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**Repetitive transcranial magnetic stimulation modifies habituation of visual evoked potentials.** A. FUMAL, V. BOHOTIN, C. BOHOTIN, P. GERARD, M. VANDENHEEDE, A. MAERTENS DE NOORDHOUT, J. SCHOENEN (Dept. of Neurology, University of Liège, Belgium).

*Background* : To study the excitability of occipital cortex in healthy volunteers and migraineurs by recording pattern-reversal visual evoked potentials (PR-VEP) before and after repetitive transcranial magnetic stimulation (rTMS).

*Methods* : We performed rTMS of the visual cortex in healthy volunteers (n= 12) and in patients suffering from migraine (n = 8) with a figure-of-eight magnetic coil placed over the occipital scalp. We delivered 900 pulses at two different frequencies : 1 Hz (15 minutes) and 10 Hz (18 trains of 5 seconds). Before and after rTMS the PR-VEPs were sequentially averaged in blocks of 100 responses during 3 minutes of uninterrupted stimulation at 3.1 Hz and analyzed in terms of peak-to-peak amplitude of N1-P1 and P1-N2 peaks.

*Results* : After the 1 Hz rTMS there was a significant reduction of the habituation or even a potentiation in healthy volunteers, but the potentiation observed in migraineurs was not modified. There was no significant change of habituation in healthy volunteers after 10 Hz rTMS, but in migraineurs we observed a reduction of potentiation or even appearance of habituation.

*Conclusion* : The decrease of cortical excitability induced by 1 Hz rTMS in normal volunteers is associated with loss of habituation or even potentiation of PR-VEPs. The increase of cortical excitability which follows the 10 Hz rTMS produces in migraineurs less marked potentiation or even habituation. Taken together these findings suggest that the deficient habituation of EPs found interictally in migraine is due to a reduced preactivation level of sensory cortices.

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**Hyperthyroidism with overproduction of the factor VIII procoagulant protein as a predisposing factor for cerebral venous thrombosis.** J. Maes<sup>1</sup>, K. Jochmans<sup>2</sup>, B. Velkeniers<sup>3</sup>, T. Stadnik<sup>4</sup>, A. Michotte<sup>1</sup>, G. Ebinger<sup>1</sup> (<sup>1</sup>Department of Neurology, AZ-VUB, Brussels, Belgium, <sup>2</sup>Department of Hemostasis and Thrombosis, AZ-VUB, Brussels, Belgium, <sup>3</sup>Department of Endocrinology, AZ-VUB, Brussels, Belgium, <sup>4</sup>Department of Radiology, AZ-VUB, Brussels, Belgium).

We report the case of a 39-year-old woman presenting with a brief history of convulsions, preceded by behavioural disturbances. Brain MRI revealed a thrombosis of the left transverse sinus. Further testing demonstrated hyperthyroidism. An increased factor VIII (FVIII) was the only abnormality in thrombophilic tests. Only a few similar cases have been described. It was shown recently that an increase of FVIII as high as 150% is responsible for an adjusted relative risk for venous thrombosis of almost 5. Our patient was treated with radioactive iodine and anticoagulants. She subsequently developed a hypothyroidism that needed substitution. FVIII levels returned to normal after correction of thyroid function and treatment with anticoagulants was subsequently stopped. This case suggests that hyperthyroidism by increasing production of FVIII might be a predisposing factor for the development of a cerebral venous thrombosis.

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**Abdominal wall weakness and lumboabdominal pain revealing neuroborreliosis : a report of three cases.** E. MORMONT, W. ESSELINCKX, TH. DE RONDE, PH. HANSON, TH. DELTOMBE, P. LALOUX (Dpts. of Neurology, Rheumatology, Gastroenterology, and Physical Medicine and Rehabilitation, Cliniques universitaires UCL de Mont-Godinne, Yvoir, Belgium).

We report on three cases of thoracic radiculoneuropathy due to neuroborreliosis. All three patients had low back and abdominal pain and two patients had marked abdominal wall paresis. Sensation was impaired in the T10, T11, and L1 dermatomes. Superficial abdominal reflexes were absent. EMG confirmed a motor involvement of the lower thoracic

roots and CSF analysis revealed a lymphocytic meningitis in all patients. Antibodies against *Borrelia burgdorferi* were present both in the serum and CSF. A favourable outcome was obtained in all three patients with appropriate intravenous antibiotherapy. The differential diagnosis of this misleading presentation includes diabetic thoracic polyradiculoneuropathy, disc herniation, herpes zoster, and syringomyelia. In conclusion, these three cases demonstrate that Lyme disease is also a possible mechanism of thoracic radiculoneuropathy, thereby requiring appropriate investigations.

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**Intracranial vertebral artery dissection revealed by a subarachnoid hemorrhage and successfully treated by coil embolisation.** S. DORBAN, A. PEETERS, P. GOFFETTE, C. RAFTOPOULOS (Depts. of Neurology, Medical Imaging and Neurosurgery, Cliniques Universitaires Saint Luc, Brussel, Belgium).

