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ABSTRACTS

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A. Moglia, G. Sirabella,
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TRIGEMINAL EVOKED POTENTIALS IN CLUSTER HEADACHE

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This study was performed with the aim of detecting trigeminal evoked potential (TEP) alterations in patients suffering from cluster headache. It was suggested that altered TEPs could demonstrate a direct involvement of the trigeminal nerve in cluster headache. We investigated 36 patients suffering from the episodic form of cluster headache. TEP recordings were performed during the bout, but outside the cluster attack in 26 patients, whereas in 10 patients it was possible to perform a recording session during the attack.

TEPs were performed according to the technique and normative data already published by Leandri et al. The infraorbital nerve was electrically stimulated percutaneously, and the evoked far-field was recorded from the scalp.

Of the 26 cases investigated in the interictal period, 4 showed a delay of some of the TEP components (W2 and W3). Meanwhile, all of the 10 cases recorded during the attack showed severe alterations of TEPs, with either absence or delay of and amplitude reduction of W2 and W3.

The first 3 components of TEPs are by far the most reliable and have been demonstrated to originate from the maxillary nerve (W1), the retrogasserian root (W2) and the pontine nuclei (W3). All the alterations found in our subjects involved W2 and W3, thus suggesting an impaired conduction of the gasserian ganglion-retrogasserian root. The same alteration had been detected in cases of space-occupying lesions of the parasellar region. In our cases, therefore, we can hypothesise that the internal carotid artery, swollen up after the start of the cluster attack, may impinge upon the trigeminal nerve, thus giving rise to those signs of impaired conduction detected by TEPs. Compression of the trigeminal nerve might then be responsible for the high intensity and very localised pain of the attack. It could also trigger trigemino-vascular activation, with further worsening of pain and inflammatory phenomena.

THE PAIN IN DISC DISEASE

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Root pain is commonly linked to nerve compression (disc-root or spine-root conflict). However, there is much evidence in clinical practice that the cause-effect ratio of root pain is not as straightforward as is generally thought. For example, we know that many people with known disc herniation live with the lesion in between pain attacks, even though the morphology of the disc lesion and consequent nerve root compression remain unchanged on CT and MR scans.

Moreover, when the disc compression is corrected by surgery, many patients continue to feel pain, either attenuated or even exacerbated, irrespective of the structural changes to the disc herniation disclosed by neuroradiological scans after the operation. These findings led us to investigate the mechanisms responsible for root pain, reviewing the literature and distinguishing two broad categories of pathogenetic mechanisms: mechanical and inflammatory.

The mechanical factors responsible for pain are linked to the mass effect of the herniated disc material. In turn, these can be divided into:

Direct mechanical factors: given the absence of nociceptors in the nerve bundle, these are linked in order of importance:
- compression of the spinal ganglion, possible in intraforaminal and extraforaminal herniation;
- deformation and flattening of the ligaments and annulus, location of the afferent nociceptors to Luschka’s nerve of the posterior root of the spinal nerve;
- deformation and flattening of nerve fibres disrupting the myelin nerve sheath with possible major conduction abnormalities.

Indirect mechanical factors, globally defined as vasculomediated and divided into:
- “ischaemic” vasculomediated factors with trophic nerve impairment due to compression on the arterial afferents and microcirculation of the nerve bundle and secondary anoxic demyelination of the nerve fibres;
- vasculomediated factors due to venous stasis with oedema and trophic nerve impairment caused by partial or total blockage of venous reflux (especially in intraforaminal herniations); this seems to be the most important mechanical factor responsible for root pain because of its effects on the spinal ganglion due to the anatomical relations between the intraforaminal vessels and spinal ganglion.

Neural and perineural inflammation plays a major role in the origin of root pain (note the frequent beneficial effects of corticosteroid therapy). The inflammatory factors include:
- Immune-mediated inflammatory reaction: experimental and other evidence shows that disc protrusion causes immune inflammatory events. The most likely hypothesis to account for this behaviour is that the adult intervertebral disc is segregated from a humoral standpoint with respect to the immune system as long as it is confined within the fibrocartilaginous structure of the annulus. Once the disc is herniated, it is recognised as “non self” by the immunocompetent system, triggering a cell-mediated reaction in other tissues. The presence of peridiscal inflammatory tissue is confirmed by the CT
and MR finding of peripheral enhancement of the disc fragment after i.v. contrast administration.

