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ERRATUM

1. The following abstracts of scientific papers presented at the Fourth Meeting of the European Neurological Society, Barcelona, Spain, June 1994, have been omitted or contain errors in the book of abstracts, Supplement 1 to Volume 241:

CONTROL OF RESPIRATION IN THE LOCKED IN SYNDROME. P Heywood, RS Howard, K Murphy, D Corfield, M Morrel, A Guz. *London, UK*

Respiratory regulation by the CNS involves automatic control from medullary respiratory centres and voluntary control via corticospinal pathways. The «Locked In Syndrome» (LIS) isolates medullary respiratory centres from rostral influences passing in the ventral pons with the corticospinal tracts. Respiratory variables during resting wakefulness were studied in a 44-year-old man with LIS due to bilateral ventral pontine infarction five months post ictus when he was breathing spontaneously via a tracheostomy. Results were compared with data from five tracheotomized control subjects. Variability about the mean in the LIS patient was significantly less than in controls for T1, TE, Vt and $PETCO_2$. Ventilatory sensitivity to CO_2 was 1.7 l/min/mm Hg (NR 1.5–3.8). In the LIS patient the mean resting $PETCO_2$ was 39.0 mm Hg and prolonged apnoea occurred when $PETCO_2$ was < 38.0 mm Hg. Although completely unable to regulate respiratory muscle activity voluntarily, when he was amused he laughed and altered his respiratory rate and volume. These findings confirm that 'isolated' human respiratory centres produce a very regular respiratory rhythm upon which variability is superimposed by separate descending systems mediating volitional and emotional control. They also show that medullary respiratory centres retain normal sensitivity to CO_2 and that resting $PETCO_2$ is held extremely close to the apnoeic threshold.

FROM SCHIZENCEPHALY TO LOCALIZED CORTICAL DYSPLASIA: A CONTINUUM OF DEVELOPMENTAL, PROBABLY MALACIC, ANOMALIES. G Sebire, B Husson, M Zerah, A Dusser, O Boespflug, M Tardieu, P Landrieu. *Bicêtre, France*

Schizencephaly was primarily described by neuropathologists as large cleft throughout a cerebral hemisphere. With the era of cerebral imaging, the distinction between a typical cleft and an abnormal sulcus inside a modified cortex became less evident. We reviewed 20 cases including 1) unusually large or deep sulcus (or cleft); 2) presence of gray matter bordering the defect; 3) one or both of the following characteristics: sulcus in a clearly abnormal location; bordering, gray matter clearly abnormal. Various clinical presentations were observed: congenital encephalopathy, infantile hemiplegia or diplegia, epilepsy and even casual discovery. All cases were sporadic. Abnormal, pregnancies were frequent and included 2 severe accidents at 4 and 5 months. Three morphological groups could be delineated: A) "typical" cleft, not corresponding to any physiological sulcus: 9 cases, 5 of them had bilateral lesions; B) primary sulcus oversized and bordered by dysplastic cortex: 7 cases, 9 of them corresponding to the syndrome of perisylvian or central macrogyria, either uni- or bi-lateral; C) in 4 cases, the cleft looked like an oversized secondary sulcus, the neighboring cortex displaying anomalies such as small cavities, ulegyric-like rearrangement or non specific dysplasia. Neuropathology could be analyzed in 1 typical schizencephaly case. The cleft was not a parenchymal defect, but a mesenchymal structure due to the locally defective expansion of the cerebral mantel. Bordering gray matter was not clusters of neurons migrated in heterotopic position, but microgyric modifications of a post migratory cortex (> 12 weeks of gestation). Late, including postnatal pathological events suggested a developmental vasculopathy. We conclude that there is no physiopathological discontinuity between the different types of cleft. All of them are abnormal sulci, formed at various steps of the gyration, following the primary cortical organisation. Morphology of the abnormal sulcus and of neighboring cortex are depending both on the timing and on the severity of the malacic process.

MULTIFOCAL MOTOR NEUROPATHY WITH PERSISTENT CONDUCTION BLOCK: RESPONSE TO TREATMENT WITH I.V. INJECTIONS OF IMMUNOGLOBULINS IN 21 PATIENTS. D Adams, JM Léger, P Bouche, G Said. *Paris, France*

Twenty six patients with multifocal motor neuropathy (MMN) and persistent conduction blocks were followed during a 3- to 5-year period. Five with a slight and stable motor disability received no treatment. The others were treated with intravenous injections of human immune globulins (i.v. Ig) at doses of 2 g/kg over a period of 5 days every month during five months then every three months. Muscle strength was serially assessed in 40 skeletal limb muscles and graded according to the Medical Research Council (MRC) Scale. Three patients did not respond at all to i.v. Ig. Eleven patients transiently improved after the first three series of injections but did not respond to further injections. Seven patients had a good response which necessitated maintenance course of i.v. Ig every 2 to 3 months. The response to treatment was not correlated with the level of anti-GM 1 antibodies. The electrophysiological studied did not show significant variation of the amount of conduction blocks or motor distal amplitude even in the patients who had the best response to treatment. The only predictive factor of i.v. Ig response was the degree of distal muscle atrophy. Patients with no or slight distal amyotrophy had the best clinical response to i.v. Ig treatment. We conclude that long term treatment with i.v. Ig seems to be useful only in one third of the patients with MMN.

