

On March 16-20, 1986, the 4th International Child Neurology Congress, sponsored by the International Child Neurology Association, was held in Jerusalem, Israel. The Congress President was Shaul Haefl, MD and the Chairman of the Scientific Program Committee was Joseph H. French, MD. The *Journal of Child Neurology* is pleased to be able to publish the abstracts of that congress. Generous financial support for the publication of these abstracts was provided by the Division of Neuropathology of the Department of Pathology of The University of Oklahoma College of Medicine and the Oklahoma Teaching Hospitals (Oklahoma Memorial Hospital and Oklahoma Children's Memorial Hospital), Oklahoma City, OK.

#### Submitted Papers to ICNA Congress

##### Carbamazepine Slow-Release in Pediatrics: A New Dimension of an Old Drug

M. Albani and R. Schringer-Mansour, Hamburg, FRG

Carbamazepine therapy in pediatrics, in particular in infancy and early childhood, carries the specific problems of carbamazepine pharmacokinetics, i.e. autoinduction of metabolism together with an age specific fast drug turnover. Thus, very variable and almost low concentrations contribute to a limited efficacy and substantial side effects. 24 hrs concentration profiles in 16 children (6 on mono-, 10 on multiple therapy) aged 8 mo to 14 years showed an about 300% variation between trough and peak level despite a 3-4 times daily dose regimen. The level prior to morning dose (p.m.d.) was very low in all cases. In spite of a mean dose of 29 mg/kg, 12 were not sufficiently controlled or developed unacceptable side effects with increased doses. They suffered from secondary generalized (4), simple partial (4), and complex partial (4) seizures. After changing therapy to carbamazepine slow release (5 mono-, 7 multiple therapy), the 24 hrs concentration profiles were repeated and the incidence of seizures and side effects carefully documented. With the retard preparation: (a) the variation was reduced to 140% with only 2(45%)-3(35%) doses/day; (b) the level p.m.d. was significantly higher in all cases; (c) the incidence of side effects significantly lower; and (d) the seizure control improved dramatically by 65-100%. Thus, it can be suggested that the slow-release preparation is essentially more effective by more stable and higher plasma concentrations which can be achieved with a far less risk for side effects and with less frequent dosages. The efficacy of carbamazepine under these conditions might extend the indications for this particular drug in childhood epilepsy with therapeutic difficulties.

##### Walker-Warburg Syndrome: Etiological Considerations

Luis A. Alvarez, Joseph Mayeal, Takeo Kato, Josefine F. Llona, Lewis P. Singer and Asao Hirano, Bronx, N.Y.

Walker-Warburg Syndrome (WWS) is a rare congenital disorder of unclear etiology that simultaneously affects ocular and cerebral embryogenesis. While many believe that the disorder is due to a disruptive process present during the entire embryogenesis (i.e. infectious disease, poor perfusion), others propose teratogenic environmental factors (i.e. alcohol). Nevertheless, an autosomal recessive inheritance is strongly suggested by the 3 sibships occurring in the 20 reported cases. In fact, recent studies suggest that WWS may be a variant of two other familiar syndromes: congenital muscular dystrophy of Fukuyama and cerebro-oculo-muscular syndrome. Some investigators postulate, however, that environmental teratogenic factors existing in one pregnancy may still be present in subsequent pregnancies, accounting for multiple affected siblings. We present a boy with the characteristic features of the WWS (i.e. severe psychomotor retardation, ocular dysplasia, hydrocephalus, and occipital encephalocele) whose dizygotic twin was a normal female. Prenatal and perinatal histories were unremarkable except for the mother's history of alcohol abuse during pregnancy. Both placentas were intact. TORCH studies and metabolic screen were negative; chromosome studies were normal. Death occurred at age 6 months. At autopsy, we found several abnormalities which included ocular dysplasia and cerebral polymicrogyria. In spite of the mother's use of alcohol, the coexistence of a normal and an affected dizygotic twin, in the presence of normal intrauterine conditions, supports genetic mapping or vascular insufficiency as the etiology rather than infectious or environmental factors. Based on neuropathological findings further etiological considerations are presented.

