Peripheral nerve disorders
Spinal cord and root disorders
Neuro-ophthalmology, Neuro-otology

P 1088

An electrodiagnostic study on efficacy of intracarpal steroid injection in carpal tunnel syndrome (CTS)

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Introduction Carpal tunnel syndrome (CTS) is one of the most common entrapment neuropathies in which the median nerve is involved in the carpal tunnel. Intracarpal steroid injection into the carpal tunnel was evaluated as a therapeutic method in this study.

Method In a prospective study 30 patients including 56 hands with electrodiagnostic criteria of CTS of mild and moderate (severe class excluded) were treated by injecting 20 mg triamcinolone H.A. into the involved canal and observed for a period of 6 months. Patients were visited and electrodiagnostically tested 30–40 days after injection, in case of no improvement in electrodiagnostic criteria, the injection was repeated and patients were re-examined 30–45 days later. In case of any disease progress despite therapy patients were referred for surgery.
Results 22 of 30 patients were 30–60 years old and 23 of them were female. In 26 CTS patients CTS was bilateral. Before therapy, 17.9% of patients were in the mild class and 82.9% in the moderate class. After therapy, 30% of patients had mild and 66% had moderate CTS, while 4% showed criteria of complete resolution.
Conclusion Ephaptic transmission in ischemic entrapped median nerve is a definition for CTS symptoms that can be ameliorated by anti-inflammatory effects of steroids, which improve the integrity of neural membranes. In view of the convenience of steroid injection with no systemic side effects apart from contraindication in diabetics, we recommend this method of therapy for mild and moderate CTS cases.

P 1089

Central motor conduction time in alcoholic axonal neuropathies

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Objective Alcoholic axonal neuropathies (AAN) are frequently associated with signs of central nervous system (CNS) impairment. Studies estimating the CNS functional state in neuropathies are not numerous. The goal of the study was to establish the diagnostic value of motor evoked potentials (MEP) in AAN.
Method 62 patients (55 males and 7 females) examined with neuropathies attributed to daily excessive consumption of alcohol (greater than 100 ml ethanol) for 3 years or more. The routine nerve conduction studies were completed by MEP studies.
Results Nerve conduction studies revealed signs of axonal neuropathy in 54 patients (87.1% cases). In the other 8 patients signs of autonomic fibres affection prevailed in the picture of neuropathy. Clinical signs of CNS impairment were detected in 37 patients (59.7% cases). They manifested by cerebellar, pyramidal signs and pseudobulbar syndrome. The central motor conduction time (CMCT) was increased in 39 patients (62.9% cases), being also elevated in cases without clinical signs of CNS disturbance. The mean CMCT value was 9.8±1.2 ms at recording from m. abductor pollicis brevis and 22.3±1.9 ms – from m. abductor hallucis. The amplitude of MEP varied considerably, being significantly lower than in the control group. In 5 cases MEPs after transcortical magnetic stimulation could not be obtained.
Conclusion MEP proved to be a sensitive method to establish the implication of the CNS motor structures in the pathological process in AAN. CMCT is frequently increased even when clinical signs of SNC affection are not present.

P 1090

Indications of lower extremity nerve grafting

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Introduction For a variety of reasons lower extremity nerve injuries have poor history of successful repair and are generally neglected despite studies of repair of nerve injuries of the upper extremity. The goal of our study was to determine the direct indications and terms of surgery at lower limbs nerve injury.
Method 65 patients with nerve lesions of the leg were evaluated at an average follow-up of 4 years. To assess the return of motor, sensory and trophic function grading systems were used. From electrophysiological methods EMG examination was carried out.
Results 17 cases had good recovery of leg function, 21 satisfying, 15 useful, and 12 poor restoration. All good results were connected with early surgical intervention – up to 2 months, as well as with young age. All cases (42) with severe trophic disturbances like deep ulcers and erosions had functional recovery. EMG examination revealed increased motor and sensory nerve conduction velocity, amplitude of compound muscle and sensory nerve action potentials.
Conclusion Even partial restoration of trophic disturbances in spite of presence of deep paresis or hypoesthesia, becomes the main determinative factor in providing gait function. Thus presence of severe vegetative symptoms is the indication of surgical intervention in traumatic damage of lower limb nerves. Early surgical treatment is most likely to have good outcome. Although the results of lower limb nerve injuries typically have been dismal, our study refutes this opinion and we believe that the repair of lower extremity nerve injuries will be proven to be successful and worthwhile.

