and four patients used in combination with plasmapheresis, tamoxifen, melphalan and multiglycosidorum tripterygii.
Conclusions: The symptoms, clinical course, and management of the patients reported here were similar to those observed in the literature. Our study confirms the existence of a close link between symptoms and immunologic changes. The patients who were diagnosed must have at least three of the manifestations: polyneuropathy and plasmagenic disorders are always present.

P1439

Neuroimaging and pathology of diabetic striatopathy

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Unilateral movement disorders and contralateral neuroimaging abnormalities of the striatum have been sporadically reported as a syndrome in association with diabetics. We describe here three patients with the syndrome, one of who had undergone a biopsy of the striatum. Patient 1: A 73-year-old man with diabetes mellitus (DM) (HbA1c 18.9) developed right hemichorea while adjusting the insulin dosage. The MRI demonstrated T1-high, T2-iso-dense signal lesion confined to the left lenticular nucleus. Patient 2: A 16-year-old boy with IDDM (HbA1c 11.6), who developed a florid left hemichorea, demonstrated T1-hyper-, T2-iso-intense lesion localized to the right lenticular nucleus. Patient 3: A 56-year-old man with DM (HbA1c 8.0) was hospitalized with left-sided transient weakness. The MRI demonstrated T1-hyperintense swollen mass-like lesion that occupied the entire striatum of the right side. A needle biopsy was performed, because a brain tumor was suspected; however, it was excluded because it was devoid of neoplastic cells. Instead, the specimen contained a patchy necrotic tissue, marked thickening of the adventitia/media of arterioles and venules, and narrowing of the vessel lumens. Lymphocytic infiltration and macrophage invasion were conspicuous. Fibrinoid necrosis, extravasation of erythrocytes, and capillary proliferation were a prominent feature. These changes appear to be unique to this particular syndrome. We suggest that the syndrome is caused in poorly controlled diabetics by the obliterative vasculopathy/vasculitis, the vulnerability of which is uniquely restricted to the striatum.

P1440

The importance of vasculitis in pathogenesis of diabetic polyneuropathy

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Background: Peripheral nerves affection is the most frequent complication of diabetes mellitus (DM). Metabolic and vascular disorders are considered to be the basic pathophysiologic mechanisms of neurologic disorders of DM.
Aim: To learn the role of vasculitis in the development of diabetic polyneuropathy.
Material and methods: 45 patients with DM (33 women and 12 men), aged from 18 to 60 years (mean 49.7 ± 0.9) have been investigated. Clinical, neurologic and immunoassay investigations were performed. Immunoassay included determination myeloperoxidase antibody level (a-MP) in blood serum. A-MP is a system vasculitis marker. Control group consisted of 20 healthy persons.
Results: All patients had diabetic polyneuropathy. They were divided into two groups. Group 1: With surface perception disorders (16 patients); group 2: With surface and vibration perception disorders and low-grade paresis of lower and upper extremities (29 patients). In group 1, the mean disease duration consisted 6.45 ± 1.4 years; in group 2, 12.0 ± 1.4 years. a-MP level in all patients was 8.31 ± 0.5 UOD, in control group, 0.8 ± 0.02 UOD. A-MP level in group 1 was 7.14 ± 0.6 un ml; in group 2, 8.96 ± 0.6 UOD, i.e. the more manifested a polyneuropathy the higher a-MP level.
Conclusion: Our results showed, that the more manifested and a longer polyneuropathy was the more significantly A-MP level increased. This suggests presence of autoimmune inflammation in peripheral nerves in DM caused by vasculitis and resulted in demyelination, axonal degeneration and diabetic polyneuropathy.

P1441

Clinical study of hypokalemic periodic paralysis with 64 cases

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Objective: To investigate the clinical features of hypokalemic periodic paralysis.
Methods: 64 cases with hypokalemic periodic paralysis admitted to the Shanghai First People's Hospital from 1991 to 2001 were analyzed.
Results: Of the 64 cases, there were 50 cases with primary hypokalemic periodic paralysis, in which there were four cases with family history and 46 sporadic cases. Ten cases secondary to hyperthyroidism and four cases secondary to hormone treatments. There were 32 cases showing typical ECG changes of hypokalemia in 42 cases tested for ECG and 21 cases showed high level serum CPK in 30 cases tested for CPK. There were six cases accompanied with limb numbness and five cases with muscle pain.
Conclusion: There were limb numbness and elevations of CPK in some cases, serum level of potassium and ECG examination were beneficial to early diagnosis and treatment. Therapy against etiology is important in secondary hypokalemic periodic paralysis and to avoid inductive factors is the key in the prevention of relapse.

P1442

Functional and electrophysiologic correlations of phrenic nerve's transfer rate in patients with COPD

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Objective: To determine if a patient with COPD, relationship between phrenic nerve functions and pulmonary function tests (PFT), and difference of phrenic nerve transfer rate, shows early indication of neuropathy diagnosis.
Method: In this study, we used 29 stable patients with (15 moderate, 14 serious) COPD and 21 healthy controls. All

subjects used neuropath symptom score (NSS) test and findings NSS < 2 include into this study. After measuring PFT and artery blood level we used McLean's method for measuring patients' latency of phrenic nerve and ampluted with surface electrodes. Patients, who have comorbid neuropathy, measured four extremities' nerve transfer rate, so motor and sensory transfer were assessed.

