

Impaired math achievement in patients with acute vestibular neuritis

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Abstract

Broad cognitive difficulties have been reported in patients with peripheral vestibular deficit, especially in the domain of spatial cognition. Processing and manipulating numbers relies on the ability to use the inherent spatial features of numbers. It is thus conceivable that patients with acute peripheral vestibular deficit show impaired numerical cognition. Using the number Stroop task and a short math achievement test, we tested 20 patients with acute vestibular neuritis and 20 healthy, age-matched controls. On the one hand, patients showed normal congruency and distance effects in the number Stroop task, which is indicative of normal number magnitude processing. On the other hand, patients scored lower than healthy controls in the math achievement test. We provide evidence that the lower performance cannot be explained by either differences in prior math knowledge (i.e., education) or slower processing speed. Our results suggest that peripheral vestibular deficit negatively affects numerical cognition in terms of the efficient manipulation of numbers. We discuss the role of executive functions in math performance and argue that previously reported executive deficits in patients with peripheral vestibular deficit provide a plausible explanation for the lower math achievement scores. In light of the handicapping effects of impaired numerical cognition in daily living, it is crucial to further investigate the mechanisms that cause mathematical deficits in acute PVD and eventually develop adequate means for cognitive interventions.

Keywords: vestibular deficit, numerical cognition, math achievement, dyscalculia, executive functions

Introduction

A peripheral vestibular deficit (PVD) leads to severe vertigo, nausea, vomiting, and imbalance (Brandt, 2003). Interestingly, in addition to the typical symptoms of PVD, patients frequently report having cognitive difficulties in daily life (Bigelow, Semenov, Trevino, et al., 2015; Black, Pesznecker, & Stallings, 2004; Harun, Semenov, & Agrawal, 2015). These complaints have received empirical support in a growing body of evidence with regard to many cognitive domains including attention, memory, executive function, and spatial cognition (see Bigelow & Agrawal, 2015; Hanes & McCollum, 2006; Mast, Preuss, Hartmann, & Grabherr, 2014). It is intriguing that cognitive deficits have not only been found in dynamic situations (i.e., dual-tasks that combine cognitive tasks with postural challenges) but are also evident in static situations with no concomitant head or body movement (Redfern, Talkowski, Jennings, & Furman, 2004; Talkowski, Redfern, Jennings, & Furman, 2005; Yardley et al., 2001).

Since the vestibular system is crucial for the cognitive representation of space (Angelaki & Cullen, 2008), deficits have been extensively studied in the area of visuo-spatial abilities (i.e., spatial cognition). It has been shown that bilateral vestibular failure is associated with hippocampal loss and impaired spatial navigation (Brandt et al., 2005; Kremmyda et al., 2016; Russell, Horii, Smith, Darlington, & Bilkey, 2003; Schautzer, Hamilton, Kalla, Strupp, & Brandt, 2003; Stackman & Herbert, 2002). Similarly, patients with unilateral PVD also perform worse than healthy controls in navigation tasks (Borel et al., 2004; Cohen, 2000; Guidetti, Monzani, Trebbi, & Rovatti, 2008; Péruch et al., 1999; Péruch, Borel, Magnan, & Lacour, 2005 but see Hufner et al., 2007). Patients with PVD are also impaired in tasks that involve the mental rotation of one's own body without actual displacement (i.e, mental imagery; Candidi et al., 2013; Grabherr, Cuffel, Guyot, & Mast, 2011; Péruch et al., 2011; but see Deroualle et al.,

2017). Taken together, there is strong evidence that PVD leads to an impaired internal representation of space (Borel, Lopez, Péruch, & Lacour, 2008).

The notion of impaired cognitive representation of space might not only explain the frequently found deficits in navigation and spatial memory following PVD. It may also play a crucial role in other cognitive functions that rely on rather abstract representations of magnitude and space. For example, the spatial organization of numbers has coined the term “mental number line” (Dehaene, Bossini, & Giraux, 1993). These number-space associations are consistently found across various experimental paradigms. For example, in parity (odd vs. even) judgment tasks, participants typically respond faster to large numbers with the right hand, while responses to small numbers are faster with the left hand (SNARC effect; Nuerk, Wood, & Willmes, 2005). Other intriguing findings have been reported by means of random number generation tasks. Passive or active body-motion towards the left lead to the generation of smaller numbers compared to head motion towards the right (Hartmann, Grabherr, & Mast, 2012; Loetscher, Schwarz, Schubiger, & Brugger, 2008; Shaki & Fischer, 2014).

Following this idea of number-space associations, Smith (2012) stated the hypothesis that there might be a link between PVD and dyscalculia. According to common definitions, individuals with dyscalculia perform poorly in mathematical achievement tests while showing normal intelligence (Butterworth, 2005; Butterworth, Varma, & Laurillard, 2011; Cohen Kadosh & Walsh, 2007; von Aster & Shalev, 2007). At a lower cognitive level, the syndrome is characterized by a single core deficit in processing number magnitude (i.e., numerosity), which correlates with functional and anatomical abnormalities in parietal areas (Isaacs, Edmonds, Lucas, & Gadian, 2001; Kucian et al., 2006; Mussolin et al., 2010; Price, Holloway, Rasanen, Vesterinen, & Ansari, 2007). Interestingly, there is first evidence in support of impaired

processing and manipulation of numbers in vestibular disorders. Risey and Briner (1990) found that vestibular patients make more mistakes counting backwards, and score lower on the arithmetic subtests and the backward digit span task of the Wechsler Adult Intelligence Scale (WAIS). However, it has to be pointed out that the patients who took part in their study suffered from vertigo due to central origin. Nevertheless, deficits have also been found in patients with PVD in a double-task that required the participants to count backwards during a continuous body orientation task on an oscillatory chair (Yardley et al., 2002).

To date, despite ample anecdotal evidence of impaired numerical abilities in clinical practice, empirical evidence is still rather scarce. For example, it is yet unclear whether numerical deficits also appear under static conditions when no physical movements could interfere with cognitive processes. Furthermore, more research is needed to better specify which aspects of numerical cognition are impaired in patients with PVD. Poor mathematical skills can substantially impair performance at the workplace (Parsons & Bynner, 2005) and in daily life. Indeed, a recent report found that vestibular dysfunction was more strongly associated with difficulty managing finances than with motor-based activities of daily living (Harun et al., 2015).