P 1091

The pronator teres syndrome in clinical practice

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Introduction The pronator teres syndrome (PTS) is a rare form of entrapment of nervus medianus between two heads of the musculus pronator teres.
Method In all patients the strength of musculus flexor hallucis longus, m. flexor digitorum profundus, m. pronator quadratus was tested as well as their sensory impairment. Needle EMG, motor nerve conduction and CMAP amplitude, as well as sensory nerve conduction with ring electrodes and SNAP of medianus were performed.
Results 6 patients were evaluated. The first 3 cases had normal EMG findings. The other 3 had sensory impairments and signs of heavy motor lesions. In 2 cases showed acute lesions with muscle fibrillations. In the third chronic case a mild lesion of flexor digitorum sublimis was detected. Motor nerve conduction was normal, CMAP amplitude was decreased. All 3 cases with sensory impairment had decreased or absent SNAP in the same fingers in which sensory impairment was present. In two acute cases full recovery was achieved, accompanied by increase of CMAP amplitude and SNAP.
Conclusion We met PTS more often than isolated anterior interosseous syndrome. Needle EMG should be done, except in the innervation area of nervus interosseous anterior in the abductor pollicis brevis and possibly flexor digitorum sublimis. Decreased CAMP is increased in course of recovery. Sensory loss is not typical and covers only some parts, exceptionally complete innervation area of medianus. Increase of SNAP amplitude follows the clinical recovery. The initial milder PTS could be without evident neurological disturbance or by EMG proven lesion.

P 1092

Bell's palsy over a four-year period: The unexpectedly high incidence during the NATO strike attacks against Yugoslavia

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Introduction Bell's palsy is a lower motor neuron, ipsilateral facial paresis affecting roughly 20 people per 100,000 per year. The origin is still unclear. The aim of this study was to investigate the incidence of Bell's palsy over a four-year period, including the time of war.
Method In a retrospective study from March 24th 1998 to March 23rd 2002, 406 Bell's palsy patients (142 male and 251 female) were analysed (in an area of 350,000 inhabitants). The total time period was split into16 intervals of 3 months each, including the air strike period, and those periods coincided with seasons (spring, summer, autumn and winter). Data were analysed according to age, gender and seasonal distribution of cases.
Results An unexpectedly high incidence of Bell's palsy was found during the NATO air strike period (77 patients or 18.96% of all cases, $p<0.001$). Women were more sensitive to the appearance of Bell's palsy than men (65.27% vs. 34.73%, $p<0.001$) in the total group. There was no difference in sex distribution between different seasonal periods.
Conclusion We suggest unusual life conditions during the air strikes and high-intensity stress as factors predisposing possible viral infection may be included in Bell's palsy appearance.

P 1093

Unusual multifocal motor neuropathy associated with lymphoma - case report

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Introduction The association of motor neurone disorders with lymphoproliferative diseases is well documented in literature. Subacute motor neuropathy is usually associated with lymphoma, although this association is not fully understood.
Case description A right-handed 60-years-old male presented with five-months history of frequently remitting pure lower motor neurone (LMN) signs. There was no evidence of bulbar, sensory or mental involvement. The past medical history was negative. Six months after the onset of signs, the disease progressed rapidly and flaccid proximal paresis occurred in the left limbs. There was no evidence of infection, immunodeficiency or paraproteinemia in serum and CSF examinations. We found no abnormality in laboratory screening including autoantibodies, viral and HIV infection tests. NCT studies identified multifocal motor neuropathy (MMN) with conduction block. The patient's clinical status improved shortly after human immunoglobulin administration and few days later deteriorated again without any response to further immunotherapy. At that time the clinical examination also showed enlargment of the left submandibular lymph nodes. Ultrasonography of the neck and supraclavicular regions revealed many augmented lymph nodes bilaterally. CT of mediastinum and ultrasonography of abdominal cavity were normal. The histopathological examination of the submandibular lymph nodes identified malignant non-Hodgkin's B-cell lymphoma (lymphocyticum diffusum). Immunophenotypic study of bone marrow identified a "mantle cell lymphoma" with co-expression of CD19/CD5 and lack of CD23 lymphocytes. After diagnosis local radiotherapy was introduced. Currently there is no improvement in the patient's neurological status and he is under continuous observation. Interestingly, the neurological presentation preceded signs of lymphoma.

P 1094

The significance of anti-gm1 antibodies in patients with Guillain-Barré syndrome

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Introduction Guillain-Barré syndrome (GBS) is an autoimmune polyradiculo-neuropathy which can be associated with significantly elevated level of serum anti-GM1 antibodies.
Methods We investigated 48 patients with GBS, 21 males and 27 females. The sera of all investigated patients were tested for the presence of anti-GM1 antibodies using an enzyme-linked immunosorbent assay (ELISA). The sera of 22 patients were examined for the presence of antibodies to 63 kDa flagellar protein from Campylobacter jejuni (Cj) serotype O.19 using ELISA and Western blot.
Results Anti-GM1 antibodies were detected in 58.3% in IgG and in 68.7% of patients in IgM class. Gastrointestinal infection was present in 42.3% of anti-GM1 positive patients. Anti-Cj antibodies were detected in 59% of these patients. In 81.3% the presence of anti-GM1 antibodies was associated with the elevated antibody titre to Cj ($p<0.01$). Patients with positive serology to GM1 were more severely affected than sero-negative patients ($p<0.05$). Electrophysiologically, 57.6% of anti-GM1 positive patients had sensory-motor neuropathy. In 93.3% the illness was due to axonal degeneration and only in 6.7% due to demyelination ($p<0.05$). Patients with negative GM1 serology had similar distribution of the neuropathy type. 73.4% of sero-positive patients had residual weakness after the treatment, while 84.8% of sero-negative patients recovered completely ($p<0.01$).
Conclusion We conclude that anti-GM1 antibodies were not only responsible for the pathogenesis of the pure motor, but are also detected in patients with sensory-motor neuropathy. Only association of anti-GM1 and anti-Cj antibodies was regarded as a bad prognostic factor in our group of patients.