Results: Patients' and controls' age average 59.4 ± 7.5 and 63.5 ± 3.1 years, respectively ($P = 0.2$), average disease period was 11.0 ± 7.6 years. Patients had an average pH of 7.38, PaO_2 69.2 ± 10.6 mmHg, $PaCO_2$ 40.6 ± 7.8 mmHg. Patients had an average of FEV1% = 48.7 ± 15.8, FEV1/FVC = 49.7 ± 7.8, FRC% = 114.5 ± 4.4, RV% = 157 ± 40, TLC% = 113 ± 14; IC = 2.6 ± 1.1. Result of applied nerve transfer studies showed total 12 (2%) carpal tunnel syndrome fact, four (13%) patients sensory, seven (24%) patients had determine mixed type polyneuropathy out of trap neuropathy. Measured phrenic nerve's distal latency was longer in patients than in controls ($P = 0.04$). However, lowness of amplitude was not statistically significant. There was only a relationship between the TLC and the phrenic nerve transfer rate (cc = 0.9, $P = 0.03$).

Conclusions: Findings showed that patients with COPD lengthen of distal latency in phrenic nerve are a part of the polyneuropathic condition. However, longitudinal studies are needed in order to evaluate prognosis and response of treatment of phrenic nerve neuropathy.

P1443

Human lung tumour cells induce changes in human astrocyte phenotype *in vitro*: Relevance to metastasis evolution

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Brain metastases arising from lung tumours are relatively common. Metastasis formation involves several events initiated by the tumour cell, including the release of proteolytic enzymes, cytokines and growth factors, and also the induction of angiogenesis, i.e. formation of new blood vessels. Tumour cells migrate through the endothelial cell layer lining cerebral blood vessels and come in contact with astrocytes (the main supporting cells of the brain). Characteristically a boundary forms around the metastasis restricting further brain invasion. The cellular processes involved in metastasis formation are largely unknown, with only a few animal studies reported. By using an *in vitro* model, we are investigating the cellular changes induced by human lung tumour cells on human astrocytes. Conditioned medium from well-characterized human lung tumour cell lines, A549 (adenocarcinoma) and SK-MES-1 (squamous), were harvested and applied to quiescent astrocytes in chemically defined medium. Striking changes in astrocytic morphology and immunocytochemical markers were seen particularly with SK-MES-1-conditioned medium. The markers assessed included basic fibroblast growth factor, nestin embryonic-neural cell adhesion molecule and epidermal growth factor receptor. These findings may have implications for the study of possible barrier formation by astrocytes around metastases and may vary according to primary lung tumour type. The definition of secreted factors producing these changes is ongoing using proteomic techniques.

P1444

Stiff-person syndrome positive for anti-amphyphisin antibodies: The presenting feature of a neuroendocrine lung cancer

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Background: Stiff-person syndrome (SPS) is a rare disease of the central nervous system, characterized by progressive rigidity of the body musculature with superimposed painful spasms. An autoimmune origin of the disease has been proposed. Amphyphisin is a protein highly concentrated in nerve terminals, where it has a putative role in endocytosis. Serum and cerebral spinal fluid IgG antibodies directed against that synaptic vesicle protein were found in patients with SPS associated, most commonly, with breast cancer and small cell lung cancer.

Subject: We report the case of a 72-year-old man, who developed a SPS, manifested predominantly as an acute gait disturbance, with severe axial and limb rigidity and induced spasms, positive for amphyphisin autoantibodies, which was the presenting feature of a neuroendocrine lung cancer. Following tumour treatment, our patient had neurological improvement.

Conclusion: Unusual acute SPS onset could suggest an overproduction of autoantibodies or a turnover point in the synaptic sensitivity. The detection of autoantibodies against amphyphisin, in patients with SPS, should be considered an indication for search an occult tumour, which could be other than the most commonly reported.

P1445

In vitro evaluation of antiproliferative potential of kynurenic acid, an endogenous glutamate receptor antagonist

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It has been found that glutamate antagonists inhibit proliferation of different human tumor cells. Kynurenic acid is a broad-spectrum antagonist of all subtypes of ionotropic glutamate receptors. Its presence has been documented in body fluids and tissues. In this study, the antiproliferative potential of kynurenic acid was investigated *in vitro*. Experiments were conducted on commercially available cell lines obtained from the European Collection of Cell Cultures and American Type Culture Collection. Cell proliferation was assessed by means of MTT assay. Kynurenic acid inhibited proliferation of human neuroblastoma (SKNAS), human brain astrocytoma (MOGGCCM), human medulloblastoma (TE671) and rat glioma (C6) cells. Moreover, it inhibited proliferation of human leukemia T-cells (Jurkat) and synovial fibroblasts, hyperplasia of these cells being one of the most striking features of inflammatory arthritis. The inhibitory effect was significant in concentration range of 1–5 mM. Interestingly, kynurenic acid at concentrations up to 5 mM did not inhibit the growth of oligodendroglia cells

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