In order to test the hypothesis of dyscalculia in patients with PVD, we set out to examine two key aspects that are implicated in dyscalculia. First, on a high level of numerical cognition, we wanted to investigate whether patients with PVD and healthy controls differ with respect to the efficient manipulation of numbers in complex tasks (i.e., math achievement). We expected that healthy controls outperform patients with PVD in a short math achievement test. Math achievement relies on a set of domain-general skills such as working memory or executive functions (De Rammelaere, Stuyven, & Vandierendonck, 1999; DeStefano & LeFevre, 2004; Passolunghi & Pazzaglia, 2004; van der Sluis, de Jong, & van der Leij, 2007; von Aster &

Shalev, 2007), which were repeatedly found to be impaired in vestibular dysfunction (Bigelow, Semenov, du Lac, Hoffman, & Agrawal, 2015; Black et al., 2004; Grimm, Hemenway, Lebray, & Black, 1989; Hanes & McCollum, 2006; Moser, Vibert, Caversaccio, & Mast, 2016).

Second, we were also interested whether PVD results in basic problems in processing the magnitude of numbers. Impaired performance in a number processing task would support the hypothesis of Smith (2012) since it reflects a key aspect of dyscalculia. For this reason, we used the number Stroop task, which typically produces abnormal results in children and adults with impaired numerical cognition (e.g., Algom, Dekel, & Pansky, 1996; Ashkenazi, Rubinsten, & Henik, 2009; Girelli, Lucangeli, & Butterworth, 2000; Rubinsten & Henik, 2005; Rubinsten, Henik, Berger, & Shahar-Shalev, 2002). In its original form, the number Stroop task consists of two subtests, subsequently referred to as the *physical* and *numerical* number Stroop. Both subtests require the participants to compare two simultaneously presented digits. In the *physical* number Stroop, the participants are instructed to indicate which digit is physically larger (i.e., has the larger font size). In contrast, the *numerical* number Stroop requires a response to the digit that has the higher numerical value. The trials differ with respect to three congruity conditions. (1) *Congruent* trials consist of two digits, where one digit is larger with respect to both dimensions (i.e., physical size and numerical value). (2) In *incongruent* trials, the digit with the smaller numerical value is displayed with larger font size or vice versa. (3) In *neutral* trials, two identical digits are presented with different font size (*physical* subtest) or two different digits are presented with the same font size (*numerical* subtest).

First, we were motivated to investigate the size congruity effect (SCE), which refers to the difference in response times between *congruent*, *incongruent*, and *neutral* trials in the *physical* number Stroop. The SCE can be divided into a facilitation and interference component.

Facilitation implies faster response times in *congruent* compared to *neutral* trials. Interference implies slower response times in *incongruent* compared to *neutral* trials. The SCE is considered to be a measure of automatic processing of the task-irrelevant number magnitude (Bugden & Ansari, 2011; Girelli et al., 2000; Rubinsten et al., 2002). Second, the number Stroop task allowed us to compare the distance effect, which is characterized by faster responses with increasing numerical distance between two simultaneously presented digits. For example, comparing two digits separated by a distance of 1 (e.g., “6” and “7”) leads to larger response time than comparing two digits separated by a distance of 5 (e.g., “2” and “7”). The distance effect serves as a marker of intentional number processing in the *numerical* number Stroop (Rubinsten et al., 2002). In contrast, a reversed distance effect (faster responses with decreasing numerical distance) has previously been observed in the *physical* number Stroop and has been interpreted as automatic number processing (Heine et al., 2010; Pina, Castillo, Kadosh, & Fuentes, 2015; Tang, Critchley, Glaser, Dolan, & Butterworth, 2006).

If patients with PVD suffer from a severe impairment of number magnitude processing similar to individuals with impaired numerical cognition (i.e., dyscalculia), we might observe a weaker SCE in the *physical* number Stroop (Ashkenazi, Henik, Ifergane, & Shelef, 2008; Ashkenazi et al., 2009; Girelli et al., 2000; Kadosh et al., 2007; Rubinsten & Henik, 2005, 2009). Furthermore, we might expect a stronger distance effect in the *numerical* number Stroop, which is another typical finding in individuals with weak numerical cognition (Bugden & Ansari, 2011; De Smedt et al., 2009; Heine et al., 2010; Holloway & Ansari, 2009; Mussolin et al., 2010; Pina et al., 2015; Rubinsten et al., 2002; Tang et al., 2006). However, as mentioned above, the term “dyscalculia” usually refers to a learning disability, which is characterized by a specific deficiency in core numerical abilities with intact function in other cognitive domains

(Butterworth, 2005; Butterworth, Varma, & Laurillard, 2011; Cohen Kadosh & Walsh, 2007). Since cognitive deficits in PVD were frequently observed in non-numerical tasks (Bigelow & Agrawal, 2015; Hanes & McCollum, 2006; Mast et al., 2014; Smith, Zheng, Horii, & Darlington, 2005), it is unclear whether numerical deficits in PVD follow a dyscalculic pattern of number Stroop performance (i.e., weaker SCE and/or stronger distance effect).

Methods

Participants

We tested 20 participants (9 female) with acute vestibular neuritis attributed to reactivation of neurotropic viruses in the vestibular ganglion, generating an inflammation of the vestibular nerve. Diagnosis of vestibular neuritis was based on extensive neurotological examination including electronystagmography with bithermal caloric testing, pendular rotatory chair testing, cervical vestibular evoked myogenic potentials (cVEMPs), video head impulse test (V-HIT), and dynamic visual acuity test (DVA). Additionally, all patients rated subjective handicap of the vestibular deficit by means of the dizziness handicap inventory (DHI; Jacobson and Newman 1990). Testing was performed during a routine follow-up shortly after initial admission to the emergency ENT-unit of the University Hospital Bern (mean interval = 11.10 days + 5.48 SD). We included patients according to the following criteria: (1) Canal paresis ≥ 20 % in bithermal caloric testing, (2) no hearing loss, (3) no vertigo due to a central lesion, (4) no history of previous neurotologic diseases. Additionally, we included 21 healthy participants (10 female) in our study. Patients with AVN (mean age = 47.11, SD = 13.55) and healthy controls (mean age = 46.19, SD = 13.75) did not differ with respect to age or gender. In order to control for the level of formal education, all participants indicated their highest academic qualification.