P 1095

The significance of anti-gm1 and anti-sulphatide antibodies in patients with polyneuropathies

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Introduction Since recent studies showed great differences in the presence of anti-GM1 and anti-sulphatide antibodies in patients with polyneuropathies, we wanted to determine the presence of these antibodies in our patients.
Methods We analysed 72 patients, 46 male and 26 female, with polyneuropathy. Pre-treatment serum samples were tested using an enzyme-linked immunosorbent assay for the presence of anti-GM1 and anti-sulphatide antibodies.
Results Anti-GM1 antibodies were present in 40.3% in IgG and in 37.5% in IgM class. Sero-positive patients had earlier onset of the disease. In 57.1% the disease started in the 4th or 5th decade ($p<0.05$). These patients had more severe neurological disability in comparison to sero-negative patients ($p<0.05$).

There was a correlation between the presence of anti-GM1 antibodies and axonal form of the disease, which was found in 71% of anti-GM1 positive patients ($p<0.05$). The presence of anti-GM1 antibodies was an indicator of the longer duration of the disease and worse outcome. No improvement was registered in 72.5% of sero-positive patients, while 56% of sero-negative patients had significant clinical improvement after the treatment ($p<0.01$). Anti-sulphatide antibodies were found in 58.3% of patients with sensory neuropathy, who also had a longer course of the disease and worse outcome, which was significantly different in comparison to sero-negative patients ($p<0.05$).
Conclusion We found anti-GM1 antibodies mostly in younger patients with greater disability and axonal neuropathy. The presence of anti-sulphatide antibodies only in patients with sensory neuropathy confirms their role in the pathogenesis of sensory nerve disorders.

P 1096

Neurophysiological determination of the carpal tunnel syndrome in patients with chronic demyelinating polyneuropathy

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Introduction The goal of our study was to determinate a specific neurophysiological method to confirm the clinical diagnosis of carpal tunnel syndrome (CTS) in patients affected with chronic inflammatory demyelinating polyneuropathy (CIDP), and to investigate the cause of the disproportionate distal motor latency of the median nerve in patients affected with CIDP with anti-MAG antibody (MAG-PN).
Methods We evaluated 4 groups of patients: a) 10 CIDP (20 hands), mean age 58 years, range 30–67, without clinical signs of CTS; b) 8 MAG-PN (16 hands), mean age 52, range 45–65, without clinical signs of CTS; c) 8 CIDP (16 hands), mean age 41, range 34–60, with clinical signs of CTS (CTS CIDP); d) 22 healthy controls, mean age 40, range 22–65. The diagnosis of CTS is based on clinical criteria. The following electrophysiological parameters were evaluated: a) antidromic sensory conduction velocity of median and ulnar nerve between wrist and, respectively, second and fifth finger. b) distal motor latency (DML) to abductor pollicis brevis and abductor digiti minimi muscles and the elbow to wrist motor conduction velocity of median and ulnar nerve. c) Terminal Latency Index (TLI) of median and ulnar nerve; d) Motor latency of the median nerve to second lumbar and of ulnar nerve to second interosseous muscle and their difference (L-I DIFF).
Results L-I DIFF was the most sensitive electrophysiological parameter in order to detect presence of CTS in patients with CIDP. Moreover we believe that median suffering at the wrist is a distinctive electrophysiological feature in MAG-PN.

P 1097

Effect of sodium valproate on electrophysiological parameters and Mc-Gill pain questionnaire in patients of diabetic painful neuropathy

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Introduction Sodium valproate is one of the first line anticonvulsants, which is very effective and well tolerated. As the available treatment modalities of painful neuropathies are not completely satisfactory and the various anticonvulsants play a definitive role in the treatment hence we sought to study the effect of sodium valproate in this indication.
Method The study was performed on 60 patients of type 2 diabetes suffering of painful neuropathy. The design of the study was double blind, placebo controlled. The patients were divided into 2 groups, patients of group A received 1200mg of sodium valproate daily in divided doses, and group B received similar type of two tablets of placebo for the duration of 1 month. Each patient was assessed by clinical examination; pain score, electrophysiological examination including motor and sensory nerve conduction velocity, amplitude and H-reflex and McGill pain questionnaire initially and at the end of the 1 month time period.
Results The present study was performed on 52 patients. In our study motor and sensory conduction including H-reflex were deranged in the beginning of the study in diabetic patients of both groups and there was no improvement in electrophysiological parameters of patients at the end of the, except mild improvement in distal latency of Tibial nerve. Significant improvement in pain score was noticed in patients receiving sodium valproate in comparison to patients receiving placebo at end of the 1-month treatment period ($p<0.05$).
Conclusions There is no improvement in electrophysiological parameters by sodium valproate in diabetic painful neuropathy but the treatment provides magnificent subjective improvement in painful diabetic neuropathy.