– Inflammatory reaction due to biohumoral factors linked to disc tissue: experimental evidence in this field includes:

  – phospholipase A2 (PLA2): the herniated disc material contains very high levels of PLA2 enzyme activity. PLA2 is a potent inducer of the inflammatory reaction since its enzymatic activation to arachidonic acid leads to the production of major chemical mediators of inflammation such as prostaglandins and leukotrienes;

  – matrix metalloproteinases (MMPs): there is significant production of these enzymes which enhance the inflammatory reaction by attacking disc tissue;

  – prostaglandin E2 (PGE2): PGE2 is produced directly by the disc tissue as well as by the enzymatic intervention of PLA2 which is known to be a potent inducer of inflammation; the same applies to interleukin 6 (IL6).

In the light of our present knowledge, root pain due to disc-root conflict should be deemed a symptom of multifactorial origin in which the neural and perineural inflammatory reaction and its biohumoral mediators play a major role flanked by venous stasis due to mass effect on the perineural circulation. Nerve compression appears to play an adjuvant role by generating nerve conduction abnormalities due to fibre demyelination with a direct or indirect anoxic-ischaemic mechanism.

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**INTERVENTIONAL CT GUIDED NEURORADIOLOGY IN PAIN SYNDROME MANAGEMENT**

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We report our experience in the management of pain syndrome in 325 subjects (oncological and non-oncological patients) from April 1996 to April 2001.

All the procedures were performed under CT guidance or under fluoroscopy with local subcutaneous anaesthesia.

We considered two different groups: oncological and non-oncological patients.

In the first group we treated, with alcoholization of the celiac plexus, 15 patients with pancreatic cancer who presented with constant abdominal pain. The treatment was performed with a 22 G needle and consisted of a percutaneous injection of 30 ml of absolute alcohol (diluted 50%) at the level of the celiac plexus to determine the neurolisis of the nervous cells. The results were evaluated with a pain score from 1 (no pain changes) to 4 (best results); 8 patients gave a pain score of 2 and 7 gave a pain score of 3.

In the second group we treated 300 patients affected by low back pain or lumbar radiculopathy with periganglionic and epidural steroid injection, with intradiscal or intraforaminal injection of 0.2-0.3 ml, with steroid or with alcohol facet joint injection.

These minimally invasive procedures were all performed under CT guidance, with a 20 or 22 G needle, injecting triamcinolone or dexamethasone acetate near the ganglion or in the epidural region.

In the patients belonging to this group, the results were evaluated using the McGill Pain questionnaire or the McNab Modified method.

We obtained a short-term success rate in 81% of the cases while the long-term success rate (two years in 80 patients) was obtained in 73%.

We also performed vertebroplasty in 10 cases, 4 with metastatic lesions and 6 with porotic fractures of the lumbar spine, under fluoroscopy.

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**EPIDURAL SPINAL CORD STIMULATION (SCS)**

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Spinal cord stimulation was introduced as medical practice in 1967 by Shealy et al. The spinal structures can be compared to an inhomogeneous volume conductor resulting from various compartments each of which has a different conductivity. In SCS the structures most likely to be involved include the dorsal columns, dorsal root entry zone, and dorsal horns.

Clinical results suggest that SCS may provide a suppressive action on dorsal horn neuronal hyperexcitability associated with signs of peripheral neuropathy.

It is clear that the evidence on SCS is limited and reports of SCS efficacy vary considerably, with success rates ranging from 18% to 86%. The number of electrodes implanted and the type of stimulation received are also very variable. The neurophysiology is unclear. It is not easy to correlate a particular pain condition with an expected success rate, but SCS seems to be more effective for pain of neurogenic origin.

The procedure offers a number of potential benefits, including: reversibility, a relatively simple implantation technique, patient control of stimulation parameters.

Bias is likely because of lack of randomization and blinding, because the procedure has been conducted by enthusiasts and reporting has been limited.

The results of two recent different Italian studies on SCS, a retrospective multicenter study based on a questionnaire administered by post and phone contact to 333 patients and a prospective study, designed in Pavia, of 119 consecutive patients, confirm the success of SCS. Patient satisfaction, which is an important measure of the success
of a pain treatment, was about 80% with an improvement of 63% in quality of life. More studies are needed to evaluate functional improvement following SCS.

PRELIMINARY EXPERIENCES WITH PROBE 2000 SPECTROSCOPIC IMAGING (SI)

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The Probe 2000 General Electric offers the possibility of performing either single or multi-voxel proton spectroscopy with automated water suppression and gradient shimming. The results are displayed as individual voxel spectra or as colour metabolite maps. We describe our preliminary experiences in acquisition and post-processing of the chemical spectra.

