Background: Peroneal palsy due to compression of the common peroneal nerve at the fibular head was described as a complication of delivery. We describe peroneal palsy caused by proximal nerve compression during childbirth.

Aims of the study: ENMG and clinical study to establish the site of compression.

Patient and methods: A 27-year-old woman had her second childbirth. The baby was large, 4500 g. The delivery was helped with suction. After childbirth there was a total left peroneal paresis. Lumbar MRI was normal. The first ENMG was performed 3 days after delivery. There was a total paresis of the left anterior tibial muscle in EMG. However, the motor conduction velocity of the common peroneal nerve was bilaterally normal between the popliteal space and ankle but the F-response was missing on the left side. The sensory response of the superficial peroneal and sural nerves as well as the H-reflex of the tibial nerve was normal on both sides. Two weeks after delivery there was still a total paresis of the left anterior tibial muscle but a few motor unit potentials were activated in the short toe extensor muscle. The left peroneal F-response appeared. 3.5 weeks after delivery there were fibrillation potentials in the left peroneal muscles. A few motor unit potentials were activated. Seven weeks after delivery there still were fibrillations in the left peroneal, but not in the L4-L5 paraspinal muscles. Nine months after delivery there were no residual signs of nerve injury of the left peroneal nerve.

Conclusion: There was conduction block in the motor axons of the left peroneal nerve leading to a total paresis of the anterior tibial muscle. The site of compression was not near the neck of fibula. There were no signs of lumbar root compression in MRI or paraspinal EMG. The probable site of acute injury was at the level of the posterior division of lumbosacral plexus, formed by the 4th and 5th lumbar roots, which has a compression-prone site on the iliac crest at the pelvic inlet.

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P28.2 Non-obstructive voiding dysfunction: Idiopathic, psychogenic or underlying neurogenic?

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Background: Since voiding dysfunction can be an expression of defective neurogenic control of the lower urinary tract, sacral nerve modulation by means of implanted devices (SNM) is one of the therapeutic options. Unfortunately, the success of treatment is unpredictable and variable. Moreover because the pathophysiology of functional urinary retention (UR) is still poorly understood, the careful choice of therapy is hindered, thus, almost as a rule, multiple treatments are tried in a stepwise

chopathological findings (by means of Minnesota Multiphase Personality Inventory MMPI2) can be recognized as responsible of a predictable failure of the therapy.

Aim, materials and methods: To assess the hypothesis of an underlying neurogenic or prevalent psychogenic involvement, we performed a detailed neurophysiological investigation (SEP, EMG, ENG, Sacral reflexes, SSR) and MMPI2 in a group of 14 female patients (42 years mean age) (nine complete and five incomplete UR) without overt neurological symptoms nor a history of trauma or neurological disease, thus classified from diopathic before implanted with SNM.

Results: Seven patients with positive results of SNM, had normal MMPI2 profile and alterations on neurophysiological assessment. Seven patients had negative results of SNM: in five patients both neurophysiology and MMPI2 fail to detect an underlying involvement (neurogenic nor psychogenic); in one neurophysiological evaluation was normal with MMPI negative and in one only neurogenic involvement.

Conclusions: Neurophysiological diagnostic selection and psychological assessment can be useful predictive factor of SNM results. Patients with an underlying neurogenic alteration show better results in SNM, while patients with MMPI2 positive and negative neurophysiology should be excluded from SNM implant.

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P28.3 Cortical neuroplasticity in sacral neuromodulation: Neurophysiological evidences in long term follow up

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Background: Neurophysiological evaluation (NPE) in patients addressed to sacral neuromodulation (SNM) revealed an undisclosed neurogenic alteration as a possible cause of imbalance in afferent input to cortical area. Moreover SNM seems to act on the afferent pathway with a specific modulating effect related to parameters of stimulation: an increase from 21 to 40 Hz leads to a decrease in pudendal somatosensory evoked potentials (PSEPs) P40 latency resulting in a sort of facilitation on afferent impulse transmission suggesting a reset of the processing mechanism.

Aim: To verify the hypothesis that the imbalance in afferent input can be modified by SNM leading to a neuroplastic effect on neurocontrol.

Patients and methods: From November 2001 to September 2005 215 pts underwent NPE (T0): in 111 pts (51.62%) implanted with SNM PSEPs after 1 (T1) and 24 months (T2) were confronted.

