30 millimetres in the different areas of the brain. Intrauterinely, there were discovered rhabdomyomas in 3 children in a term 35-37 weeks of pregnancy. In spite of focal lesions of brain structures, in all children were noticed generalized tonic-clonic seizures (resistant) from 5 to 10 a day. During the treatment of Topamax (to 10 mg/kg), frequency of attacks decreased, however, there was no control. Apart from anticonvulsants (Topamax) the children started to receive the preparation Vigabatrin (from 40 to 100 mg/kg).

Results: combination of anticonvulsant therapy led to the decrease in the number of attacks in 4 children from 1 attack in a day to 1 attack in a month. In 5 children attacks were halted.

Conclusion: this work stresses the necessity of timely magnetic resonance tomography and combination of Topamax and Vigabatrin for optimization of treatment of resistant epileptic attacks in children with a tuberous sclerosis.

589

CONGENITAL CENTRAL HYPOVENTILATION SYNDROME IN THREE INFANTS

V. Schiza, M. Kazantzi, D. Paraschou, H. Barbaressou, P. Kalabalikis, T. Hatzis

Pediatric Intensive Care Unit, Agia Sofia Children's Hospital, Athens, Greece

Background and aims: Congenital central hypoventilation syndrome (CCHS) is a rare syndrome characterized by the failure of automatic control of breathing with absent or negligible sensitivity to hypercapnia and hypoxaemia, primarily during sleep. It is associated with a number of conditions affecting or derived from the autonomic nervous system.

Methods: Three infants with CCHS were diagnosed in our tertiary pediatric hospital over five years.

Results: All three infants were born full term. During neonatal period they presented prolonged apneas and intermittent cyanosis, in the absence of cardiac, pulmonary or neuromuscular disease. They were treated by tracheostomy and mechanical ventilation. After initial respiratory dependency, they became able to sustain normal gas exchange during wakefulness. Two of three cases had Hirschsprung disease and mild generalized hypotonia. One experienced seizures unrelated to antecedent hypoxemia and cardiac arrhythmias. Dilated,

nonreactive pupils and lack of circadian temperature variation occurred in one. Cerebrovascular malformations, tumors of neural crest origin and pulmonary hypertension were not observed. Mutations of the PHOX2B gene were detected in all cases. At the age of 2 to 5 years, they are still under ventilatory support during sleep. The eldest child has been successfully decannulated and he is on NIV through a nasal mask. All three children are managed at home, with adequate growth, progress in achieving motor milestones and low morbidity.

Conclusions: Infants with CCHS represent a spectrum of severity. With early diagnosis and careful ventilatory management these children can engage in activities typical of children of their age.

590

THE ROLE OF SICAM-1, SVCAM-1 ADHESION MOLECULES IN THE HIPOXYC-ISHEMIC ENCEPHALOPATHY OF NEWBORNS

P. Orucova, N. Tagiyev, S. Alaskarova

Neonatology, Azerbaijan Medical University, Baku, Azerbaijan

Background and aim: Soluble intracellular cell adhesion molecule-1 (sICAM-1) and soluble vessel cell adhesion molecule-1 (sVCAM-1) produced from endothelium by the influence of hypoxia, infection, immunocomplex ensures migration of leukocytes transendothelial to ischemic tissue.

The aim of our work is to determine the level of adhesion molecules (AM) in Central Nervous System (CNS) damages of risk group newborns, and its role in this process.

Methods: Umbilical cord blood and peripheral blood serum samples of newborns were collected for measuring the levels of sICAM-1, sVCAM-1. CNS damages were determined by neurosonography and dopplerographic examinations.

Newborns are divided into 3 groups in order to determine the degree of causal relationship between the level of AM and CNS damages. 1st group (main group) included 35 (43,75%) newborn infants with CNS hypoxic-ischemic damages, 2nd group (comparison group) 25 (31,25%) newborn infants entering risk group but not observed CNS damages, 3rd group (control group) 20 (25%) healthy newborn infants whose condition are sufficiently appreciated.

