Relative Power of Delta_avg_AVG (0.523±0.155 vs 0.356±0.183, p=0.0030), Decorrelation Time_std_AVG (0.019±0.006 vs 0.013±0.008, p=0.0261), Decorrelation Time_snr_STD (2.753±1.052 vs 2.278±1.618, p=0.0358), Relative Power of Delta_avg_SNR (6.581±6.194 vs 4.634±5.239, p=0.0065), Relative Power of Gamma_std_SNR (0.656±0.252 vs 0.491±0.244, p=0.0483), and Relative Power of Delta_snr_AVG (8.590±8.433 vs 4.640±1.589, p=0.0065) in well-controlled group than in refractory group. On the contrary, there were significantly lower Spectral Edge Frequency_avg_AVG (4.232±1.145 vs 5.530±1.338, p=0.0083), Kurtosis_snr_STD (1.298±0.327 vs 1.694±0.306, p=0.0044), and Wavelet_db4_EnergyBand_5_snr_STD (1.900±0.788 vs 2.497±0.617, p=0.0261) in well-controlled group than in refractory group.

Conclusions: Therefore, the developed method is a useful tool to identify the possibility of developing refractory epilepsy in patients with idiopathic epilepsy. The analysis yielded a weighted precision rate of 94.2%.

29 The Chinese-Western Integrative and Effective Therapy on Children of ASD

中西整合有效治療自閉症

Ching-Ding Roan
Hsin-An Clinic, Hsinchu
阮慶定
新竹市欣安診所

Background: We use non-invasive, no medication and no side effect therapy to treat children of ASD. We practically find all treatable symptoms and signs to perform integrative and effective management on children of ASD.

Methods: Children's ASD symptoms & signs must be managed, which can be categorized as insomnia, breathing problems while sleeping, diseases causing sleep problems, and gastrointestinal problems. We treat children of ASD with medical instruments via meridians, such as Mora-Bioresonance, Color Therapy, Theragem, and Airnergy. After further management, we use seven indicators to evaluate effectiveness, such as: times of treatment, eye contact, comprehension, oral expression, reciprocal communication, cognitive learning and negative performance.

Results: Totally 39 cases of ASD were enrolled. There was a male predominance with 37 boys and 2 girls in study. Thereinto, 46%(18 of 39) in 2~3Yr, 21%(8 of 39) in 4~5Yr, 25.4%(10 of 39) in 6~8Yr, and 7.6%(3 of 39) in >9Yr. Four categories were necessary to be managed, including 95%(37 of 39) of total cases with insomnia, 85%(33 of 39) with breathing problems while sleeping, 87%(34 0f 39) with diseases causing sleep problems, and 77%(30 of 39) with gastrointestinal problems. The more improvements parents observed, the more confidence they had and continued treatment persistently. Therefore, firstly estimate times of treatment: <10 times is 7.6%(3 of 39), >10 times is 31%(12 of 39), >20 times is 25.4%(10 of 39), >30 times is 21%(8 of 39), >40 times is 15%(6 of 39); Other estimations of effectiveness after management:1.Eye contact improved in 67%(26 of 39) 2.Comprehension improved in 90%(35 of 39) 3.Oral expression improved about 82%(32 of 39) 4.Reciprocal communication improved in 67%(26 of 39) 5.Cognitive learning improved in 72%(28 of 39) 6.Negative performance decreased in 75%(29 of 39)

Conclusions: If children of ASD could be managed well in insomnia, breathing problems while sleeping, diseases causing sleep problems and gastrointestinal problems, their parents could see children's significant changes and improvement. In those cases, healing success of comprehension and oral expression is especially remarkable.