We report the case of a 44 year-old woman who presented with severe occipital headaches rapidly followed by a coma with signs of decerebration and oculomotor disturbances. The cranial CT revealed a hemorrhage in the third and fourth ventricles. A subarachnoid hemorrhage (SAH) was confirmed by MRI and lumbar puncture. Furthermore, the MR angiography showed an aneurysmal dilatation of the left intracranial vertebral artery (VA). On conventional angiography, this lesion corresponded to a VA dissection, located distally to the origin of the left posterior inferior cerebellar artery (PICA). A therapeutic occlusion of the left VA dissected segment was performed by coil embolisation, with preservation of the ipsilateral PICA. Two days later, a second MRI revealed large bifrontal infarctions and a small right insular ischemic lesion probably due to a transient cerebral vasospasm. In addition, multiple cerebellar and occipital ischemic lesions were observed, as a possible embolic complication of the endovascular procedure. The long-term clinical outcome was favourable with a normal neurological examination at 6 months. In conclusion, the intracranial VA dissection is a rare cause of SAH. An early neurosurgical or endovascular treatment is justified by the high risk of short-term rebleeding with a high mortality rate. There is actually no consensus between these therapeutical approaches.

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**Neurological involvement in a case of hypophosphatemia.** A. JANSEN, B. VELKENIERS, G. EBINGER (Department of Neurology, VUB, Jette).

A 69-year-old woman was admitted to the hospital, for dyspnea, confusion, and weakness. Three days after admission she developed respiratory failure, mental obtundation, and rapidly progressive quadriparesis. She was found to have severe hypophosphatemia due to internal redistribution and decreased intestinal absorption of phosphate. Administration of intravenous phosphate supplements resulted in rapid clinical improvement, with subsequent normalization of serum phosphate levels. Hypophosphatemia occasionally causes neurological manifestations. Presenting symptoms and pathophysiological mechanisms are discussed.

In conclusion, hypophosphatemia should be considered in patients with encephalopathy and/or rapidly progressive weakness.

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**Neuropsychological assessment of Parkinsonpatients 1 year after bilateral subthalamic stimulation.** K. PORKE, B. NUTTIN, R. DOM (Dept. Of Neurology and Neurosurgery, K.U.Leuven, Belgium).

The positive effects of chronic bilateral stimulation of the subthalamic nucleus (STN) in the treatment of Parkinsonian patients with severe levodopa-related motor adverse events has been repeatedly shown. Symptoms such as rigidity, tremor and akinesia decrease. Also walking- and speakingproblems diminish to a certain extent. However, few studies have examined the effects of chronic STN stimulation on cognitive functions. The aim of our study is to investigate whether there are some cognitive changes one year after STN.

21 patients are examined with an extensive standard battery of neuropsychological tests, before and one year after surgery. General cognitive functioning, memory, attention, frontal executive functions, visuospatial perception, visuomotor skills and degree of depression are measured.

At one year after surgery, most task measures did not change. The encoding process of verbal information was slightly impaired, but delayed recall remained the same. There was a significant decreased performance on word fluency and most of all in lexical word fluency tasks. Also a slight impairment was observed in frontal executive functioning and visuomotor skills. On the Beck Depression Inventory patients scored less depressed.

Our study results demonstrate that surgery may have a slight negative impact on cognitive functioning. Further research will evaluate a larger group of patients over a longer period.

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**Creatine supplementation in patients with Huntington's chorea.** P. VERBESSEM, P. HESPEL, J. LEMIERE, B. OP'T EIJNDE, L. VANHEES, M. VAN LEEMPUTTE, R. DOM (Depts. of Neurosciences and Psychiatry, of Kinesiology and of Rehabilitation Sciences, K.U. Leuven, Belgium).

*Background* : This study investigated the potential of oral creatine supplementation as a therapeutic strategy in patients with Huntington's disease (HD).

*Methods* : A double-blind randomized placebo-controlled study was performed over a 1-year period. Forty-one patients in the initial stage of the disease participated. At the start of the study and after 6 and 12 months of supplementation cognitive function, functional capacity and general motor function were scored by means of the Unified Huntington's Disease Rating Scale (UHDRS). In addition muscle strength of the elbow-flexors was assessed by an isokinetic dynamometer and cardiorespiratory fitness was evaluated in a maximal incremental exercise test (10 + 10 watt/min) on a bicycle ergometer. After the baseline measurements the subjects were assigned to either a creatine (CR, n = 26 ; 5 g/day), or a placebo group (PL, n = 15).

*Results* : In the total group of subjects (n = 41) scores on the functional checklist decreased from  $18 \pm 0.8$  to  $16 \pm 0.9$  from the start to the end of the study ( $p < 0.001$ ). Maximal static torque of the elbow-flexors decreased by 10% ( $p < 0.001$ ). Peak oxygen uptake after one year follow-up ( $1630 \pm 83$  ml/min) was lower compared with baseline measurements ( $1730 \pm 91$  ml/min ;  $p < 0.01$ ). There were no significant changes from baseline to the end of the study in cognitive function and general motor function. All of the above measurements at any time of the study were independent of the treatment received.

*Conclusions* : Creatine supplementation (5 g/day) does not improve the neuromuscular, cognitive and functional status in patients in the early stages of HD.

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