Results: sICAM level in the main group {543,2 (267-729)ng/ml} was relatively higher than comparison {399(230-584) ng/ml, $p < 0,05$ } and control groups {379 (117-507) ng/ml; sVCAM-1 level in the main group {350 (700-2100) ng/ml} was higher than comparison and control groups, too {820 (630-1100) ng/ml; $p < 0,05$; 625 (530-1020) ng/ml; $p < 0,001$ }

Conclusions: Our study showed, that endothelial injury play a major role in the CNS pathology of newborns.

591

MRI AND OUTCOME OF NEONATES TREATED WITH MILD HYPOTHERMIA AFTER PERINATAL ASPHIXIA

S. Malguzzi, G. Paterlini, G. Kullmann, C. Provero, P. Tagliabue

NICU, MBBM Foundation, S. Gerardo Hospital, Monza, Italy

Background and aims: HIE severity and MRI findings are predictors of neurological outcome in asphyxiated neonates. Mild hypothermia may reduce the severity of basal ganglia and thalamus lesions, which are predictive of abnormal outcome. Moreover hypothermia does not induce any unexpected cerebral lesions. We compare MRI lesions with HIE grade and neurological outcome.

Methods: From December 2006 in S. Gerardo Hospital NICU, mild hypothermia (selective head cooling) is the standard of care for the hypoxic-ischemic newborns. Patient's selection and treatment were done according to the protocols employed in the published randomized trials. The treated neonates were 26, 12 infants had hypoxic-ischemic encephalopathy (HIE) grade II and 14 had HIE grade III.

MRI (T1-T2 and diffusion-weighted sequences) was performed within 15 days of life and classified according to the Mercury's classification. The neurological outcome was assessed at 12 months.

Results: MRI showed cerebral lesions in 71% (mostly mild). Unexpected MRI lesions weren't seen. There is no correlation between severity of HIE and MRI lesions; moreover there is not correlation between the severity of MRI cerebral lesions and motor delay or cerebral palsy.

Conclusions: in our population of cooled infants, neurological outcome is not associated with the degree of HIE and the severity of MRI lesions

592

CEREBRAL AUTOREGULATION DURING 24 HOURS IN PRETERM INFANTS MEASURED BY NEAR-INFRARED SPECTROSCOPY

E.A. Hummel, E.M.W. Kooi, E.A. Verhagen, A.F. Bos

Pediatrics, Division of Neonatology, University Medical Center Groningen, Groningen, The Netherlands

Background: Preterm infants might have a disturbed cerebral autoregulation. An indirect way to monitor cerebral blood flow is by Near-Infrared Spectroscopy (NIRS). NIRS measures the regional cerebral tissue oxygenation (r_cSO_2). By combining r_cSO_2 with the transcutaneous arterial oxygenation ($tcSaO_2$), the Fractional Tissue Oxygen Extraction (FTOE) can be calculated. In case of diminished cerebral blood flow, FTOE will increase.

Aim: To determine whether preterm infants have cerebral autoregulation.

Patients and Methods: In this observational study we included preterm infants < 32 weeks gestational age, in the first 72 hours after birth. For 24 hours, r_cSO_2 , $tcSaO_2$, and mean arterial pressure (MAP) were measured every five minutes, and FTOE was calculated: $FTOE = (tcSaO_2 - r_cSO_2) / tcSaO_2$. Clinical data were collected (gestational age, birth weight, Apgar scores, Hb, PCO_2 , ventilation mode). Spearman's correlation coefficients were calculated in each individual patient to show absence of autoregulation, indicated by a significantly negative correlation between MAP and FTOE.

Results: We included 25 infants with a median gestational age of 28.8 weeks and a median birth weight of 1245 gram. In 10 infants we found a negative correlation (ρ ranging from -0.432 to -0.156) between MAP and FTOE over 24 hours, suggesting absent autoregulation. None of the clinical variables influenced the presence or absence of autoregulation.

Conclusion: Cerebral autoregulation appears to be absent in almost half of the studied infants, suggested by the presence of a negative correlation between MAP and FTOE. We could not explain the presence or absence of autoregulation by clinical variables.