We categorized the level of education according to the typical academic qualifications obtained in the Swiss educational system: compulsory education, vocational training, university entrance diploma, higher vocational training, college of higher education, and university degree. More detailed demographic and clinical data of the participants is provided in Table 1. Before starting data collection, the study was approved by the ethics committee of the Canton Bern. The experimental procedure adhered to the ethical standards defined by the Declaration of Helsinki. All participants gave informed consent prior to the experiment. Participants did not receive money or other compensation for participation.

----- Insert Table 1 about here -----

Stimuli and Procedure

In the number Stroop task, participants were instructed to decide as quickly as possible which number was presented “physically larger” (*physical* subtest) or “numerically larger” (*numerical* subtest). Responses were collected using a custom-made response box. Participants were asked to indicate the side (left vs. right) of the larger stimulus by pressing a key on the corresponding side of the response box. Each subtest consisted of 96 trials, which included 24 congruent, 24 incongruent, and 48 neutral trials. Ten randomly generated additional trials at the beginning of each subtest served as practice trials and were excluded from data analysis. The order of the two subtests was counterbalanced across participants. We used the digits 1 through 9, without 5. The pairs of digits were presented with numerical distances of zero (1-1; 2-2; 3-3; 4-4; 6-6; 7-7; 8-8), one (1-2; 3-4; 6-7; 8-9), two (1-3; 2-4; 6-8; 7-9), and five (1-6; 2-7; 4-9). Each size and digit was presented equally often at each side of the screen. Stimulus presentation

was generated with PsychoPy (Peirce, 2007) on a 15'' laptop monitor at a resolution of 1600 x 900 pixels. Viewing distance was approximately 60 cm. The sizes of the digits were set at 100 pixels (15 mm) for the smaller, and 110 pixels (18 mm) for the larger digits. Center-to-center distance between the digits was 90 pixels (12 mm).

Each trial started with a blank screen for a random duration between 800 and 1200 ms. Subsequently, a pair of digits was presented and remained on the screen until the participants pressed a key on the response box. If participants did not respond within 5000 ms of stimulus onset, the pair of digits disappeared and a prompt on the screen reminded the participants to provide a response on the next trial. Feedback for wrong answers was only given for the ten practice trials at the beginning of the two subtests.

After the number Stroop task was completed, participants received the brief math assessment (BMA-3; Steiner & Ashcraft, 2012) with an additional empty sheet of paper. The BMA-3 was used as a time-effective math achievement test. It consists of ten items that strongly correlate with performance in larger tests of math achievement (i.e., WRAT3 and WRAT4; Wilkinson & Robertson, 2006). Moreover, despite its brevity the BMA-3 covers a wide range of arithmetic problems (addition, subtraction and multiplication of whole numbers and fractions, solving equations, and simplifying complex fractions).

Participants were instructed to solve as many problems as possible within ten minutes. They were informed that the mathematical problems of the BMA-3 are ordered by increasing difficulty. The participants were free to choose in which order they wanted to solve the problems and were allowed to take notes on the empty sheet of paper. After exactly ten minutes, the experimenter collected the test.

Analysis

Group performance (patients with PVD vs. healthy controls) was compared using a Bayesian approach. Bayesian inference implies substantial advantages over traditional null hypothesis significance testing (NHST). In contrast to NHST, the interpretation of data in Bayesian inference is unaffected by sampling intentions and time restrictions of the experimenter. Most importantly, the Bayesian approach allows assessing the relative credibility of parameter values given the data, which is not possible with frequentist methods (Kruschke, 2012; Lee & Wagenmakers, 2005; Wagenmakers, 2007).

All analyses were conducted in R (3.3.2; R Core Team, 2015) using the brms package for Bayesian generalized (non-)linear mixed models (Bürkner, 2017). The parameter estimates were obtained using Markov Chain Monte Carlo sampling (MCMC) with 4 chains of 2000 iterations each (the first 1000 iterations were used as warm-up). We used weakly informative priors on all estimated parameters to minimize the influence of the priors on the estimated posterior distributions. In order to assess model convergence, we visually inspected the chains and verified that the R-hat diagnostic was close to 1. R-hat values larger than 1 are indicative of insufficient convergence of the MCMC chains (Gelman & Rubin, 1992). The estimated parameters were interpreted in terms of their 95 % credible interval (95 % CrI). The 95 % CrI provides the range which contains the true value of a parameter with a probability of 95 % (given the data). This interpretation is not possible (however often erroneously used) with frequentist confidence intervals (Hoekstra, Morey, Rouder, & Wagenmakers, 2014; Nicenboim, 2016). Consistent with previous research using a Bayesian framework, we considered effects to be “strong” if the 95 % CrI did not include zero, or one for odds ratios respectively (Frank, Trompenaars, & Vasisht,

2016; Hofmeister & Vasishth, 2014; Husain, Vasishth, & Srinivasan, 2014; Kruschke, 2012; Nicenboim, 2016).

The odds of giving a correct answer in the BMA-3 were analyzed using a generalized linear mixed model (GLMM) with a binomial link function. We modeled a fixed effect $b_{patients}$, which refers to the dummy-coded effect of patients with PVD vs. healthy controls (i.e., the reference category). We were interested whether the odds of providing a correct answer was smaller for patients with PVD compared to healthy controls (i.e., the odds ratio being smaller than 1). Additionally, we controlled for the fixed effects of each educational level (b_{educ2} : vocational training; b_{educ3} : higher vocational training or university entrance diploma; b_{educ4} : college of higher education or university degree) compared to compulsory education as the reference category (b_{educ1}). Furthermore, we included two random effects for the intercept in order to account for the variance between participants and between items.

Results from the *numerical* and *physical* number Stroop task were analyzed by means of two separate Bayesian GLMM with an ex-gaussian link function for the response times. We used the ex-gaussian link function since it has repeatedly been shown to yield appropriate fits for response time distributions including Stroop tasks (Gu, Gau, Tzang, & Hsu, 2013; Heathcote, Popiel, & Mewhort, 1991; Hervey et al., 2006; Palmer, Horowitz, Torralba, & Wolfe, 2011; Steinhauser & Hübner, 2009). Fixed effects were estimated for *group* (patients with PVD vs. healthy controls), *numerical distance* (1 vs. 2 vs. 5), and *congruency* (congruent vs. neutral vs. incongruent) and all possible interaction terms. We used effect coding for all predictors in order to ease the interpretation of the regression weights (i.e., interpreting an effect at the grand mean instead of setting all remaining effects to zero with dummy coding). Random effects were added

for the intercepts of the participants and trials. Weakly informative priors were used for all effects.

