the concept of zinc deficiency anemia in healthy school children which was not described before. Smaller head circumferences in zinc deficient children might contribute to cognitive deficiencies.

**NUTRITIONAL STATUS AND FEEDING PROBLEMS IN PATIENTS WITH CEREBRAL PALSY**

M Ceylan, Y Karal, S Karaslınhoğlu, B Tokuç, Department of Pediatrics, Division Pediatric Neurology, Department of Public Health, Medical Faculty of Trakya University, Edirne, Turkey

**Aim** The nutritional status and feeding problems in children with cerebral palsy results in growth retardation, increased morbidity and mortality. The aim of this study is to evaluate the nutritional status and feeding problems of cerebral palsy patients followed up by Medical Faculty of Trakya University, Department of Pediatrics, Division Pediatric Neurology.

**Methods** The medical histories and full physical examination findings of 66 cerebral palsy patients were recorded as weight, height, body mass index, circumference measurements of head, arm, chest, fathom length, biacromial length, lower leg length, and subcutaneous fat thickness of triceps and subscapular region. The 3-day diet for each patient was evaluated by a special computer program called BERIS. Patients’ blood hemoglobin, iron levels and binding capacity and serum ferritin, vitamin B12, folic acid levels were also evaluated.

**Results** The majority of patients were Spastic Cerebral Palsy (%89), %6 were Ataxic and %5 were Mixt type (Quadriplegic/Distonic). The weight, body mass index, subcutaneous fat thickness of subscapular region, biacromial length, arm-chest circumference, blood iron levels, dietary calorie and fiber intakes of patients with severely affected cerebral palsy, especially in the Quadriplegic/Mixt types, were lower than the mild and moderately affected ones.

**Conclusions** The nutritional status and dietary intakes of the severely affected cerebral palsy are worse than the others in our study, as mentioned in the literature. To prevent complications a multidisciplinary approach and close follow up have to be done.

**EFFECTS OF LAMOTRIGINE ON THE LANGUAGE AND PROBLEM SOLVING ABILITIES IN NEWLY DIAGNOSED PEDIATRIC EPILEPTIC PATIENTS**

SJ Kim, SH Chae, YM Choi, KS Kim. Department of Clinical Speech Pathology, Chonbuk National University Medical School, Jeonju Public Health Center, Jeonju, Republic of Korea

**Purpose** The purpose of this study was to investigate the effects of lamotrigine on language and problem solving abilities in newly diagnosed pediatric epileptic patients.

**Methods** Sixty-seven newly diagnosed pediatric epileptic patients. (Male:Female=43:24, Mean age: 8y 9m ± 2y 4m), who were investigated from 2006 to July 2011. We performed a standardized full articulation tests and Peabody picture vocabulary test-revised. Test of Problem Solving (TOPS), Mean Length of Utterance in words (MLU-w), comparison of Precise Articulation, Computerized Speech Lab were used to assess the language function before and after initiation of lamotrigine. Starting dosage of lamotrigine was 1mg/kg for the first 7–14 days; increased to 2mg/kg for the next 14 days and increased up to 7mg/kg/day (or 200mg/day).

**Results** First, TOPS showed that the abilities of problem solving were not improved after initiation of lamotrigine (32.3±13.0 vs 34.5±12.5, P >0.05). All parameters: Causal reason (11.6±4.5 vs 12.1±4.2), solution rationcination (13.4±5.4 vs 13.6±5.6), beginning guess (7.9±4.2 vs 8.7±4.0) were not changed after initiation of lamotrigine. Second, MLU-w did not reduce after taking medicine (4.2±1.4 vs 4.2±1.3). Third, the receptive language function was significantly improved after taking lamotrigine in PPVT (8y ±4m2y 4m vs 8y 10m±2y 4m, P <0.01). However, there were no significant changes in percentages of precise articulation and error pattern of consonants after taking lamotrigine (98.3% to 99.1%, P >0.05).

**Conclusions** Our results suggest that lamotrigine can be used without significant negative effects on language function. Moreover, language functions, especially receptive language, were improved after lamotrigine initiation.

**SPECTRUM OF NEURAL TUBE DEFECTS AFTER PRENATAL ANTI-EPILEPTIC DRUG EXPOSURE: EXTENSIVE CASE SERIES**

MH van Dijk, S Bulk, ACC van Oppen, ACG Egberts, ER Heerdink, Lindhout. Department of Clinical Pharmacy, Department of Medical Genetics, Department of Obstetrics and Perinatology, University Medical Center Utrecht; Department of Pharmacoeconomics and Clinical Pharmacology, Utrecht Institute for Pharmaceutical Sciences; SEIN - Epilepsy Institute, Utrecht, The Netherlands

**Background** Most pregnant women with chronic active epilepsy need to use antiepileptic drugs (AEDs) during pregnancy to prevent epileptic seizures that may threaten maternal and fetal well-being. Valproic acid (VPA) and carbamazepine (CBZ) have been associated with an increased risk of neural tube defects (NTDs) in the exposed fetus.

**Aim** To investigate the spectrum of neural tube defects and associated central nervous system (CNS) and non-CNS malformations after prenatal exposure to CBZ and/or VPA.

**Methods** NTDs in pregnancies in which CBZ and/or VPA were used during the first trimester were collected from 1970–2012 in the Netherlands. Type and location of the NTDs, associated CNS and non-CNS major malformations and relevant patient characteristics were analysed.

**Results** 87 pregnancies were included. NTDs after exposure to CBZ or VPA were mostly caudally located, whereas a combination of CBZ and VPA was associated with a location shift of the NTD to the rostral side (Figure 1). There were no differences between CBZ and VPA in the percentage of associated CNS malformations and non-CNS malformations circa 75% and 45%.

Abstract 1508 Figure 1 Antiepileptic drugs versus type and location of NTDs

**Conclusions** The combination of VPA and CBZ shows a tendency towards a more rostral location (lumbar) which may have more severe functional consequences. Current findings confirm that NTDs associated fetal exposure to VPA and/or CBZ are serious and frequently accompanied by other CNS and non-CNS malformations.