P 1098

Axonal multifocal motor neuropathy

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Introduction Multifocal motor neuropathy (MMN) is a demyelinating neuropathy in which conduction blocks (CB) are considered mandatory for the diagnosis of the disease.
Method We reviewed data from patients presenting to our hospital with: pure motor neuropathy; asymmetric weakness with individual peripheral nerve distribution; no signs of upper motor neuron involvement; electrodiagnostic testing that failed to show CB or any other demyelinating features.
Results We identified 2 male patients. Pat.1: 70 years old with 3 years duration of illness presenting with asymmetric weakness in the hands and – to a lesser extent – proximal arms with right predominance and slight muscular hypotrophy. No sensory signs or upper motor neuron involvement, normal cervical spine MRI, no anti-GM1, anti-MAG, anti-sulfatide antibodies, no CB or other features of demyelination were detected. He was treated with IVIg without improvement.
Pat.2: 18 y.o. with 2 years duration of illness presenting with weakness and muscular hypotrophy in the right hand and forearm and with minimal involvement of the left one. No sensory or upper motor neuron signs, normal cervical spine MRI (performed in hyperflexion too), no anti-GM1 antibodies, no CB or demyelinating features were detected. He was treated with IVIg with clinical and EMG improvement.
Conclusion Our data confirm other previous observations that patients with clinical features of MMN without evidence of demyelination and GM1 antibodies should receive an empiric treatment with immune modulating therapy. The lack of demyelinating features could be due to inability to find focal lesions in difficult testing sites or Multifocal Axonal Motor Neuropathy could represent a primarily axonal process.

P 1099

The aetiology of acute peripheral facial nerve paralysis in diabetic patients: is it also viral?

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Objective The goal of this study was to examine the aetiology of acute peripheral facial paralysis (APFP) in diabetic patients.
Introduction There is a lot of recent evidence suggesting that reactivation of latent herpes simplex virus (HSV) or varicella-zoster virus (VZV) infection of the geniculate ganglia is the main cause of APFP (Bell's palsy). Whether diabetes mellitus makes the nerve more vulnerable to virus infection or it is the vascular theory that explains the increased prevalence of DM among patients with APFP is not known yet.
Method 40 diabetic patients with APFP were enrolled in the study (23 males and 17 females, 54.42+8.67 years). Another 40 non-diabetic patients were enrolled as a control group. Paired sera were obtained from all patients in both groups within the 4th day after disease detection and 2 weeks later. We measured IgM and IgG antibodies to HSV and VZV in these sera by ELISA. Demonstration of IgM antibodies or a 4-fold increase in IgG antibody titer were considered positive evidence for the infection of either of these viruses.
Results There was no significant difference between diabetic and non-diabetic patients in sero-positivity to VZV (35% vs. 25%, P>0.05) and HSV (57.5% vs. 37.5%, p>0.05). There was no significant difference between the diabetic and non-diabetic patients for the sero-positivity for both viruses (72.5% vs. 82.5%, p>0.05)
Conclusion APFP in diabetic patients is mostly due to reactivation of either HSV or VZV infection.

P 1100

Painful familial amyloid polyneuropathy type I associated with permanent pacemakers for sick sinus syndrome

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Introduction This paper reports a family in Russia with familial amyloid polyneuropathy type I (FAP type I) with aggressive painful sensorimotor syndrome and permanent pacemakers for sick sinus syndrome.
Method The investigated family had 4 members of two generations (3 brothers and the mother's sister). Other causes of neuropathy were excluded. Sural biopsy and electrophysiological studies were carried out on all patients. Also all 4 patients had implantations of permanent DDD pacemakers for bradyarrhythmias.
Results Sural biopsy in all patients showed positive Congo red staining. Electrophysiological studies revealed mixed axonal and demyelinating polyneuropathy.
Conclusion FAP type I is a rare disease characterized by sensorimotor polyneuropathy, autonomic nervous system, and cardiac conduction system dysfunction. The initial symptoms were cardiac, such as sick sinus syndrome with the sensory loss of lower extremities, painful constant acroparesthesias and joint discomfort. The age of onset was about 1 to 2 decades. As the disease progressed, the upper extremities and motor ability were also involved. To our knowledge, this is the first human report of the painful FAP type I polyneuropathy and pacemaker implantation.

P 1101

Polyneuropathy as an adverse reaction to alprazolam – first reported case

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Introduction Polyneuropathy is often a drug-induced disease. Our intention is to present the first case of polyneuropathy as an adverse reaction to alprazolam.
Clinical case 78-year-old lady, with anger and anxiety, taking verapamil, magnesium, alprazolam (0.5 mg t.i.d.) and lorazepam (1mg o.d.) since 1998. In August 1999 the patient started to show a slowly progressive mainly sensitive polyneuropathy picture. In May 2000: she was observed by a neurologist, as she already showed severe walking disability, distal motor and sensory deficit with distressing dysesthesias and symptomatic xeroftalmia and xerostomia. She was on started gabapentin for treatment of the dysesthesias. Electromyography revealed axonal polyneuropathy with marked involvement of sensitive fibres. Analytic study was normal, including sedimentation rate, reactive protein C, haemoglobin A1C, proteinogram, folate, B12, syphilis, ANA and anti-SSA/SSB serology and thyroid function. In June 2000 the drug regime was changed from alprazolam to loflazelate 1mg o.d., as an association between alprazolam and xerostomia had been previously reported. A rapid improvement of mucous dryness and slowing down of polyneuropathy, both on clinical and electromyographic terms, was verified. The patient recovered and returned to a normal life.
Conclusion Following the OMS-1991 probability score, we could only classify this adverse reaction as probable, as we have not re-challenged the patient with the drug, for ethical reasons. Improvement after introduction of loflazelate and maintenance of lorazepam are against the hypothesis of an idiosyncratic group-effect of benzodiazepines. The authors could not find any description in the literature connecting polyneuropathy with alprazolam or other benzodiazepine, stressing the idea that theoretically any drug can be incriminated on the aetiology of a polyneuropathy, and alerting for the detection of other cases.