Results: In 4 pts (3.6%) implanted for idiopathic detrusor overactivity, clinical efficacy never was fully achieved



P40 was found in T2: SNM can modify the plasticity of neurocontrol mechanism, but need to be reinforced, perhaps in correlation with the underline pathophysiology of symptom. In 5 pts (4.5%) implanted for dysfunctional voiding in whom SNM was switched off with a persistent clinical efficacy no difference in PSEPs at T1 and T2 was seen: if a physiological restoration is achieved with SNM, the effect on neurocontrol mechanism persists in a normal fashion.

Conclusion: NPE shed light to the mechanism by which central nervous system modify its organization under SNM.

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P28.4 Role of penile sympathetic skin response in the neurophysiological evaluation of erectile dysfunction C. Valles Antuña, J.M. Fernández Gómez, S. Escaf, J.L. Martín Benito, F. Fernández González

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Introduction: Within the limits of sexual and urinary malfunction the sympathetic skin responses (SSR) from limbs have been used in the evaluation of possible autonomic malfunction in patients suffering from erectile dysfunction (ED).

Aims: To evaluate the role of penile SSR in the assessment of neurological function in males suffering from ED.

Patients and methods: We studied 83 males diagnosed of ED. They were clinically evaluated and put to a set of neurophysiological tests which included nerve conduction studies, evoked potentials, EMG, bulbocavernous reflex, quantitative sensory test and SSR from limbs and penis.

Results: In ED patients, the percentage of SSR was considerably lower in the penis (52%) than in the hand (90%) or the sole (89%). We found correlation between SSR percentages and the severity of ED, assessed by means of the International Index of Erectile Function (IIEF). Latency of SSR show a statistical association between the palm and the sole but not with the penis. Regarding the penis, no significant associations were found between the results of the two tests used to asses the function of C type fibers (SSR and Heat Pain Thresholds).

Discussion: The simultaneous register of SSR, performed in the palm, the sole and the penis increased considerably the diagnostic efficacy of this test. The percentage of blockings showed to be useful as an indicator of the affectation of efferent C fibres. Despite SSR is a polysynaptic potential of long latency and regulated by the cerebral cortex, the results of our study make advisable to value the latencies of SSR in the three areas of register and especially

marker or of alterations of anatomic via at lumbosacral y/or pudendal level.

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P28.5 Peripheral neuromodulation using surface electrode for treatment of overactive bladder (oab): initial experience <u>F. Monti</u>, M. Semenic, S. Siracusano, S. Ciciliato, G. Sau, G. Pizzolato, D. Pizzolato, G. Sau, G. Pizzolato, D. Pizzolato, D. Pizzolato, G. Sau, G. Pizzolato, D. Pizzolato,

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Percutaneus tibial nerve stimulation is effective even if it requires a needle insertion. We report preliminary results of posterior tibial nerve neuromodulation (PTNN) using surface electrode in patients affected by overactive bladder (AOB). Twelve female patients (age 54.4 ± 16.0) affected by AOB and non-responders to antimuscarinic underwent TNM using surface electrode. The voiding diaries, SF-36 and King's health questionnaires were administered before starting the first week of treatment and at the end of therapy. A bipolar surface electrical stimulator was placed behind the medial malleolus, along the course of the posterior tibial nerve, with the cathode in proximal position and the anode 3 cm distally. Couple electrodes in Ag/Ag were used for recording, with the cathode placed over the abduttor hallucis muscle belly and the anode on the tendon. A disk ground electrode was placed between the stimulating and recording electrodes. Electrical square-wave stimuli of 0.1 ms duration were utilized. The stimulus intensity was set in order to obtain a maximal CMAP (compound muscle action potential). Stimulation protocol was 1 stim/s for 20 min, and the stimulation was repeated twice every week for seven weeks for each subject. We observed a statistically significant reduction of voiding frequencies during night $(2 \pm 0.5 \text{ to } 1.4 \pm 0.5 \text{ times }$ p < 0.0001) and day (13.3 ± 1.9 to 8.9 ± 2.5 times – p < 0.0001). No side effects were observed. SF-36 and KHQ questionnaires showed a statistically significant improvement of QoL at the end of therapy. The use of surface electrode in PTNN seems to be a non-invasive and successful treatment option for AOB.

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P28.6 Relationship between the autonomic dysfunction of multiple system atrophy and external anal sphincter electromyography

Han Wang