Sixteen patients suffering from cerebral tumours (9), traumatic brain injury (3), dementia (4) and 8 controls underwent multi-voxel PROBE-SI using a PRESS pulse sequence (TR/TE 1000/144, 1 NEX, FOV 22x22 cm, thickness 15 mm, acquisition grid 16x16).

PROBE-SI allows a simultaneous evaluation of large areas of the brain, permitting contralateral comparative information and the sampling of individual voxels in complex lesions. This option seems particularly useful in the diagnosis of diseases which do not affect the anatomical structure of the brain but change its chemistry or metabolite levels. In fact it shows areas of regional metabolic impairment in degenerative diseases, such as dementia, and provides parameters for the quantification of diffuse axonal injury in traumatic brain injury. Moreover, PROBE-SI is useful for characterizing and differentiating lesions such as radiation necrosis versus post-operative changes or residual/recurrent tumour thus reducing the invasive diagnostic and therapeutic procedures that are required.

FATIGUE IN IMMUNE MEDIATED NEUROPATHIES

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Immune-mediated polyneuropathies (PN) including Guillain Barré (GBS) and Miller Fisher syndromes, chronic inflammatory demyelinating polyneuropathy (CIDP), multifocal motor neuropathy, and PN associated with monoclonal gammopathy (PN+MG), are deemed to be caused by an autoimmune attack to nerve antigens. Even though these PN are mainly characterized by motor or sensory impairment or both, the presence of fatigue is often reported by the patients as a prominent symptom and, despite good recovery or stabilization, many patients with GBS, CIDP or PN+MG give fatigue as the main cause of the restriction on their daily and social activities and of the decrement in their quality of life. This issue was recently confirmed by Merkies et al., who examined the prevalence of fatigue, as assessed by a fatigue severity scale, and its relation to other PN symptoms (weakness, sensory loss) or PN severity in 113 patients with previous GBS (83) or with stable CIDP (22) or PN+MG (8) and in 113 age/gender matched healthy controls. In this study fatigue was more severe in immune PN than in controls (p<0.0001), and in GBS than in CIDP and PN+MG (p<0.0001). In addition, 80% of patients with immune PN had severe fatigue including 81-86% with normal strength or sensation, and fatigue was one of the 3 main disabling symptoms for 80% of patients and 13% of controls. Fatigue is a major complaint in immune PN, having a significant impact on quality of life even after several years of disease and represents an independent symptom and not a mere consequence of impaired strength or sensation.

ELECTROPHYSIOLOGICAL EVALUATIONS OF FATIGUE IN MULTIPLE SCLEROSIS

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Fatigue is a disabling symptom that is very frequent in multiple sclerosis (MS). Some features of fatigue in MS are quite specific and can appear at the onset of the disease. The fatigue in MS depends mainly on mechanisms originating in the central nervous system (central fatigue), although these are not clearly understood. Different electrophysiological techniques have been used to evaluate this symptom. Magnetic stimulation of the motor cortex, based on changes in cortical excitability, reveals changes in the cortical threshold of the motor evoked potential (MEP) when fatigue begins and increases; in particular, excitability changes of cortical MEPs by double cortical stimulation at different intervals (absolute and relative refractory periods), changes in cortical MEP amplitude/area after different kinds of fatiguing efforts, changes in cortical silent period and in the amplitude of
post-silent period cortical MEPs during fatiguing isometric contractions. Biomechanical tests seem to constitute the most sensitive and reliable methods for measuring fatique in MS.

In our experience, central fatigue evaluation by the technique of the interpolation of stimulated muscle twitch in the voluntary mechanogram is able to measure different degrees of central fatigue in patients with MS; furthermore, we observed that this method is able to evaluate well the mechanisms of physiological fatigue, optimising the treatment for fatigue in MS with fluoxetine and 4-aminopyridine.

QUANTITATIVE EVALUATION OF BODY COMPOSITION USING MR IN PATIENTS AFFECTED BY DUCHENNE MUSCULAR DYSTROPHY

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Duchenne muscular dystrophy (DMD) is the most common and severe form of inherited muscular disease whose genetic defect causes a progressive destruction of the skeletal muscle with a subsequent replacement by adipose and fibrous tissue. This altered body composition has been taken into account to explain patients’ tendency to put on weight even though they are on a diet. Anthropometric measures used to calculate common physiological parameters in diets only consider the amount of subcutaneous fat and not the adipose replacement in dystrophic muscles. We propose a rapid and easily applicable MR protocol which gives a quantitative evaluation of the total adipose and lean tissue distribution.