P 1102

Peripheral neuropathy in rheumatoid arthritis

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Introduction The occurrence of polyneuropathy in Rheumatoid Arthritis (RA) and the correlation with the clinical features of the disease has not yet been well defined. It is often difficult to diagnose these slight or early neuropathies and neurological evaluation of RA patients may be difficult to interpret, since some neuropathic findings, particularly muscle weakness and sensory disturbances, may be confused with symptoms resulting from pain in joints and limitations of movement. Aim of this study was to assess the ENG pattern of polyneuropathy, its relationship with RA clinical parameters and with neuropathic clinical scores.
Method Thirty-eight (28 females) outpatients with RA classified by ACR criteria, aged 26–65 years (mean 53), with a disease duration of 1–34 years (mean 8.7) were studied.

Polyneuropathy was evaluated with a questionnaire (Neuropathic Symptoms Score, NSS), physical examination (Neuropathy Disability Score, NDS), quantified sensory testing (QST), and neurophysiological measurements.
Results Abnormalities as indicated by the questionnaire (NSS) were found in 8/38 patients, while an abnormal neurological examination (NDS) was found in 17/38. In 13/38 patients ENG signs of polyneuropathy were detected, mostly consisting of mild or moderate sensory axonal neuropathy.
Conclusion Clinical examination alone is not sensitive enough in detecting polyneuropathy in patients with RA, since associated symptoms (pain, limitation of movement) may be confused with neurological dysfunctions such as sensory and motor disturbances. The inclusion of an ENG examination therefore is recommended in the global assessment of RA patients, even when history and clinical examination are not suggestive of peripheral nerve involvement.

P 1103

Incidence of blink-reflex abnormalities in patients suffering from idiopathic trigeminal neuralgia

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Objective We performed a study to determine the incidence of Blink-reflex abnormalities and EEG changes in patients suffering from idiopathic trigeminal neuralgia.
Method The study was performed on 25 patients diagnosed according to the ICD-X criteria. We measured amplitudes and latencies of the ipsilateral early phasic component (R1) and the bilateral late tonic component (R2, R2'). EEG was recorded using the International 10–20 EEG system. The results were compared to those of the control group.
Results In the group of subjects suffering from idiopathic trigeminal neuralgia the Blink-reflex results showed prolonged latencies of both R2 and R2' when stimulating the afflicted side, and normal latencies of all components when stimulating the contralateral side. The latencies of late components have exceeded the normal range (+2.5 SD) in 35% of the cases. EEG changes, mostly diffuse or bitemporal appearance of theta waves, were found in 27% of the subjects tested. EEG findings and Blink-reflex late components latencies showed a high level of correlation with each other as well as with frequency of symptoms.
Conclusion We believe that Blink-reflex changes are more frequently encountered among patients with idiopathic trigeminal neuralgia than EEG abnormalities.

P 1104

Guillain Barré syndrome: New therapeutic trial

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Objective The goal of this study was to evaluate the response of Guillain-Barré syndrome (GBS) patients to IV pooled γ-globulin in combination with IV pulse methylpredenisolone therapy.
Introduction GBS is an acute immune-mediated disease of the peripheral nervous system. Plasma exchange (PE) and high-dose IV immunoglobulin (IVIg) showed to be equally effective in the past, but still therapeutics are costly and morbidity is still considerable. Additional treatment needs to be developed.
Method We studied 9 patients (group I), and diseased control group of 7 patients (group II) with GBS through diagnostic criteria (Asbury and Cornblath, 1990). Electrodiagnostic testing was performed at entry and after 8 weeks, (Meulstee, and van der Meche, 1995). The therapeutic effect on group I through IV pooled γ-globulin in a dose of 30 mg/kg/day and IV pulse methylpredenisolone in a dose of 20-30 mg/kg/day for 5 days was compared with the therapeutic effect on group II through IVIg (3 patients) and PE (4 patients). Function was ranked on admission, according to the scale of Hughes (1990). Outcome was measured after 8 weeks, the end point of this study.
Results After the beginning of combined IV pooled γ-globulin and pulse methylpredenisolone in group I, 5 patients showed improvement by 2 functional grades, and 4 patients showed complete recovery. Among group II, 3 patients improved by two functional grades, 2 patients improved by 1 functional grade, and the other 2 patients remained stationary.
Conclusion GBS patients can benefit from the use of IV pooled γ-globulin and pulse methylpredenisolone therapy.