Results

Number Processing

Numerical Number Stroop. Visual inspection of the MCMC chains and the R-hat diagnostic (all ≤ 1.01) indicated good convergence for all estimated parameters. The posterior estimates of the Bayesian GLMM revealed a strong effect for patients vs. controls ($b = 64.04$; 95 % CrI = [0.38; 125.94]). Irrespective of numerical distance and congruency, patients with PVD ($M = 724.96$; 95 % CrI = [679.37; 766.13]) responded slower than healthy controls ($M = 659.56$; 95 % CrI = [620.67; 703.97]). Furthermore, we observed a strong effect for numerical distances of one compared to five ($b = 61.70$; 95 % CrI = [51.43; 72.41]) and numerical distances of two compared to five ($b = 36.55$; 95 % CrI = [26.70; 46.26]). Response times for numerical distances of one ($M = 721.76$; 95 % CrI = [689.75; 750.83]) and two ($M = 696.77$; 95 % CrI = [665.15; 725.80]) were slower compared to numerical distances of five ($M = 660.34$; 95 % CrI = [628.46; 689.31]). Considering the NCE, we observed both a facilitation ($b = -28.38$; 95 % CrI = [-38.78; -17.97]) and an interference effect ($b = 46.18$; 95 % CrI = [33.66; 59.46]). Congruent trials ($M = 658.73$; 95 % CrI = [626.38; 687.80]) lead to faster response times compared to neutral trials ($M = 687.13$; 95 % CrI = [655.04; 715.91]). In contrast, response times were substantially slower for incongruent trials ($M = 733.30$; 95 % CrI = [701.00; 763.03]) vs. neutral trials.

We were particularly interested in possible differences of the SCE or the distance effect in patients vs. controls. However, groups did neither differ in facilitation ($b = 0.51$; 95% CrI = [-19.34; 21.14]) nor interference ($b = 0.60$; 95 % CrI = [-23.27; 25.27]). Furthermore, there was no

difference between patients and controls with respect to the distance effect. Distance effects of one vs. five ($b = -2.26$; 95 % CrI = [-22.90; 18.06]) and two vs. five ($b = -0.87$; 95 % CrI = [-19.98; 18.21]) did not differ between groups.

The absence of group differences in facilitation, interference, and the numerical distance effect is depicted in *Figure 1*, and *Figure 2* respectively.

----- Insert Figure 1 about here -----

----- Insert Figure 2 about here -----

Physical Number Stroop. Computation of the posterior distributions was successful for all estimated parameters as indicated by visual inspection of the MCMC chains and the R-hat diagnostic (all < 1.01). In analogy to the *numerical* number Stroop task, the Bayesian GLMM showed a strong effect of group ($b = 69.46$; 95 % CrI = [26.19; 112.25]). Patients with PVD ($M = 663.34$; 95 % CrI = [630.87; 695.78]) responded generally slower than healthy controls ($M = 593.98$; 95 % CrI = [563.50; 626.21]). We observed no reversed distance effect for numerical distances of one ($b = 0.24$; 95 % CrI = [-8.55; 9.20]) and two ($b = -8.14$; 95 % CrI = [-17.57; 1.23]) compared to five. Participants did not respond differently when the irrelevant dimension (i.e., numerical value) of the two stimuli was separated with a distance of one ($M = 632.43$; 95 % CrI = [601.22; 665.72]), two ($M = 623.86$; 95 % CrI = [592.22; 657.53]) or five ($M = 632.10$; 95 % CrI = [600.30; 665.08]). In terms of the SCE, we only found a strong difference between congruent and incongruent trials ($b = -39.49$; 95 % CrI = [-47.83; -31.30]). Participants responded faster to congruent ($M = 612.66$; 95 % CrI = [582.66; 640.95]) compared to

incongruent trials ($M = 652.05$; 95 % CrI = [622.53; 680.49]). However, we found no facilitation ($b = -9.44$; 95 % CrI = [-79.56; 58.27]) or interference effect ($b = 30.04$; 95 % CrI = [-40.69; 96.96]). Neutral trials ($M = 621.64$; 95 % CrI = [569.28; 679.65]) did not differ from congruent or incongruent trials. Considering the two-way interactions, there was neither a difference in facilitation ($b = 6.75$; 95 % CrI = [-121.93; 132.03]) nor interference ($b = 14.19$; 95 % CrI = [-115.44; 138.21]) between groups. Furthermore, patients and controls showed no differences with respect to the distance effects. Distance effects of one vs. five ($b = -4.03$; 95 % CrI = [-22.13; 14.38]) and two vs. five ($b = -6.47$; 95 % CrI = [-25.19; 11.78]) did not differ between groups.

Posterior means and credible intervals of the group x congruency interaction are plotted in *Figure 3*. The interaction of group x numerical distance is depicted in *Figure 4*. Both figures indicate that there were no differences in facilitation, interference, and numerical distance effects.

----- Insert Figure 3 about here -----

----- Insert Figure 4 about here -----

Math achievement

Visual inspection of the MCMC chains indicated good model convergence. Consistently, the R-hat diagnostic was roughly 1 for all parameters (all R-hat ≤ 1.01). Controlled for educational level, patients with PVD showed more than three times lower odds of providing a correct answer compared to healthy controls ($OR = 0.27$, 95% CrI = [0.05; 0.74]). A model summary is provided in *Table 2*.

In light of the slower response times of patients in the Stroop task, we conducted an additional analysis for the math achievement test. We added the median response time per subject as a marker of individual number processing speed to the Bayesian GLMM. Using this approach, we aimed at controlling for the effect of (low level) number processing speed on performance in the time-restricted arithmetic test. However, the Bayesian GLMM revealed that even after controlling for individual processing speed, the odds of providing a correct answer were substantially lower for patients with PVD vs. controls ($OR = 0.28$, 95% CrI = [0.05; 0.84]).