Nine boys affected by DMD, ranging in age from 6 to 12, were selected to undergo MR. Transversal T1-weighted spin-echo sequences (0.5T, TR = 300 ms, TE = 10 ms, thickness = 10 mm, slice-gap = 1 mm) were used for all acquisitions, each consisting of 8 slices and lasting 54 seconds. Whole-body examination needed an average of nine acquisitions. Images were downloaded on to an independent workstation and, through their electronic segmentation with a reference filter, total volume and adipose tissue volumes were calculated.

Our study gave an accurate quantitative evaluation of adipose and muscle tissue volumes in affected children and proved to be rapid and easy to apply. We recommend this method in planning DMD patients’ diets and as a useful instrument to better understand the progression of the disease.

QUANTITATIVE ASSESSMENT OF MUSCLE STRENGTH (QMA) AND MYOTONIA IN MYOTONIC DYSTROPHY TYPE 1 (DM1)

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There is no standardised optimal procedure for documenting muscle strength progression and myotonia in neuromuscular disorders.

The aim of this study was to present a protocol to measure muscle strength and myotonia using quantitative muscle assessment (QMA) measures.

Ten patients with moderately severe myotonic dystrophy type 1 (DM1, E2 expansion range) before and after 1 month mexiletine treatment (200 mg bid) underwent: i) manual muscle testing (MMT) in 15 muscles using the 5-point MRC strength scale and ii) quantitative muscle assessment (QMA) in 11 muscles measuring strength expressed in kg after maximum voluntary contraction (MVC). Myotonia was assessed by: i) subjective self-evaluation scales of severity; ii) timed functional tests; iii) measure of relaxation time by QMA after MVC; iv) measure of relaxation time by EMG.

Our data demonstrate that muscle strength and relaxation time after MVC improve after mexiletine treatment in patients with DM1.

Although preliminary, our data suggest that QMA is a sensitive tool for measuring strength and myotonia. Intra- and interrater reliability measures in a larger sample will also provide useful information on the possibility of using QMA in studies of natural history data and in therapeutic trials.

MUSCLE STRENGTH AND VOCAL EXPLOSION

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In a number of sporting events, it is common to note a vocal utterance of variable intensity and frequency coinciding with the athlete’s intended movement. It occurs at the moment of maximum development of muscular strength, although the exact temporal relationship between the two phenomena is not clear.

Does the vocal utterance represent only a release
phenomenon, or does some mechanism of real interaction, direct or mediated, between the utterance and the sporting action take place? Is it merely a different type of forced expiration, or is it part of more complex activating-inhibitory mechanisms? And, even before answering these questions, is a vocal utterance able to modify muscle strength?

Seven male subjects (age range: 18-23) were studied. They performed maximal activation of the quadriceps muscle, in isometric contraction, for 5 seconds under the following conditions: a) with forced expiration, without vocal utterance; b) with true vocal utterance occurring simultaneously with the start of the exercise; and c) just thinking of uttering at the start of the exercise.

We considered the surface EMG area, and the peak torque and the time to peak torque of the mechanic response. During the test with true vocal utterance, we observed a significant increase in the area of the EMG signal (p<0.04). We also found an upward trend for the peak torque and a downward trend for the time to peak torque.

A study on a larger number of subjects is under way, which may confirm these encouraging preliminary results.

QUALITY IN A NEURORADIOLOGY UNIT

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Quality in a neuroradiology unit is neither absolute nor universal, but derives from several elements that combine to give it its particular dimension.

In this study, the authors analyse its various elements, such as: relational quality, environmental quality, the quality of the image, economic quality, and organisational quality, all of which are conditioned by the fact that the patient is regarded as an out and out customer, with the capacity to make judgements.

Moreover, taking a critical view of their own work, they relate how they approach various problems, bearing in mind that quality of radiological services is determined to a great extent by the behaviour of the equipe, while technical quality contributes to a much lesser degree (in the literature 60% vs 9%, respectively). From this, it is deduced that technical quality and relational quality interact, and often, given that the former (professionality) cannot be eliminated, it is the latter that makes the difference. Human quality will always improve a service.

Quality is therefore an organisational strategy that is created through the development of human resources, remembering that “success is a consequence and not an objective” (Flaubert) and that it must be maintained over time.

MR-RADICULOGRAPHY: RETURN TO THE FUTURE

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Currently, MRI is certainly the most appropriate method for studying lumbar pathology (low back pain). That said, it is not always able to demonstrate without doubt the involvement of the nerve roots. This gives rise to the need to complete examinations with MR-myelography, procedure which has long execution and processing times.