P 1105

Epidemiological data about polyneuropathies in Romania

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Introduction Epidemiological data from Eastern European countries are still very difficult to collect and lack consistency. We present the first study about epidemiology of polyneuropathies (PN) in Romania based on data from the National Medical Statistic Committee (NMSC).
Method Incidences of different types of PN at three main levels of medical care (general practitioner – family doctor, specialist from ambulatory and specialist from hospital care) were collected from NMSC for the year 2000. The classes of PN registered were those from the International Classification of Diseases. Data were also analysed by age, sex and urban/rural distribution and availability of methods of investigations.
Results Globally, PN represented 15% (63797 cases) of all neurological disorders newly diagnosed in 2000. 43% (27433 cases) of diagnoses were made almost exclusively clinically by family doctors, with similar proportions between rural and urban patients. Ambulatory care specialists first diagnosed PN in 38% cases, in an overwhelming majority in urban population. In hospitals (19% diagnoses) urban and rural population have similar addressability. The much larger access to investigations allowed cathegorisation of PN as: inflammatory (26.1%), hereditary (10.8%), toxic-drugs induced (56.4%), autonomic (6.7%).
Conclusion In Romania, diagnosis of PN is made mainly in ambulatory settings, where it is largely clinical and lacks etiological specificity. Some types of PN are surely underestimated. This situation is reversed in hospitals; the most refined etiological diagnosis (including nerve and muscle biopsy, genetic testing etc.) can be accomplished in a few centres. The situation is improving with increasing development of private medical centres.

P 1106

NADPH-diaphorase expression in the peripheral nerve and its changes in diphtheritic polyneuropathy and Guillain-Barré syndrome

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Introduction It is known that only select cell populations have nitric oxide synthesis (NOS), and until recently there was no knowledge about its presence in peripheral nerves (PN). We were the first, who histochemically demonstrated the existence of this enzyme in Schwann cells (SC) of PN by using NADPH-diaphorase (NADPH-d). We found that NADPH-d activity is permanently present in all parts of SC cytoplasm and is ultrastructurally localized on nuclear membranes and membranes of the endoplasmic reticulum. Axon and myelin were devoid of enzyme activity.
Method In the present study the localization and ultrastructural distribution of NOS was determined in biopsies of n. suralis specimens from 7 patients suffering from diphtheritic polyneuropathy (DP) and 6 patients suffering from Guillain-Barré syndrome (SGB).
Results In DP, SCs showed destructive and reparative processes. A reduction of enzyme activity was detected in those cells that took part in utilization of destroyed myelin. In the remyelinating SCs, NADPH-d activity was prominent not only in cytoplasm but in the nucleus as well.
In GBS, the main participants of pathological processes are immunocompetent cells. High diversity and temporal gradient of NADPH-d activity were found in mononuclear cells. NADPH-d activity was high in migrating monocytes and those and interacting with SC, and low or absent in phagocytes
Conclusion Our results suggest that NADPH-d in SCs is associated with the constitutive form of NOS, which takes part in myelin maintenance and remyelination. In immunocompetent cells, NADPH-d belongs to the inducible form of NOS, that produces nitric oxide periodically, in response to induction, in addition to known cytokines.

P 1107

Myasthenia gravis and functional status of the afferent ways of the spinal cord

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How does realization of excitation on the afferent ways of a spinal cord (AWSC) change at the presence of Myasthenia gravis (MG)? Patients suffering from MG first of all have infringement of transfer of excitation from nerves to muscles. The question of the functional status of the afferent ways has not been studied until now. We have developed a new method of rating for the functional status of the AWSC in our laboratory (Russian patent for the invention No. 2136328).
Method This method is carried out through magnetic stimulation of the central nervous system (CNS). Using this diagnostic approach we investigated 26 patients (19–46 years) with MG (the general form) and 30 healthy individuals.
Results In the healthy group the speed of realization of excitation on the AWSC was 90.3±1.7 m/s (mean±standard error, m/s – meters per one second). When studying the functional status of the AWSC we found attributes of infringement of realization of excitation on the AWSC (decrease) in 20 patients (76.9%) with a speed of up to 56.1±2.6 m/s. In 17 patients (85.0%) the changes were bilateral.
Conclusion The insufficiency of realization of excitation on the AWSC could be due to difficulties of realization of stimulating processes. The reason of these changes may consist of infringement of synaptic transfer of excitation between afferent neurons of the CNS. Besides, the insufficiency of realization of excitation on the AWSC to CNS paths may be caused by activation of braking processes.