----- Insert Table 2 about here -----

Discussion

In order to examine the effect of vestibular information on numerical cognition, we compared patients with unilateral PVD and healthy controls by means of two tasks that differed with respect to their complexity. By using a low level numerical cognition task (number Stroop), we were interested whether patients and healthy controls differ with respect to automatic and intentional magnitude processing. A higher level of processing was examined in order to assess whether PVD affects the efficient manipulation of numbers. We found that patients with PVD performed worse in a math achievement test. Considering the number Stroop task, patients with PVD showed slower reaction times compared to healthy controls. However, we found no evidence of impaired automatic or intentional magnitude processing, which is inconsistent with the hypothesis of a specific number deficit (i.e., dyscalculia). Patients with PVD did not differ from healthy controls in terms of facilitation or interference from neither the task-irrelevant numerical value in the *physical* subtest nor the task-irrelevant physical size in the *numerical*

subtest. Furthermore, there was no difference between patients and controls in the number distance effect, which further corroborated the evidence in favor of normal magnitude processing in PVD.

Our findings of impaired math performance are consistent with the frequent subjective complaints of patients with PVD (Harun et al., 2015). They are also in line with the existing, yet scarce empirical evidence of impaired numerical cognition in vestibular patients (Andersson, Hagman, Talianzadeh, Svedberg, & Larsen, 2003; Bessot, Denise, Toupet, Van Nechel, & Chavoix, 2012; Risey & Briner, 1990; Yardley et al., 2002). Unlike previous studies, we demonstrated impaired arithmetic performance under static conditions. Thus, we provide evidence that previously reported numerical deficits cannot be exclusively attributed to dual-task interference (i.e., impaired cognitive performance due to prioritization of an ongoing postural challenge). This idea is supported by other studies showing various cognitive deficits in PVD in static conditions even though the deficits were more pronounced with an ongoing postural challenge (Redfern et al., 2004; Talkowski et al., 2005; Yardley et al., 2001).

We hypothesize that the observed pattern of arithmetic deficits and normal magnitude processing might be a consequence of metabolic changes in parietal areas during acute PVD. It has been shown that acute PVD leads to a complex activation-deactivation pattern of glucose metabolism. While glucose metabolism is increased in the parieto-insular vestibular cortex, metabolic downregulations are observed in the inferior parietal cortex (Bense et al., 2004; Dieterich & Brandt, 2008). There is a large consensus that parietal areas are crucial for numerical cognition (Ansari, 2008; Arsalidou & Taylor, 2011; Dehaene, Piazza, Pinel, & Cohen, 2003; Hubbard, Piazza, Pinel, & Dehaene, 2005) and play an important role in impaired numerical cognition (Ashkenazi et al., 2008; Butterworth et al., 2011; Delazer & Benke, 1997; Kadosh et

al., 2007; Mussolin et al., 2010; Price et al., 2007). While the intraparietal sulcus is mainly involved in magnitude processing, inferior parietal areas such as the angular gyrus have repeatedly been shown to support arithmetic fact retrieval (Delazer, Girelli, Grana, & Domahs, 2003; Göbel, Rushworth, & Walsh, 2006; Grabner et al., 2007, 2009; Zamarian, Ischebeck, & Delazer, 2009). In this light, it is conceivable that PVD leads to transient metabolic downregulations in inferior parietal areas, and this in turn affects arithmetic performance in PVD.

In contrast to evidence in favor of impaired math achievement, normal SCE and distance effects in patients with PVD point to intact automatic and intentional magnitude processing. In this light, our clinical sample clearly differed from the number Stroop performance in individuals with dyscalculia (Ashkenazi et al., 2008, 2009; Kadosh et al., 2007; Rubinsten & Henik, 2005, 2009; Rubinsten et al., 2002). Furthermore, the lack of group differences in terms of the distance effect is inconsistent with the idea of impaired number-space associations in vestibular patients (Smith, 2012). The distance effect is usually interpreted as an indicator for the preciseness of spatial representations of numbers on the internal mental number line (Bugden & Ansari, 2011; De Smedt et al., 2009; Heine et al., 2010; Holloway & Ansari, 2009; Noël, Rousselle, & Mussolin, 2005). In the same vein, it is important to note that we were able to demonstrate that unilateral vestibular deficit does not affect the mental representation of numbers in a random number generation task (Moser et al., 2016). Consistently, unilateral galvanic vestibular stimulation did not induce a bias on the mental number line in healthy participants (Ferrè, Vagnoni, & Haggard, 2013). Our current data adds important evidence that arithmetic difficulties in PVD are not caused by impaired mental representation of numbers.

Taken together, the results from this study suggest that the observed arithmetic deficits are due to impaired arithmetic fact retrieval rather than by abnormal magnitude processing. However, it cannot be excluded that the arithmetic deficit in PVD might – at least partly – reflect a domain-general cognitive deficit rather than a number-specific deficit. In fact, the growing literature on the cognitive consequences of PVD has found deficits in many cognitive domains (see Bigelow & Agrawal, 2015; Hanes & McCollum, 2006; Mast et al., 2014; Smith et al., 2005). If the observed arithmetic deficit is caused by metabolic changes in the inferior parietal cortex – as hypothesized above – it is noteworthy that those structures (e.g., the angular gyrus) do not only support arithmetic fact retrieval but are associated with a general language system that mediates the retrieval of verbally stored facts by phonological processing (Binder, Frost, Hammeke, Rao, & Cox, 1996; Paulesu, Frith, & Frackowiak, 1993; Price, 1998, 2000; Simon, Mangin, Cohen, Le Bihan, & Dehaene, 2002).

Importantly, it is possible that the cause of arithmetic deficits goes beyond the role of impaired fact-retrieval. For example, weak arithmetic performance of PVD patients could be a consequence of the time-restricted nature of the arithmetic test. In fact, despite normal SCE and distance effects, the patients showed slower response times in both number Stroop subtests. Slower response times might reflect an indirect effect of decreased alertness due to disruption of the circadian rhythm, which has been reported in rodents and humans with vestibular loss (Martin et al., 2015, 2016). Thus, the observed arithmetic deficit could be attributable to general slower processing speed in PVD. Nevertheless, we consider this possible interpretation unlikely since additional analysis showed that the patients with PVD still performed worse in the arithmetic test if the effect of individual processing speed from the number Stroop task was controlled for.

