test**

The present study is focused on performance on a Labyrinth (LA) test administered to participants during neuropsychological examination (HAWIK-III 2001) based on the WISC-III (Wechsler Intelligence Scale for Children, third edition) adapted to German population. This test is aimed at assessing the ability for visual spatial navigation, topographical skills, and way finding. Although the LA test represents a simplified model of real-world navigation, it taps the most important aspects of visual navigation and has a number of practical advantages in clinical testing. For example, the test is not time consuming. Being a part of HAWIK-III battery based on the WISC-III, the LA test is psychometrically standardized and provides with normative scores obtained in a large healthy population. The LA test consists of 10 paper-and-pencil LA tasks increasing in their complexity. In each task, a participant has to find the way out from the center of a 2-dimensional maze (Fig. 2). Two examiners, a principal investigator (M.P.) and her assistant, tested participants individually. Participants were told that they have to start traversal the maze without trying to find the way in mind, or in other words, they have to solve the problem by doing. Each participant was required to start immediately once the maze was presented. They were also informed that each LA had a specific time limit for its passing. Both examiners together did control for the following given instruction. When assessing performance on the task, both accuracy (the number of errors such as dead ends, going through the walls) and time needed for passing through a LA (as a specific time limit for each maze ranging according to the LA complexity from 30 to 150 s) are taken into account. For each LA, the number of errors corresponds to specific raw scores given in the HAWIK-III Manual (Tewes and others 2001). For example, for the most complex LA number 10, 0 error yields score 5, and 5 and more errors yield score 0. According to the HAWIK-III Manual, raw values (sum of scores with a maximum 28 resulting from summing up the highest possible scores for each of 10 LAs, namely, \(6 \times 2 + 1 \times 3 + 2 \times 4 + 1 \times 5\)) are then transformed into the standardized normative scores ranging from 1 (floor performance) through 10 (normal performance for this age) to 17 (ceiling performance for this age). Although the patients were almost free from motor disorders of upper limbs (see above), when assessing performance on the task we did not consider the motor component while passing the maze (e.g., touching the LAs walls by a pencil).

**Results**

**Visual Navigation Ability**

On the LA test, the performance level of patients with PVL was significantly lower than in both control groups (mean scores 4.818 [SD 4.167], 9.375 [SD 3.503], 10.125 [SD 3.314], for patients, premature-born, and term-born controls, respectively; \(t_{17} = 3.089, P < 0.007\), pairwise comparison with term-born controls; \(t_{17} = 2.583, P < 0.02\), comparison with premature-born controls). As can be seen in Figure 3, the performance scores of 8 out of 11 patients are below the normal range. Most families did report specific problems in way finding and visual spatial navigation when they were asked about the patients’ difficulties in daily life and in the school. For example, the father of patient SSA, a school principal, told that for many years his daughter has been unable to find the way from one classroom to the other without external support and also has had serious difficulties in way finding in their local place. Note that to the date of examination, verbal IQ of this patient was rather high (113).

There are no significant differences in performance between both control groups, that is, between former preterms without lesions and term-born participants (\(t_{14} = 0.44\), two-tailed, \(P = 0.667\)). This indicates that poorer performance on visual navigation task in patients with PVL is not simply a consequence of brain prematurity.

**Visual Navigation, IQ Factors, and Visual Perceptual Abilities**

No linkage was found between performance of PVL patients on the LA test and the scores on either general (\(r = 0.431\), not significant [NS]) or verbal IQ (\(r = 0.16\), NS). Most important for the purpose of the present work, no substantial link occurs between the visual navigation ability and performance IQ (\(r = 0.415\), NS). Moreover, performance on neither Perceptual Organization (PO) tasks (IQ factor PO based on visual perceptual tasks, namely, picture completion, event arrangement, block design, and object assembly) nor visual attention tasks (IQ factor Processing Speed [PS], based on 2 visual attention tasks, visual search and coding) was substantially related to performance on the LA test (\(r = 0.487\), \(r = 0.44\), NS, for PO and PS factors, respectively; Fig. 3). This underscores the specificity of the impairments of the visual navigation ability as compared with other visual spatial abilities in the sample of PVL patients.
Visual Navigation and PVL Extent