Our aim is to propose a single shot turbo T2 acquisition (FA 160, TR 2800, Mtr 240x256, Th 40, FOV 300, AT 4 sec.) which, thanks to its short execution time, allows us to use the MR-myelography procedure to complete every single examination of the lumbar rachis, hence increasing its diagnostic value. The advantages are mainly: overall view of the nerve roots also in sciotic patients; evaluation of the stenosis of the canal by evaluating cerebrospinal fluid signal alterations; definite demonstration of the presence of nerve root compression.

CLINICALLY SILENT BRAINSTEM HYPERTENSIVE ENCEPHALOPATHY WITH ODEMA OF THE PONS

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Hypertensive encephalopathy is a neurological syndrome that results from a sudden increase in blood pressure. Usually, the symptomatology includes headache, altered mental functioning, focal neurological signs, seizures and cortical blindness. If untreated, hypertensive encephalopathy is progressive and may lead to coma and death. In the early stages, CT and T2-weighted MR images typically show diffuse brain swelling of parieto-occipital cortices. Involvement of other brain regions is relatively rare. Recently, however, isolated brainstem oedema has been reported in 10 patients with hypertensive encephalopathy. We describe a further case of a 36-year-old male who presented with headache and elevated arterial blood pressure (280/180 mmHg). T2-weighted MR scans showed hypertense lesions that were massive in the pons and less evident in temporal, insular, striatal and thalamic regions. Treatment with betablocker labetolol dramatically improved the clinical conditions and MR findings. Although a brainstem glioma was suspected initially, it later became clear that MR abnormalities resulted from oede-
ma that affected predominantly the brainstem but spared, quite peculiarly, posterior cortical regions. This may result from a loss of sympathetic blood flow autoregulation in a small, specific territory of the basilar artery.

INITIAL EXPERIENCE OF CEREBRAL AVM EMBOLISATION WITH ONYX: ANALYSIS OF TREATMENT IN SIX PATIENTS

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The endovascular approach to arteriovenous malformations (AVM) using different embolising agents is a well-established treatment option. The procedure allows complete closure of the malformation in around a quarter of all cases reported in different literature series with significant variability among different authors. The incidence of major complications also varies widely: in our opinion, this is because successful treatment is highly operator-dependent. In turn, this operator-dependency is linked to the use of valid but difficult-to-manage embolising agents, like acrylic glue (Histoacryl). Choice of cyanoacrylate dilution, the speed and technique of glue injection in relation to microcatheter position are empirical skills that are difficult to transmit from one operator to another and often applied on the basis of personal experience. The outcome is that although acrylic glue is a valid embolising agent that has been applied for many years, it remains difficult to use, and involves significant risks for the patient. Another negative feature of the glue is the fact that because of the potential risk of serious complications, acrylic glue manufacturers have not given the product the EU trademark for endovascular use, which is included among the prohibited indications. This situation has recently created difficult working conditions, especially from the legal standpoint, despite widespread application for many years (also acknowledged by the Italian Ministry of Health, which can authorise the use of glue on an individual patient basis).

This situation accounts for the ongoing search for safer alternative embolising agents whose application can be standardised within limits feasible for any interventional procedure.

A new embolising material, Onyx (Onyx System MicroTherapeutics, Inc., MTI), was marketed some years ago as a “foam” with the theoretical feature of progressive occlusion rather than immediate gluing.

We report the results obtained in the first six patients treated with Onyx in the last six months, analysing the application technique and our impressions during treatment in relation to our past experience with cyanoacrylate glue. In one patient we obtained complete angiographic occlusion of the AVM. In another case we reached an 80% occlusion and the patient completed the treatment with surgical approach, without any problem. The other patients are still under treatment.

In our experience Onyx gives good results and it seems easier to use than cyanoacrylate.

TREATMENT OF GIANT INTRACRANIAL ANEURYSMS WITH BALLOONS AN “EVERGREEN” METHOD

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Occlusion of the afferent vessel is a technique commonly used for the treatment of giant intracranial aneurysms. It contributes particularly to the treatment of surgically inaccessible aneurysms, such as those of the cavernous segment of the internal carotid artery, although sacrifice of the internal carotid artery can sometimes lead to major permanent neurological deficits. Endosaccular coil treatment with or without the “remodelling technique” is not indicated in giant or wide-neck aneurysms.

Even with a clinically tolerated temporary balloon occlusion test, the failure rate of subsequent permanent occlusion reported in the literature is about 5%. Moreover, there are potential problems concerning the “safety” of permanent internal carotid artery occlusion and the interpretation of the results.

Our experience, in agreement with literature, prove that silicone balloons are simple, easier nowadays to prepare and less traumatic, even though they are semipermeable and thus capable of deflation.