P 1108

Isolated spinal neurosarcoidosis – a challenging diagnosis

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Introduction Sarcoidosis is a chronic inflammatory multisystemic disease of unknown cause that in some cases may exclusively affect the nervous system. The isolated involvement of the spinal cord is rare and may cause problems of differential diagnosis because a biopsy of the lesion is not always feasible. The authors present a challenging case of possible isolated spinal neurosarcoidosis.
Clinical presentation Female; 68 years old with type 2 diabetes mellitus. She was admitted due to fluctuating complaints of backache, paresthesias and leg weakness with one year of evolution, which had worsened in the previous month. On neurological examination she had areflexic paraparesis (grade 3), sensitive level at T9, loss of vibration in the ankles and knees, urinary incontinence and constipation. Spinal MRI showed diffuse enlargement of the cord from T6–T12, with poor intramedullary enhancement after gadolinium. Gallium-67 SPECT revealed elective fixation in the spinal cord at the same level. CSF studies, cerebral MRI, chest radiograph, thoracic CT and serum angiotensin-converting enzyme levels were unremarkable. Infectious, neoplastic, toxic, and endocrine causes were excluded. A biopsy was not performed due to the localization of the lesion. We introduce corticosteroid therapy with sustained clinical improvement. She was able to walk two months later.
Conclusion This case reflects the difficulty in making a definitive diagnosis when histology is not available. Nevertheless the clinical picture, neuroimaging, response to therapy and exclusion of other pathologies lead to the diagnosis of possible isolated spinal neurosarcoidosis.

P 1109

Vascular chronic ischemic myelopathy: clinical, imaging and electrophysiological examination

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Introduction We present the longitudinal clinical, neuroimaging and electrophysiological study of 75 patients with vascular chronic ischemic myelopathy.
Method All cases were confirmed by neuroimaging examinations: CT-myelograhpy and/or MRI and/or selective spinal angiography. Electrophysiological examination included needle electromyography, sensitive and motor electroneurography, F-wave study, Hoffman reflex, bulbo-cavernosus reflex and motor-evoked potentials. 30 healthy subjects were considered as a group of references. Three vascular syndromes were considered: anterior spinal artery syndrome, syndrome of complete transversal lesion and posterior spinal arteries (artery) syndrome. Clinical and electrophysiological findings were assessed in each cases with etiological factors and the level of ischemic spinal lesion.
Results Electrophysiological abnormalities were founded in 100% of cases. Based on statistical analysis of the results, electrodiagnostic criteria were elaborated for the discrimination of each syndrome of vascular chronic ischemic myelopathy. In addition to this, data were found indicating that ischemic damage of spinal cord tissue causes the morpho-functional re-

organization of motor units and segmental neuro-muscular apparatus. Moreover, as a result of neuronal plasticity at this level of nervous system new programs of motor function are established.
Conclusion The general conclusion of this work is that multimodal electrophysiological investigation as a consciously extension of clinical examination is very important in the positive and differential diagnosis of ischemic disturbances of spinal cord blood circulation. An electrophysiological examination is also useful for the prognosis of ischemic vascular myelopathy and for the control of treatment efficacy.

P 1110

A 65 year-old woman with acute transient encephalopathy and Tarlov cysts

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Introduction Tarlov or perineural cysts are lesions of the nerve roots often found on the sacral region. Although usually asymptomatic and diagnosed incidentally, they are sometimes associated with low back, sciatic and sacrococcygeal pain, sensory and motor disturbances in the lower extremities, and sphincter disturbances, perhaps as a result of CSF pressure changes.
Case report A 65 year-old woman with previous recurrent episodes of nausea and vomiting attributed to biliary dyskinesia, was admitted to the hospital with a 16-hour history of nausea and vomiting, followed by painful paresthesias and dysesthesias of her legs, buttocks and perianal region, and finally fever and confusion. On evaluation she was obtunded, disoriented, able only to follow simple commands, and with neck stiffness. General examination was otherwise unremarkable. The CSF analysis revealed pleocytosis (990 WBC/mm^3), very high protein content (8.0 g/l) and normal glucose level. Blood and CSF cultures were sterile. Routine blood counts, chemistries, and urine analysis were normal except for leucocytosis. A lumbosacral MRI demonstrated multiple thin-walled fluid-filled sacral perineural cysts with partial sacral erosion. The clinical picture resolved completely and spontaneously in the next 24 hours. She remains well after two years of follow-up.
Conclusion The rupture of intracerebral arachnoid cysts is sometimes associated with chemical meningitis. Our hypothesis is that that during vomiting effort, the transiently high intracranial pressure caused the rupture of a perineural cyst into the subarachnoid space. This in turn could have caused the lower limb symptoms and the subsequent transient encephalopathy, as the result of chemical meningitis.

P 1111

Comparison of tensilon test with ice pack test in diagnosing ocular myasthenia gravis

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Introduction Myasthenia gravis is an autoimmune disorder in which acetylcholine receptor antibodies attack the postsynaptic membrane of neuromuscular junction. Patients often have ophthalmologic symptoms. Myasthenia gravis should be considered in every patient with ptosis and/or diplopia. The main diagnostic test for myasthenia is Tensilon test that may produce serious side effects, including significant bradycardia, loss of consciousness, and death. The goal of our study was to compare the results of Ice test with Tensilon test in ptotic patients.
Method Between September 1996 and October 1999, we entered 124 patients with ptosis into our study, and did both ice test and Tensilon test on every patient. The distance between upper lid margin and corneal light reflex was measured before and after a 2-minute application of ice to the ptotic eyelid, two or more millimetres of improvement after ice application was considered a positive Ice test result, then every patient underwent Tensilon test.
Results A positive Tensilon test result was noted in 43 of 124 (34.7%) patients, and the result of ice pack test was completely the same as Tensilon test, and there was no false positive and no false negative.
Conclusion The ice pack test is a simple, short and non-invasive test, which has the same results as the Tensilon test, and can be used instead of Tensilon test for diagnosing myasthenia gravis in ptotic patients.