As another domain-general mechanism that is implicated in mathematical tasks, it is important to consider the role of executive functions. By definition, executive functions are responsible for the control and regulation of cognitive processes during performance in complex tasks (Miyake et al., 2000). In fact, a closer look at the previous studies that found numerical deficits in vestibular patients reveals that those deficits cannot be explained independently of executive functions. Yardley et al. (2002) found deficits in counting backwards while patients with PVD were seated on an oscillatory chair and performed a body orientation monitoring task. Performance in dual-tasks relies on intact executive functions in order to constantly allocate attention between concurrent tasks (Logie, Cocchini, Delia Sala, & Baddeley, 2004). Moreover, counting backwards requires monitoring and dynamically manipulating the contents in working memory. As such, the counting process is dependent on the updating component of executive functions (Logie & Baddeley, 1987; Miyake et al., 2000). Similarly, in the first study that claimed dyscalculia in vestibular patients (Risey & Briner, 1990), patients with central vestibular disorders performed worse in counting backwards and in the backward digit span. In our view, these results demonstrate primarily impaired updating and do not allow for direct conclusions regarding an arithmetic deficit.

Consequently, we argue that impaired executive functions play a crucial role in the arithmetic deficit of patients with PVD. It is broadly accepted that executive functions (primarily updating) do not only support counting but also exert dominant influence on arithmetic performance (Andersson, 2008; Bull & Scerif, 2001; Deschuyteneer, Vandierendonck, & Muyllaert, 2006; DeStefano & LeFevre, 2004; Jarvis & Gathercole, 2003; Logie, Gilhooly, & Wynn, 1994; Passolunghi & Pazzaglia, 2004; St Clair-Thompson & Gathercole, 2006; van der Sluis et al., 2007; van der Ven, Kroesbergen, Boom, & Leseman, 2012). In complex arithmetic

problems, updating is needed to replace items held in working memory with newer, more relevant information for the solution of a given (arithmetic) problem (Hitch, 1978; Morris & Jones, 1990). Moreover, it has been proposed that updating also influences performance in simple arithmetic problems where the result can be retrieved from long-term memory (Deschuyteneer et al., 2006).

Furthermore, it is important to point out that executive deficits were found in patients suffering from PVD. Grimm et al. (1989) reported executive deficits in patients with perilymph fistula syndrome. Black et al. (2004) found difficulties in prioritizing tasks in patients with complete vestibular loss. Moreover, we recently showed that patients with PVD produce more redundant sequences in a random number generation task, which is indicative of impaired updating (Moser et al., 2016). Considering the essential role of executive functions in mathematical tasks, there is strong evidence that an executive deficit is causally relevant for impaired math achievement in patients with PVD. Identifying the pathways through which lack of vestibular input might affect executive functions requires further investigation. For example, Hitier et al. (2014) have discussed a pathway that might transmit vestibular information via the cerebellum and the ventral lateral nucleus to the parietal cortex. Intriguingly, both the cerebellum and the parietal cortex play an important role for executive functions (Jahanshahi, Dirnberger, Fuller, & Frith, 2000; Koziol, Budding, & Chidekel, 2012).

Conclusions

We were able to empirically support anecdotal evidence of impaired math performance in PVD. Patients with PVD performed worse in a math achievement test but showed normal number magnitude processing. Our findings are inconsistent with the hypothesis of a specific

number processing deficit in PVD (i.e., dyscalculia). We argue that impaired executive functions are – at least partly – responsible for the observed arithmetic deficit. Poor math performance can have severe consequences for activities of daily living such as handling finances (Harun et al., 2015). Moreover, it might also substantially impair performance at the workplace (Parsons & Bynner, 2005). Thus, it is crucial to follow-up on the mechanisms that cause mathematical deficits in PVD. A relatively large body of experimental evidence can be used to design adequate tools for the diagnosis and rehabilitation of patients with PVD suffering from arithmetic or other cognitive deficits.

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Table 1

Demographics and clinical data of the patients with peripheral vestibular deficit (PVD) and healthy controls.

	Patients with PVD	healthy controls
N	20	21
sex (female / male)	9 / 11	10 / 11
Age (M; SD)	47.11 (13.55)	46.19 (13.75)
Handedness (right /left)	18 / 2	19 / 2
Vertigo onset interval (days)	11.1 (5.48)	-
Neurotological examination (abnormal / normal / n.a)		
- bithermal caloric testing	20 / 0 / 0	-
- pendular rotatory testing	11 / 9 / 0	-
- cervical vestibular evoked myogenic potentials	5 / 14 / 1	-
- video head impulse test	10 / 10 / 0	-
- dynamic visual acuity	5 / 14 / 1	-

Notes.

n.a. not assessed

Table 2. *Model summary of the Brief Math Assessment (BMA).*

Parameter	Estimate	CrI	Eff.sample	Rhat
intercept	1.41	[-3.44 ; 6.73]	1070	1
grouppatients	-1.54	[-2.91 ; -0.30]	2742	1
educ3	2.31	[-0.54 ; 5.4]	1955	1
educ4	2.86	[0.09 ; 5.95]	2025	1
educ5	3.56	[0.59 ; 6.67]	2006	1