In PVL patients, performance on the LA test correlates negatively with the volumetric extent of PVL over the right parietooccipital complex (Pearson product-moment correlation, \( r = -0.617, P < 0.05 \)). The performance level drops with an increase of the lesion extent (Fig. 4C). For the left hemisphere, this correlation was not significant. No relationship occurs between performance on the LA test and the lesion extent in the temporal region (\( r = -0.111, r = 0.305, \text{NS} \), for the right and left hemisphere, respectively).

Notably, the strongest correlation was found between performance on the LA test and the extent of PVL in the right frontal regions (\( r = -0.641, P < 0.05 \); Fig. 4A), whereas the extent of lesions in the left frontal region did not significantly relate to the visual navigation ability (\( r = -0.517, \text{NS} \)). In both control groups, neither the volumetric ventricular extent over the right frontal regions (\( r = -0.083, r = -0.215, \text{NS} \), for premature- and term-born controls, respectively) nor the right parietooccipital ventricular extent (\( r = -0.503, r = -0.476, \text{NS} \), for premature- and term-born controls, respectively) related substantially to performance on the LA test (Fig. 4B, D).

In earlier work (Pavlova and others 2005), we reported a strong negative link between the extent of bilateral parietooccipital PVL and performance on the task requiring visual processing of point-light biological motion representing human locomotion. We also established the inverse relationship between the extent of bilateral parietooccipital PVL and the scores on IQ factors PO and PS. The negative link to the extent of the right frontal PVL found in the present study is specific for the visual navigation ability, as it did not occur for other visual perceptual tasks (namely, picture completion, event arrangement, block design, object assembly, symbol search, and coding) administered in the course of neuro-psychological examination and for visual processing of point-light biological motion.

Relation of Visual Navigation to the WM, GM, and CSF Volumes

The specificity of the link between the severity of the right frontal and parietooccipital PVL and the navigation ability is also revealed by the fact that there is a lack of relationship between the WM volume, which is often considered a distinct feature of PVL severity, and performance on the LA test (\( r = 0.193, \text{NS} \); Fig. 5). Likewise, the performance level is not related to the GM and CSF volumes (\( r = 0.16, r = -0.269, \text{NS} \), respectively).

Visual Navigation and Locomotion Ability

We tested the hypothesis that if early restrictions in active locomotion were related to deficits in visual navigation, then the performance level on the LA test would decrease with increases in the severity of motor disorders. We were mainly interested in this relationship because of two decisive reasons. First, most PVL patients involved into the study suffer leg-dominated BS-CP. Second, active exploration of environment is essential for development of the navigation ability (Wang and Spelke 2000). As expected, a strong relationship was found between performance on the LA test and the severity of functional motor disorders of lower extremities (\( r = -0.697, P < 0.05 \); Fig. 6). The severity of motor impairment of upper extremities was not substantially related to performance on the LA test.

If performance on the LA test is related to the extent of lesions in the particular brain regions and the severity of motor disability, do these factors affect the visual navigation independently or are they associated to each other? In particular, the extent of more

Figure 4. The scores on the LA task plotted against (A) volumetric extent of PVL (ventricular extent plus gliosis) in the right frontal regions in patients (Pearson product-moment correlation, \( r = -0.641, P < 0.05 \)), (B) volumetric lateral ventricular extent in the right frontal regions in term-born (open diamonds, \( r = -0.215, \text{NS} \)) and preterm-born controls (filled diamonds, \( r = 0.476, \text{NS} \)), (C) volumetric extent of PVL in the right parietooccipital regions in patients (\( r = -0.617, P < 0.05 \)), and (D) volumetric lateral ventricular extent in the right parietooccipital regions in term-born (open diamonds, \( r = -0.476, \text{NS} \)) and preterm-born controls (filled diamonds, \( r = 0.503, \text{NS} \)).
PVL along with the severity of CP with performance on the LA test. Combined correlation of the frontal NS, for the right hemispheric frontal lesions that highly correlate with the lateral extent of lesions affecting the pyramidal tract, that is, the cortico-spinal projections to motor areas (Staudt and others 2003). The volumetric extent of PVL over the frontal regions is not substantially related to the severity of motor disorders ($r = 0.365$, NS, for both hemispheres together; $r = 0.223$, NS, for the right hemispheric frontal lesions that highly correlate to the navigation ability). Combined correlation of the frontal PVL along with the severity of CP with performance on the LA task is higher (-0.774) than correlation of each of these two factors alone with performance on the LA test. We conclude therefore that in PVL patients, the extent of the right frontal lesions and the severity of leg-dominated CP are likely to affect visual navigation independently.