P 1112

Pregabalin shows no evidence of retinal toxicity

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Introduction Vigabatrin has been shown to cause a predominantly asymptomatic, irreversible, constriction of the visual field in 30%–40% of patients. In this paper we report the results of prospective ophthalmologic testing obtained in therapeutic clinical trials with pregabalin.
Methods Formal visual fields, Snellen acuity, and funduscopic examinations were performed at 3 or 6 month intervals in 3615 patients participating in randomised controlled trials (RCTs) and open-label treatment with pregabalin for treatment of epilepsy, pain and anxiety disorders. 1390 patients were followed for more than 1 year. Cases of visual deterioration were defined through adverse events, judgment by the examining ophthalmologist, and quantitative criteria.
Results In RCTs of up to 3 months in duration, incidence of visual field loss, visual acuity loss and funduscopic change was similar in pregabalin treated and placebo treated patients. For pregabalin and placebo groups respectively, the incidence of cases of visual field loss was 5.3% and 4.8%; visual acuity loss 4.1% and 3.3%; funduscopic change 1.7% and 2.1%. No dose-dependent increase in the incidence of abnormalities was observed (range: 50–600 mg/day). In long-term open label studies some increase in treatment emergent visual dysfunction was seen in high-risk groups (diabetics and elderly). The nature and severity of these events were similar to those seen in placebo treated patients in RCTs. No increase in the frequency of these events was seen in lower risk patients.
Conclusion Following review of more than 3600 pregabalin-treated patients, no evidence of retinal or optic nerve toxicity was found.

P 1113

Medial plantar nerve conduction study in diabetic neuropathy diagnosis

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Introduction The standard diabetic neuropathy diagnosis is

based on: 1– symptoms; 2– signs; 3– conduction studies and electromyography; 4– quantitative sensory testing; 5– quantitative autonomic testing. Motor and sensory nerve fibre conduction studies present the so-called golden standard in diabetic neuropathy diagnostic process. Less favourable anatomic conditions in comparison to upper extremities allow in routine clinical practice the investigation of a sole lower extremities sensory nerve – the sural nerve. In our opinion this causes the impossibility to diagnose neuropathy in the early disease stage when only digital nerve branches are affected.

Method The aim of our work is to compare the diagnostic value of medial plantar nerve and sural nerve conduction velocity in diabetes mellitus I. type patients group (39 patients) with respect to the disease duration. The standard superficial antidromic technique was used for both nerves: 1– sural nerve (calf stimulation, lateral ankle registration); 2– medial plantar nerve (medial ankle stimulation, toe registration).

Results The diagnostic value of medial plantar nerve conduction study in diabetic neuropathy is significantly higher than that of the sural nerve. The first pathologic change is the conduction velocity slowing followed by reduction of the SNAPs amplitude and area. The ultimate pathologic change is the absence of the response. All these changes copy the disease evolution along the time

We recommend the antidromic medial plantar nerve neurogram as a useful and easy to realize test in the electrophysiological diagnosis of early stages of diabetic neuropathy.

P 1114

Prolonged optokinetic stimulation generates podokinetic after rotation

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Introduction In previous studies we showed that after prolonged stepping-in-place on the center of a rotating platform, blindfolded subjects could no longer step in place on firm floor. Instead they invariable rotated themselves relative to space without perceiving their rotation, a phenomenon termed podokinetic after-rotation (PKAR). We speculated that prolonged optokinetic stimulation (OKs) alone may generate similar PKAR.

Objective To evaluate the effects of OKs on podokinetic (PK) responses.

Methods Eight healthy subjects participated in the study. After a baseline stepping test they were seated in a closed cabin and randomly (right or left) exposed to an OKs (45deg/sec) covering the whole visual field for 30 min. Following this procedure, blindfolded subjects attempted stepping-in-place on the stationary floor for 30 min.

Results When trying to step in place after OKs all subjects turned relative to space without any perception of rotation. The direction of this optokinetically after-rotation (oPKAR) was opposite to that of the direction of OKs. Mean peak velocity of oPKAR was 7.5 deg/sec and it was reached after about 5–6 min of stepping. After that, there was a progressive velocity decay which exhibited a discharging time constant on the order of 8–10 min.

Conclusions OKs alone causes oPKAR. Long-term OKs probably charges a storage element for podomotor activity with a relatively prolonged time constant. This novel form of neural interaction and adaptive plasticity may have significant implications for treatment of vestibular and other locomotor disorders.

P 1115

Computer program for peripheric monopareses diagnosis

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P 1116

Nerve conduction study in diabetic polyneuropathy

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P 1117

Carpal tunnel syndrome related to automobile accidents

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P 1118

Neuroprotective treatment of diabetic peripheral neropathy in children

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P1119

Functional state of blood-brain barrier of patients with spinal cord pathology

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P 1120

Spinal nerve root stimulation in radicular pain due to postoperative epidural fibrosis

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