Taking into account the current safety of detachable balloons, it is possible to use the same materials both for the test and for the permanent occlusion. In this way, having fulfilled the necessary tolerance criteria, permanent occlusion can be performed immediately upon conclusion of the occlusion test.

To stabilise this procedure, permanent occlusion of the parent vessel with a balloon overlying the neck of the aneurysm is followed by the positioning of a second balloon in the petrocarvenous junction of the ICA.

Occlusion of carotid-ophthalmic aneurysms must be performed at the level of the neck of the aneurysm with closure of the ophthalmic artery, which will then be countercurrently reperfused through the anastomosis with the external carotid artery.

We encountered neither clinical complications concerning the visual system nor thrombosis of central artery of the retina.

In order to confirm balloon inflation, patients are followed up both clinically and with serial skull films. The increased use of magnetic resonance imaging, resonance angiography and helical computed tomography
have made these techniques reliable and useful for assessing lack of flow inside the aneurysm and other potential pathologies during the patient’s follow up.

**PSYCHOPHYSIOLOGICAL FOLLOW UP OF A SKILLED MOTOR ACT IN HEMIPARETIC PATIENTS**

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Cortical functions concerned with the execution of skilled movements can be studied through the movement related potentials (MRPs) elicited by complex paradigms. In particular the skilled performance task (SPT) is a complex, interactive, bimanual, sequential task which offers the possibility of analysing both behavioural data (percentage of correct performances) and the electrophysiological components reflecting the phase of pre-programming (Bereitschaftspotential - BP) and the control activity (skilled performance positivity - SPP) of a skilled motor act.

Ten patients with hemiparesis due to cerebral vascular injury underwent SPT in order to evaluate the electrophysiological trend during clinical recovery.

In a one-year period four clinical and electrophysiological evaluations were performed. At the first recording, hemiparetic patients scored a smaller number of correct performances (p<0.03), associated with a lower BP amplitude (p<0.002), than normal subjects. SPP was lacking.

A progressive recovery of BP and SPP amplitude, with an improvement in correct performances, was noted in the subsequent recordings. Moreover, the first recording showed an asymmetric distribution of BP compared with the subsequent evaluations. Our data suggest that the preprogramming and control activity of a skilled motor act has a different recovery trend, possibly reflecting cerebral plastic reorganisation phenomena.

**MUSCLE TONE CHANGES IN RELATIONSHIP TO SLEEP: PHYSIOLOGICAL AND PATHOLOGICAL ASPECTS**

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In physiological conditions during quiet (non-REM) sleep there is a gradual decline in muscle tone. During active (REM) sleep there is a strikingly potent tonic suppression of muscle activity, which often culminates in the complete loss of muscle tone (atonia). In addition, a number of specific patterns of somatomotor suppression occur within the state of REM and non-REM sleep. Some of these patterns of suppression operate in conjunction with the concurrent excitation of motoneurons, whose discharge results in myoclonic twitches and jerks. The two basic mechanisms responsible for atonia during REM sleep are postsynaptic inhibition and disfacilitation (i.e., a reduction in the discharge of tonically active presynaptic excitatory neurons). The two mechanisms may operate continuously, be superimposed on each other at certain times, or act individually for restricted periods.

During sleep several pathological conditions characterised by muscle tone change may be observed: nocturnal cramps, periodic leg movements (PLM), propriospinal myoclonus, REM sleep behaviour disorder (RBD). Nocturnal cramping is a common condition in the elderly and during pregnancy. PLM is described as rhythmical extensions of the big toe and dorsiflexions of the ankle with occasional flexions of the knee and hip (for 0.5 to 5.0 sec) with a frequency of one every 20 to 40 sec (mainly in the first part of the night). Propriospinal myoclonus arising from a spinal generator is observed during drowsiness and it may cause severe initial insomnia. RBD is characterised by the intermittent loss of REM sleep atonia and by the appearance of elaborate motor activity associated with dream mentation. The animal model of RBD indicates that the lesions to the perlocus coeruleus disrupt the excitatory connection to the nucleus reticularis magnocellularis in the descending medullary reticular formation and disable the hyperpolarisation of the alpha spinal motoneurons.

**HAND SURGERY FOR SPASTIC CONTRACTURE**

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We can operate on true spastic patients or patients with a prevalent pyramidal type. It is important that they have an IQ of over 70% in order to be able to collaborate with rehabilitation. We distinguish between a prevalent intrinsic spastic contracture (with thumb deformity in flexion and abduction and swan-neck deformity of the long fingers) and a prevalent extrinsic spastic contracture (with shoulder adduction and internal rotation, elbow flexion, forearm pronation, wrist flexion-ulnarization and finger flexion).