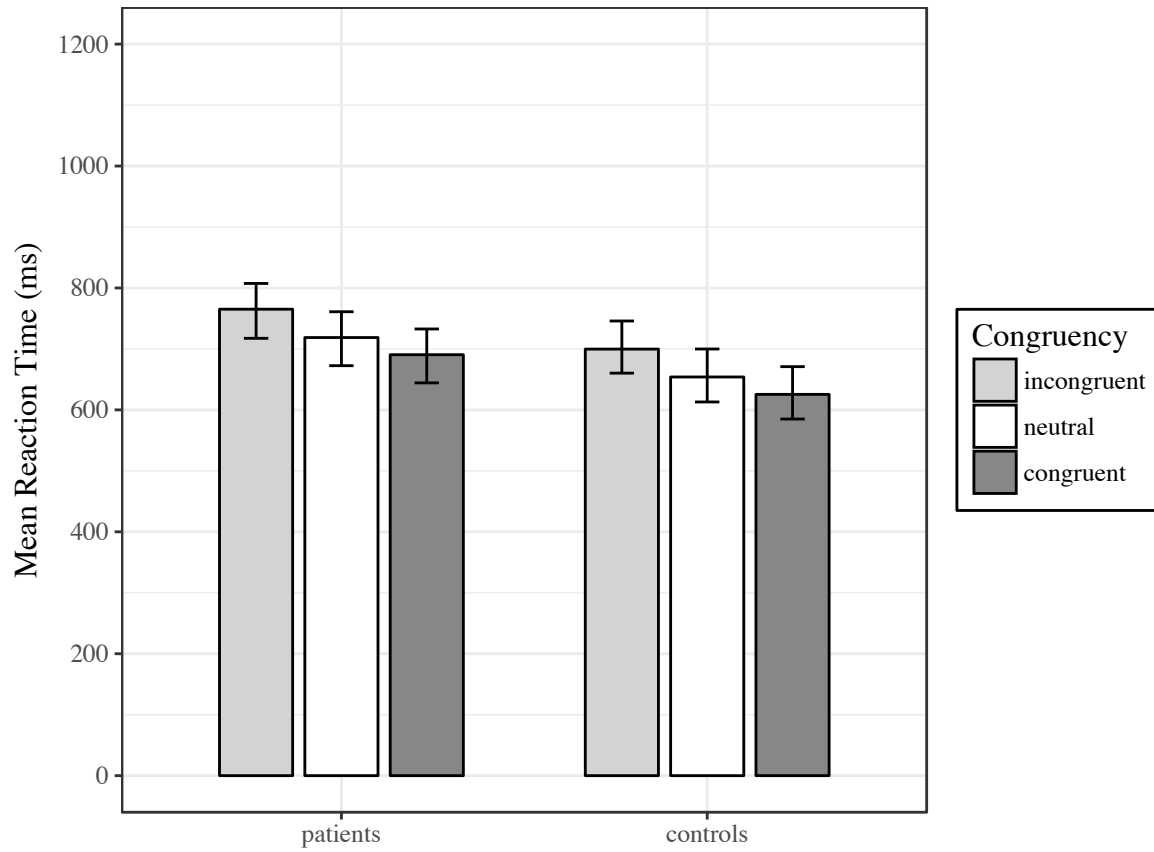


Figure 1. Posterior estimates of mean reaction times and 95 % credible intervals for the congruency effect in the numerical number Stroop task.

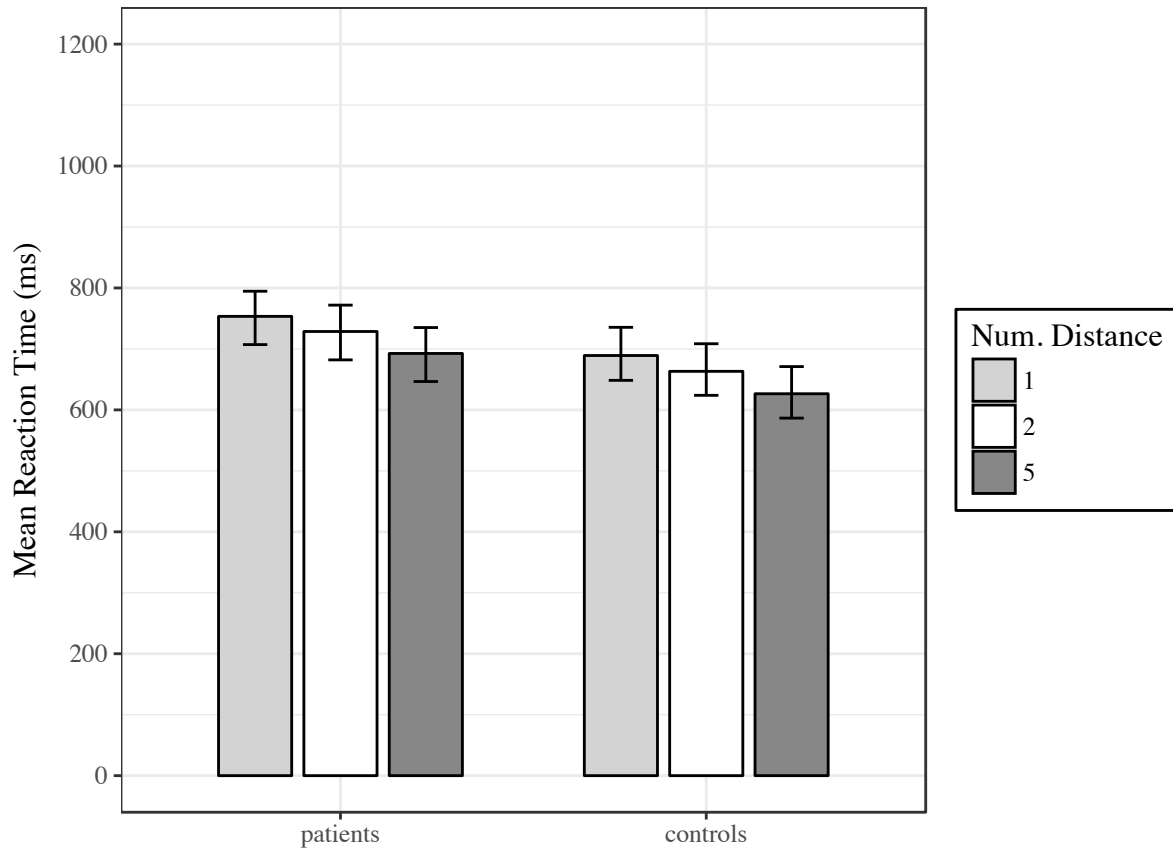


Figure 2. Posterior estimates of mean reaction times and 95 % credible intervals for the number distance effect in the numerical number Stroop task.