We further submitted the data to a stepwise multiple regression analysis, with the dependent variable navigation ability and 3 independent variables, namely, motor disability, extent of right parietooccipital PVL, and extent of right frontal PVL. The motor disability was entered first and explained a significant percentage of the variance (49%) in the navigation ability ($F_{1,9} = 8.491, P < 0.017$). The extent of right frontal PVL was entered second and explained a further 25% of variance ($F_{1,9} = 7.409, P < 0.026$). The third variable (extent of right parietooccipital PVL) did not explain any additional significant proportion of the criterion variance and, therefore, could not be considered as an independent predictor of the navigation ability. We suspect, therefore, that the right frontal and parietooccipital PVL affect the same neural network subserving visual navigation.

Discussion

Combining volumetric analysis of the structural MRI with neuropsychological investigation, we addressed the issue of whether and, if so, how visual navigation is impaired in adolescents who were born premature and suffer periventricular brain damage. The main outcome of the work indicates that the visual navigation ability is profoundly compromised in patients with PVL even if lesions are relatively small in size and the verbal IQ is within normal range. Moreover, the severity of this impairment is specifically related to the topography and extent of PVL: performance on the navigation tasks is inversely related to the volumetric extent of damage to the frontal and parietooccipital regions in the right hemisphere solely. The lack of differences in performance on the navigation tasks between former preterms and healthy term-born controls with normal MRI scan indicates that the factor of brain prematurity alone is irrelevant for normal development of the visual navigation ability.

Damage to periventricular regions that contain many interconnecting fibers projecting to the cortex might interrupt functioning of the distributed cortical-subcortical network subserving visual navigation and way finding. Periventricular lesions might break the reciprocal thalamocortical interrelations impinging on posterior thalamocortical fibers (Krägeloh-Mann and others 1999). Recent diffusion tensor imaging (DTI) findings suggest that PVL affects the posterior thalamic radiation (Hoon and others 2002), which connects the pulvinar and the lateral geniculate nucleus to the parietal cortex (Behrens and others 2003). Interruptions of this connection may affect performance on a number of visual perceptual and attentional tasks. In earlier work (Pavlova and others 2005; Pavlova, Sokolov, and others 2006), we showed that deficiencies in visual processing of point-light displays representing human locomotion are associated with the severity of parietal periventricular lesions in both hemispheres. We also established the inverse relationship between the extent of bilateral parietooccipital PVL and the scores on the factors PO and PS constituting performance IQ (Pavlova and others 2005). Here, we found that the navigation ability is specifically linked to right hemispheric damage to parietal and, in particular, frontal periventricular regions. Bearing in mind that the brain network subserving visual navigation involves the right prefrontal lobe and the right hippocampus (Maguire and others 1998; Grön and others 2000; Spiers and others 2001), we suggest that more anterior lesions lead to disintegration in the functioning of this network breaking the interrelations between the right hippocampus and the frontal cortex. Moreover, as indicated by the outcome of a stepwise multiple regression analysis, the extent of right frontal lesions may be considered as an independent predictor of the impaired navigation ability.