##### The Growing Role of Prematurity in the Etiology of Infantile Spasms

Noami Amir, Aaron Klair and Ruth S. Shalev, Jerusalem, Israel

Whereas the advent of the neonatal intensive care units (NICU) has reduced the incidence of neurologic impairment in term babies, their ultimate effect on the morbidity of premature infants is still controversial. Follow up studies on premature infants have not mentioned infantile spasms (IS) as a complication (Dubowitz LMP, Dubowitz V, Palmer PG et al: Correlation of neurologic assessment in the premature newborn infant with outcome at one year. *J Pediatr* 1984; 105:452-56) nor has prematurity been cited as a specific etiology in the pathogenesis of this disorder (Lombroso CT: A prospective study of infantile spasms: clinical and therapeutic correlations. *Epilepsia* 1983; 24:135-58). In order to assess the role of prematurity on IS, we retrospectively reviewed the charts of 41 children with IS diagnosed between the years 1970-1984. The diagnosis was based on typical seizure phenomena, a hypsarrhythmic EEG, and appropriate age. Between the years 1970-1976, before the advent of the NICU, 18 children with IS were diagnosed, 9 of whom had symptomatic IS. One of these 9 children had been born prematurely. By contrast, of the 23 children diagnosed between 1977-1984, after the opening of NICU in Jerusalem, 17 were symptomatic of which 6 (35%) were premature babies. This increase in the incidence of IS as a sequela of prematurity is significant ( $p$  less than 0.001). Considering the incidence of prematurity in the general population is 7%, 35% may indicate an increased susceptibility of these infants for IS. Since the early identification and treatment of IS is of prognostic importance, we suggest that premature infants be followed as a special risk group for this disorder.

##### Normal Sleep Characteristics in Sotos' Syndrome

Noami Amir, Ruth S. Shalev, and Avraham Steinberg, Jerusalem, Israel

Sotos' syndrome is characterized by abnormally rapid growth during infancy and early childhood, typical facies, macrocephaly, and mental retardation. Other neurological abnormalities include seizures, hypotonia, abnormal EEG, and enlarged ventricles. The prominent neurological features and the absence of demonstrable endocrine disturbances have led to the hypothesis that the condition reflects a diffuse disorder in the central nervous system, including mechanisms mediating growth. Sleep, a brain stem function, is known to influence neurological function and growth. Opportunities to study sleep structure in accelerated growth are few. We therefore thought it appropriate to study the sleep characteristics of three children with Sotos' syndrome. All 3 were males aged 5, 8, and 13 years. Serial polysomnograms were performed on 3 consecutive nights. Eye movements, electromyograms, ECG, and respiration were monitored in addition to the EEG. Although 1 of the children had epileptogenic activity on his EEG, it did not interfere with sleep staging. Sleep stages were scored according to Rechtschaffen and Kales.

Our results demonstrated normal sleep patterns with normal percentages of stage 3-4 and near normal REM sleep. The values of stage 3-4 ranged from 24-37% of total sleep and for REM 17-19% of total sleep. There was normal transition from deep sleep to REM and age appropriated distribution of sleep stages throughout the night. Our results indicate that cerebral nervous system function influencing sleep patterns are not impaired in these children with Sotos' syndrome.

##### The Use of Computers for Augmentative Communication for Very Young Children with Cerebral Palsy

Noami Amir and Judith Wine, Jerusalem, Israel

Augmentative communication systems in the form of picture, symbol and alphabet boards have been developed for the nonspeaking child with cerebral palsy (CP). The Bliss symbol system is a language based on ideographic visual symbols which becomes more complex commensurate with the child's cognitive development. It has been adapted to answer the communication needs of brain damaged pre-school children whose deficits make vocal and gestural language impossible. The symbols are displayed on a board and the child learns to point manually or focus visually to indicate the sequence of symbols expressing his thoughts. The micro-computer is the logical extension to the symbol board in that it provides a permanent printed record of the child's communication and may in the future be able to "speak" for the child. However, due to severe motor deficits and limited hand function, many CP children are unable to operate the standard keyboard and alternative methods of accessing must be sought. Moreover, our experience

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