MIGRAINE AND TENSION TYPE HEADACHES. P Monteiro, R Maio. *Porto, Portugal*

In order to establish a score for classification of primary headaches based on amount of points rather than the criteria of the International Headache Society (IHS), we performed a stepwise logistic regression analysis of a 2008 population-based study at Oporto (Portugal) in May 1992. In our series we obtained a prevalence of 8.8% for migraine, 62.5% for tension-type headaches, for combined migraine and tension headache 12.1%, and 5.3% for other headaches. Based on the results of stepwise logistic regression, we built a score ranging from zero to eleven points (unilateral = 1, pulsating = 1, aggravated by exercise = 1, severe = 1, moderate = 1/2, nausea = 3, photophobia = 2, phonophobia = 1, duration superior to 4 hours = 1/2 and frequency < 120 per year = 1/2). Scores from 0 to 4 are tension-type headaches and scores from 6 to 11 are migraine; scores 4.5, 5 and 5.5 are intermediate forms corresponding to a combination of two types of primary headaches. We define four levels of disability 0 (none), I (mild), II (moderate) and III (severe) based on severity and frequency. Different levels of disability imply different therapeutic modalities (0 and I – symptomatic therapy and II, III – prophylactic therapy). In levels II and III they are: 97.6% of chronic-TTH, 52.9% of migraine with aura, 39.6% of migraine without aura and 17.4% of episodic-TTH. Using our score we found 92% in concordance with IHS classification in primary headaches.

A BRAIN MRI STUDY OF THE SHORT-TERM EFFECTS OF I.V. METHYLPREDNISOLONE AND ORAL PREDNISONE IN THE ACUTE RELAPSES OF MULTIPLE SCLEROSIS. G Comi, M Filippi, V Martinelli, M Rovaris, S Mammi, P Reganati, B Colombo, N Canal. *Milan, Italy*

This study was undertaken to evaluate the efficacy of two different steroid schedules on brain MRI and clinical findings of patients with multiple sclerosis (MS) who had at least 4 MRI enhancing lesions during an acute relapse. Eighteen clinically definite MS patients at the onset of a relapse received either i.v. methylprednisolone (MP; 1 g daily for 5 days; 10 patients) or oral prednisone (P, 1 mg/kg/day tapering in 3 weeks; 8 patients). Brain MRI (T2WI and T1WI after gadolinium-DTPA injection) were performed at the onset, after 1 and 2 weeks and after 1 and 3 months. One/10 of patients treated with MP and 6/8 of those treated with P had further clinical relapses during the follow up ($P < 0.02$). At basal evaluation the number of enhancing lesions was not significantly different in the 2 groups. After 1 week a significant greater reduction of such lesions was observed in the MP group compared to the other ($P < 0.05$). Enhancing lesions completely disappeared in 9/10 patients treated with P ($P < 0.01$). From the 2-week evaluation this difference was not ever found. Our MRI data indicate that MP has a stronger affect than P on MS activity during an acute clinical relapse. MP seems also to be more effective in preventing early new relapses.

IN VITRO STUDIES OF OLIGODENTROCYTES DERIVED FROM ADULT HUMAN WHITE MATTER. NJ Scolding, J Susman, DAS Compston. *Cambridge, UK*

In multiple sclerosis, oligodendrocytes and the myelin sheats they synthesise and maintain are selectively and often repeatedly damaged, ultimately

resulting in permanently demyelinated lesions. Recent findings indicate that the adult CNS does, however, possess an inherent capacity for repair, that oligodendrocyte precursor-like cells are abundant in acute lesions, and that the early stages of remyelination are initially apparent. Understanding the developmental biology of oligodendrocytes may not only yield helpful insights into the failure of repair in acutely demyelinated areas, but also suggest strategies by which remyelination might be promoted or indeed augmented by glial transplantation.

Much information is already available concerning the developmental neurobiology of rodent oligodendrocytes but differences between rat and human cells are apparent. Recently, however, techniques for culturing human glia have become available. We have studied cells derived from the white matter of adult patients (age 16–40) undergoing a variety of neurosurgical procedures for non-malignant conditions, mostly epilepsy. Oligodendrocytes are readily identifiable and can be maintained in vitro; in addition, novel preliminary evidence has also emerged of the presence in low numbers (approximately 1% of oligodendroglial-lineage cells) of a precursor cell which appears to be the human counterpart of the well-characterised rat bipotential oligodendrocyte progenitor.

2. The list of authors of the following papers should be corrected as follows:

THE MANAGEMENT AND OUTCOME OF SEVERE GUILLAIN BARRÉ SYNDROME. KKP Ng, RS Howard, DR Fish, NP Hirsch, NMF Murray, DH Miller. *London, UK*

INTERLEUKIN LEVELS IN THE CEREBROSPINAL FLUID OF STROKE PATIENTS. R Gilad, Y Eshel, Y Lampl, V Barak, I Sarova-Ponchas, *Tel Aviv, Israel*

DISTAL MYOPATHY OF MIYOSHI: A REPORT OF 4 PATIENTS. J Gamez, M Fernandez, C Navarro, C Cervera, NL Viguera, F Crespo, A Codina. *Barcelona, Spain*

OPTIC NEURITIS: CORRELATION OF VEP, PERIMETRY AND MRI FINDINGS. A Lugaresi, A Tartaro, P d'Aurelio, L Lobefalo, A Thomas, G Malatesta, PE Gallenga, D Gambi. *Chieti, Italy*

ODOUR IDENTIFICATION IN PARKINSON'S DISEASE. LC Potagas, M Ziegler, N Bathien, P Rondot. *Paris, France & Athen, Greece*

SHORT-TERM EFFECTS OF 2-CHLORODEOXYADENOSINE (2-CDA) IN REMITTING-RELAPSING MULTIPLE SCLEROSIS (MS). Z Stelmasiak, J Solski, J Nowicki, B Jakubowska, M Ryba, P Grieb. *Lublin & Warsaw, Poland*