Various surgical techniques can be employed (Zancolli’s muscle-aponeurosis releases, fractioned elongations of flexor tendons, tendon transpositions, tenodesis and...
tenotomy, arthrodesis) depending on the type and severity of the deformity (Zancolli’s classification), functional deficit, patient’s age and surgeon’s experience.

In Zancolli’s type I, complete finger extension is possible if the wrist is in neutral position. We must only correct severe wrist ulnarization. In Zancolli’s type II A, active wrist extension is possible and flexor contracture is slight: muscle-aponeurosis release with or without flexor ulnar carpis (FUC) tenotomy gives satisfactory results. In Zancolli’s type II B we resolve wrist extensor paresis with FUC transposition surgery on extensor radialis brevis carpus and finger flexor retraction with muscle-aponeurosis release or fractioned elongation of flexor tendons. In Zancolli’s type III it is possible to resolve severe wrist flexion with wrist arthrodesis.

A complex surgery (metacarpophalangeal arthrodesis, thenar muscle disconnection, brachio radialis transpositions on extensor longus pollicis, abductor longus pollicis abduction tenodesis and, if necessary, flexor longus pollicis tendon elongation) can resolve flexum-adduced thumb deformity. Swan-neck deformity of the long fingers spontaneously improves with wrist flexion correction. A serious deformity type is corrigeable with superficial flexor tenodesis on the middle joint.

**ORAL CONTRACEPTIVE DRUGS AND CARPAL TUNNEL SYNDROME: RISK OR PROTECTIVE FACTOR?**

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Considering the role of hormones in various conditions (high prevalence in females, pregnancy, obesity, menopause) in which carpal tunnel syndrome (CTS) is present, we investigated the role of oral contraceptive drugs (OCD) in this pathology.

We performed electromyography in 189 females according to the following criteria: 1) female of childbearing age (not pregnant at the time of the study or at least the previous year) 2) absence of obesity (Body Mass Index < 28); 3) absence of endocrine disorders involving sexual hormones; 4) absence of diabetes or other known causes of neuropathy.

We considered two groups: females with carpal tunnel syndrome (CTS) (93 cases, CTS group) and patients without CTS (96 cases, NO-CTS group).

The groups were divided into two subgroups: subgroup A, including females younger than 35 years old, and subgroup B, including those over 35 years. In subgroup A data confirmed a higher percentage of patients without CTS among females taking OCD (58.9% vs 47.3%) and a higher percentage of patients with CTS among females not taking OCD (44.4% vs 19.6%); this difference was found to be statistically significant (chi-squared test: p=0.02). On the other hand in subgroup B we found the same percentage of patients with and without CTS among females taking OCD (26.4% vs 25%).

Our data suggest that in females younger than 35 years OCD may play a protective role as regards the development of CTS. After 35 years this protective role is less evident, probably due to other age-related factors.

**VEGETATIVE STATE: REHABILITATION, REINSERTION IN THE FAMILY SETTING AND COSTS**

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We conducted a ten-year retrospective study (January 1990 - December 2000) of patients in a vegetative state (VS) in the rehabilitation unit of our hospital. VS is a clinical condition of complete unawareness of self and surroundings accompanied by natural sleep-wake cycles with full or partial maintenance of autonomic hypothalamic and brainstem functions. In all cases we examined personal data, aetiology, length of hospital stay, type of rehabilitation, evolution and survival over one year after injury, current placement of surviving patients and healthcare costs.

We studied 38 patients, 27 males and 11 females, aged between 16 and 86 years (average 54.6). The aetiology was traumatic in 14 cases, anoxic in 10 and vascular/tumoral in 14. Hospitalisation ranged from 1 to 41 months amounting to a total of 19,301 days: 7,095 days (36.3%) for post-traumatic patients, 5,409 (28%) for post-anoxic cases and 6,797 (35.7%) for post-vascular injury.

During their stay in hospital all patients received early neuromotor rehabilitation and sensory stimulation. Sixteen patients died whereas 22 are still alive (14 in a permanent VS, 7 in minimally conscious state and 1 in a locked-in syndrome). In particular, 3 post-traumatic cases died and 5 evolved into a minimally conscious state within a year of injury except one; the remaining 6 are in a permanent VS. Of the post-anoxic cases, 6 died and 4 are in a permanent VS. Of the post-vascular cases, 7 died, 4 are in a permanent VS, 2 evolved into a minimally conscious state within three months after injury and 1 into a locked-in syndrome within 6 months.