30 PRRT2 Mutations in Paroxysmal Kinesigenic Dyskinesia Patients in Taiwan

台灣原發性陣發性動作型運動不良症之PRRT2基因變異性研究

Yu-Tzu Chang[1], Wei-De Lin[2,3], I-Ching Chou[1,4], Chung-Hsing Wang[1,5], Zheng-Nan Chin[1], Fuu-Jen Tsai[1,2,6], Chang-Hai Tsai[1,7]
Department of Pediatrics, China Medical University Hospital[1], Taichung, Taiwan; Department of Medical Research, China Medical University Hospital[2], Taichung, Taiwan; School of Post Baccalaureate Chinese Medicine, China Medical University[3], Taichung, Taiwan; Graduate Institute of Integrated Medicine, College of Chinese Medicine, China Medical University[4], Taichung, Taiwan; Graduate Institute of Clinical Medical Science, China Medical University[5], Taichung, Taiwan; School of Chinese Medicine, College of Chinese Medicine, China Medical University[6], Taichung, Taiwan; College of Health Science, Asia University[7], Taichung, Taiwan
張鈺孜[1]、林瑋德[2,3]、周宜卿[1,4]、王仲興[1,5]、陳震南[1]、蔡輔仁[1,2,6]、蔡長海[1,7]
中國醫藥大學附設醫院小兒科[1]、醫學研究部[2]；中國醫藥大學學士後中醫學系[3]、中西醫結合研究所[4]、醫學科學研究所[5]、中醫學院[6]；亞洲大學健康管理學院[7]

Background: Paroxysmal kinesigenic dyskinesia (PKD, OMIM 128000) is a rare paroxysmal movement disorder and is often misdiagnosed clinically as epilepsy. It is characterized by recurrent, brief attacks of dyskinesia that are triggered by sudden voluntary movement. Mutations in the proline-rich transmembrane protein 2 (PRRT2) have recently been identified in patients with familial PKD. To extend these recent genetic reports, we investigated the frequency and identified of PRRT2 mutations in Taiwanese patients

Methods: A total of eight patients (seven males and one female) from five unrelated families were identified according to PKD criteria. Among them, six patients from three families had familiar PKD, and two were apparently sporadic cases. The complete PRRT2 coding region, including the intron/exon boundaries, were sequenced on PKC patients

Results: Among them, one insertion mutation c.649_650insC (p.P217fsX7) was identified in 3 patients of one family. In this family, all of the affected members carried one mutated allele. Their mean age of the first attack was around early puberty, and the symptoms became remarkable during early adulthood. The father had spontaneous remission at the time they visited us, and the two sons receiving low dose carbamazepine treatment became attack-free without decline in school performance

Conclusions: This study confirms the high sensitivity of PRRT2 for PKD phenotype. Considering the limitation of directed DNA sequencing, it does not exclude the possibility of other mutation types in PRRT2 gene, such as inversions, translocations, mutations within the introns and promoter. Identification of the genes that underlie pathogenesis will enhance diagnosis and clarify the mechanisms of disease progression in PKD. The function of PRRT2 and its role in PKD need to be further investigated

31 Traumatic Injury to the Immature Frontal Lobe: A New Murine Model

兒童額葉腦創傷的新動物模式

Chien-Yi Chen, Donna Ferriero[1], Linda J Noble-Haeusslein[2], Bridgette D Semple[2]
Department of Pediatrics, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan; Departments of Pediatrics[1], Neurological Surgery[2], University of California San Francisco, CA, USA
陳倩儀
國立台灣大學醫學院附設醫院小兒部；加州大學舊金山分校小兒部[1]、神經外科部[2]

Background: Traumatic brain injury in children commonly involves regions of the frontal lobes and is associated with distinct structural and behavioral changes. To the best of our knowledge, there are currently no rodent models of frontal traumatic injury to the developing brain. The aim of this study was to establish a new model of traumatic injury to the frontal lobe of the developing mouse brain.

Methods: Anesthetized male C57Bl/6J mice at postnatal day (p)21, an age approximating a toddler-aged child, received a craniotomy (3.5mm; ~0.5mm anterior to bregma) and controlled cortical impact at 2.5cm/sec (1.73mm depth; 150ms). Sham-operated mice underwent identical surgical procedures excluding the impact. Brains were collected at either 24 h or 7 d after injury for standard histology, immunohistochemistry and TUNEL (n=8/TBI, n=5/sham).

Results: Immediate swelling was evident in the impacted frontal cortex, with moderate-to-severe bleeding in 12% of TBI mice, but no apnea or mortality. All mice showed normal weight gain post-surgery. A necrotic cavity and local inflammatory response were largely confined to the unilateral frontal lobe, dorsal corpus callosum and striatum anterior to Bregma. The lateral ventricles were dilated by 7d post-injury. While abundant cell death(TUNEL, FluoroJadeC) and accumulated beta-amyloid precursor protein were characteristic features of the peri-contusional