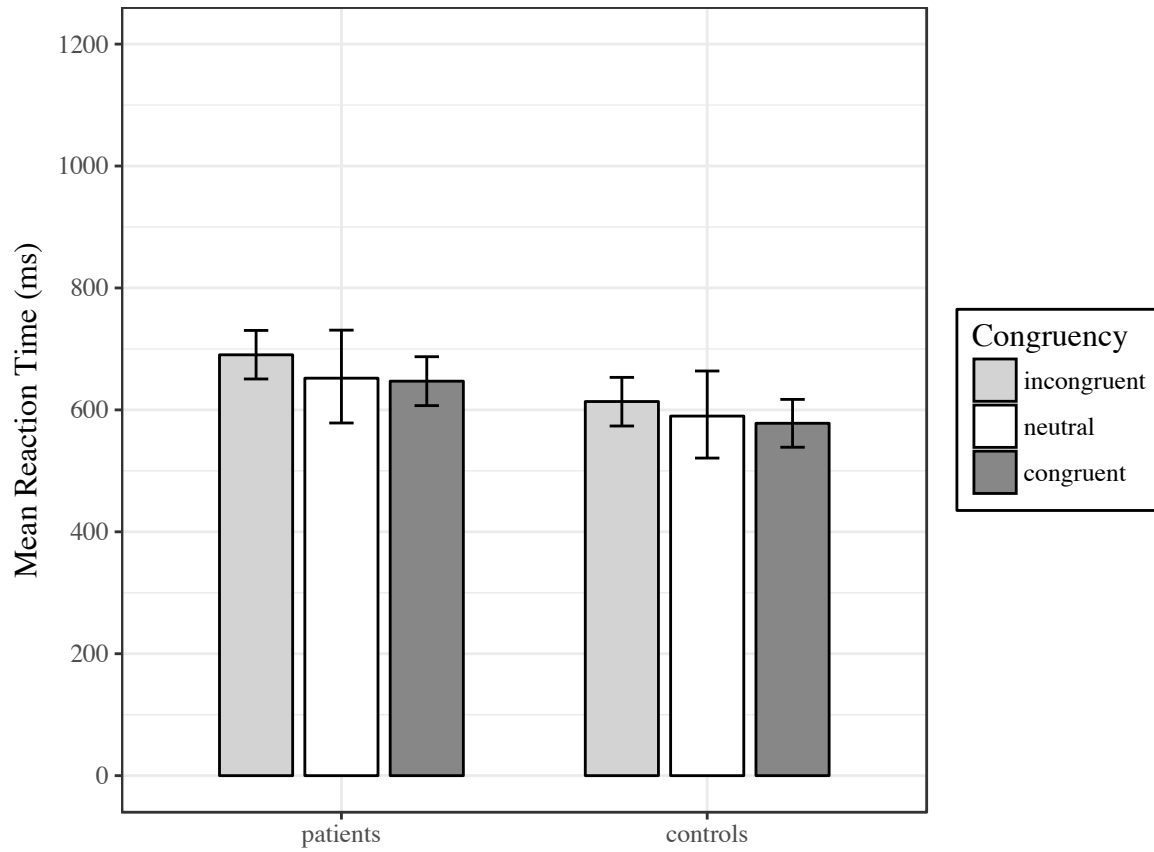


Figure 3. Posterior estimates of mean reaction times and 95 % credible intervals for the congruency effect in the physical number Stroop task.

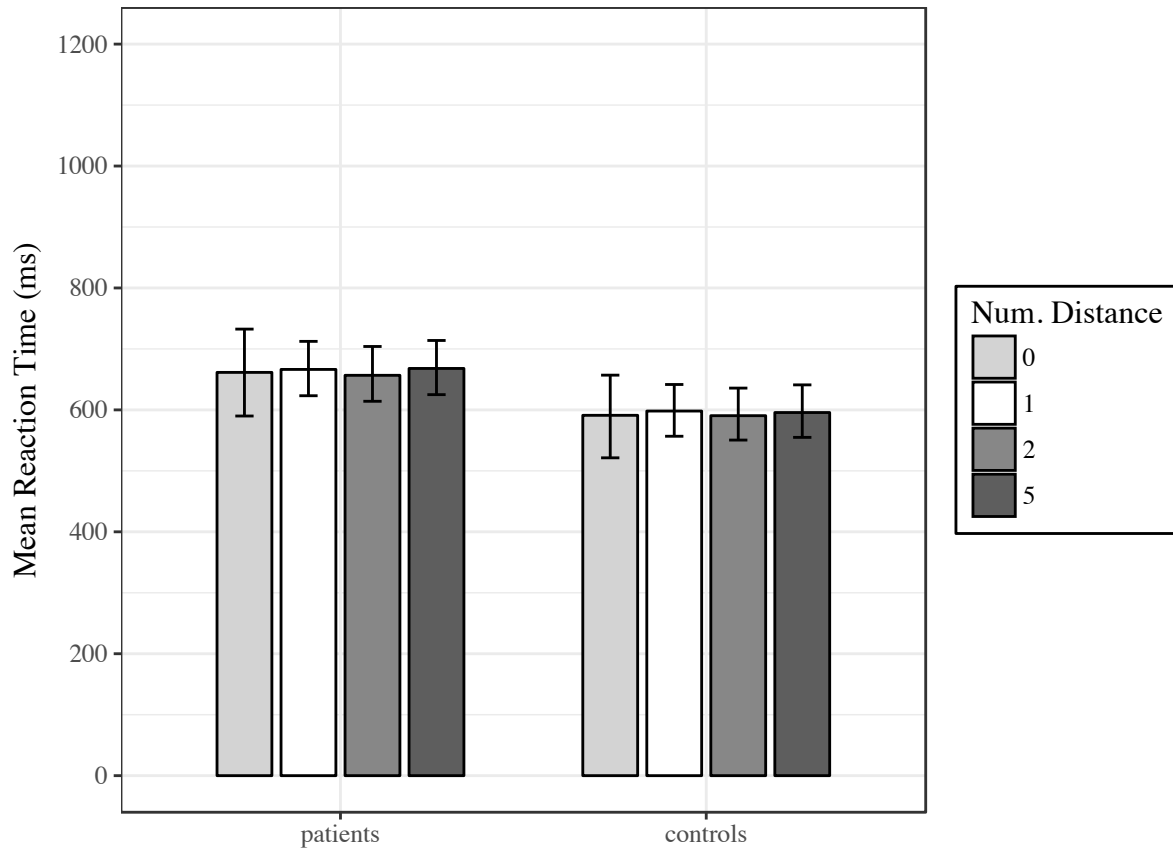


Figure 4. Posterior estimates of mean reaction times and 95 % credible intervals for the number distance effect in the physical number Stroop task.