The findings provide substantial evidence in favor of, and further elaborate, the notion that it is brain connectivity rather than integrity of a single brain structure that constitutes an essential requirement for successful visual navigation (e.g., Aguirre and D’Esposito 1999; Holscher 2003). This view also corresponds to the findings showing that oscillatory theta brain...
activity that might be considered a putative indicator for interconnections between neural networks exhibits specific enhancements during visual navigation tasks (Kahana and others 2001; Raghavachari and others 2001). Notably, theta oscillations occur more frequently in more complex mazes (Kahana and others 1999), visual navigation in which might involve more complex neural networks. For further elaboration of the intriguing brain connectivity hypothesis, we need to combine functional brain imaging, namely, magnetoencephalography (MEG) providing with high time resolution and fMRI providing with high spatial resolution, with DTI.

Additional possibility that should be considered for future research is that PVL can be accompanied by neuronal impairments (Marin-Padilla 1997). Although necocortical and other neuronal structures do not display noticeable pathological abnormalities in PVL, strong cytokine immunoreactivity has been recently detected in the hippocampus, basal ganglia, and thalamus (Kadhim and others 2003).

The other essential finding of the present study is that the visual navigation ability is inversely related to the severity of motor disorders, leg-dominated BS-CP. Previously, we reported that visual processing of point-light displays representing human locomotion did not depend on the ability to produce biological movement challenging the idea that motor experience is an obligatory prerequisite for the perception of human locomotion (Pavlova and others 2003). Motor experience does not appear to be necessary for visual analysis of human movement presumably because a hard-wired schema for biological motion processing is inherent for the human brain.

The present data highlight the role of active spatial exploration for a proper development of visual navigation. Active exploration in patients with leg-dominated BS-CP has been impaired from the very beginning in life, and, therefore, may prevent normal development of the visual navigation ability. This outcome agrees well with the findings demonstrating that the ability to navigate substantially improves with practice (Maguire and others 2000; Winocur and others 2005) and might be compromised in individuals with restrictions in active spatial exploration. Like insects and rodents, humans continuously update their spatial representation of the environment as they move (Wang and Spelke 2000).

One may assume that the link between motor disability and performance on the LA task might be explained by an inability of PVL patients to simulate movement. It is unclear, however, whether simulation of locomotion constitutes an essential requirement for successful performance on this task. It is also unknown whether simulation of movement is impaired in patients with BS-CP. For example, it was shown that as in healthy controls, the perceptual judgments in patients with congenitally absent limbs are restricted by biomechanical constraints (Brugger and others 2000). However, this occurs only when aplasic patients have phantom limb sensations (Funk and others 2005). The present findings show that impairments in visual navigation are closely related to disorders in locomotion ability. It is important to stress that we did find the link between the leg-dominated motor disorders and the impairments in visual navigation, whereas motor component of the LA task was confined solely to the movements of upper extremities. No substantial link occurred between motor impairments of upper extremities and performance on the LA task.

In patients with meningomyelocele and hydrocephalus, a tight relationship occurs between locomotion ability and the scores on performance IQ that is based mainly on visual perceptual tasks (Rendeli and others 2003). By contrast, in the present study, the lack of link between the severity of leg-dominated BS-CP and performance IQ in patients with PVL points to the specificity of connection between active spatial exploration and the visual navigation ability, at least, in this sample of patients. Moreover, as revealed by the stepwise multiple regression analysis, the severity of locomotion disability may be considered a strong predictor of deficiencies in visual navigation ability.

Taken together, the present findings for the first time show that the ability for visual navigation inversely relates to the severity of motor disability, leg-dominated BS-CP, and, thereby, further illuminate the role of active spatial exploration in a proper development of visual navigation. This opens a window for early training in active spatial exploration and way finding in familiar and unfamiliar surroundings in patients with motor disabilities. Congenital periventricular brain damage over the parietal and, in particular, frontal regions in the right hemisphere is associated with long-lasting impairments in visual spatial navigation in adolescence presumably causing disintegration of the neural network engaged in successful visual navigation. A further step toward uncovering the neuropathology of visual navigation would be an analysis of functional brain connectivity by combining fMRI and MEG with DTI.

Notes

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