Of the 29 surviving patients, 16 are in a permanent VS, 2 of whom have an exceptionally long survival after injury of 9 and 10 years. Seven are still at our hospital, 5 are hospitalised elsewhere and 10 are cared for at home.

The cost of acute hospital care ranged from a daily
cost of approximately EUR 516.5 to EUR 154,937 for a year’s hospital stay. Subsequently the cost was reduced to care and accommodation expenses, around EUR 186 a day for a total of around EUR 67,140 a year. This sum is the same for hospital and domiciliary care.

**MOTOR CONTROL AND THE CORPUS CALLOSUM**

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In recent years, we have studied a series of partial and total callosotomy patients in motor tasks ranging from simple visuomotor integration to praxis and tactile exploration. The patients were operated on either in Rome (Prof. Rossi) or in Ancona (Prof. Papo, Prof. Ducati) with the aim of reducing drug-refractory epileptic seizures.

Interhemispheric visuomotor integration can be accomplished in 2–6 ms in the normal brain (interhemispheric transmission time, IHTT). IHTT rises to 100 ms in total callosotomy patients, both in unilateral and bilateral distal movements as well as in unilateral proximal movements, while there is no increase in bilateral proximal and in axial movements. Synchronization of bilateral movements is normal. No partial sections excluding the splenium increase IHTT.

The praxic deficit in total callosotomy patients is peculiar insofar as: 1) it is limited to the left hand and 2) imitation is always successful. In contrast with classical neurological notions, both hemispheres of the callosotomized brain can control the praxis of contralateral limbs, and left dyspraxia depends on the incapability of the right hemisphere to understand verbal commands and/or translate them into appropriate motor acts. The exclusion of the right hemisphere from language is even more striking in tactile naming tasks: total callosotomy patients are unable to produce names or even to describe the use of the objects explored using their left hand with full dexterity.

**CLINICAL AND IMMUNOLOGICAL PROFILE OF PATIENTS WITH AUTOIMMUNE ATAXIA**

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Autoimmune ataxia is usually reported associated with gynecological tumours, such as paraneoplastic disorder. There have recently been reports of a few patients with cerebellar ataxia associated with antibodies to glutamatic acid decarboxylase (GAD-Ab). We describe 4 patients with cerebellar ataxia whose sera harbored GAD-Ab as demonstrated by immunohistochemistry on rat cerebellum; the specificity was confirmed by radioimmunoassays (RIA) and by immunoblot of recombinant human GAD65. Three patients were women, with late onset IDDM and personal or family histories of other autoimmune disorders. All the patients developed gait ataxia, limb ataxia, nystagmus and dysarthria. Extracerebellar signs included localized stiff-person syndrome (1 patient) and peripheral neuropathy (1 patient). All the patients had oligoclonal IgG bands in the CSF. Radiological studies showed no brainstem involvement, but 2 patients had pure cerebellar atrophy. Upon RIA, the GAD-Ab levels were found to be similar to those associated with stiff-person syndrome and higher than those associated with polyendocrine autoimmunity without neurological disease. Recombinant GAD-65 protein was recognized by immunoblot of the sera of patients and intrathecal GAD-Ab synthesis was observed in the two patients studied. These results suggest a link between high level of GAD-Ab and some cases of idiopathic cerebellar ataxia, particularly in women with other autoimmune disorders.

**MYOCLONUS OF THE FLOOR OF THE MOUTH WITH ANTI-GAD ANTIBODIES IN AUTOIMMUNE DIABETES AND THYROIDITIS**

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Palatal myoclonus (or tremor) occurs usually in association with myoclonus of branchial muscles (usually up to C5 innervated muscles). Pathologically, the inferior olive nucleus becomes hypertrophic probably because of continuous neuronal discharge that is consequent to the loss of an inhibitory input from the dentate-rubro-olivary pathway. Palatal myoclonus can be idiopathic or secondary but, to date, there is only one report of anti-GAD antibodies in palatal myoclonus. We observed myoclonus of the floor of the mouth in a 39-year-old man suffering from autoimmune diabetes and thyroiditis. Neuropsychological investigations showed that the myoclonus was rhythmic with a 2–2.5 Hz frequency and that the masseter reflex was lost. Immunological investigations showed oligoclonal band in the cerebrospinal fluid and anti-GAD antibodies in blood serum. After clonazepam 1 mg/day the myoclonus disappeared. For this patient, we hypothesize the occurrence of a limited somatotopic impairment of the gabaergic inhibitory...
pathway that projects from Purkinje cells to the dentate nucleus.

**CROSSED CEREBELLAR HEMIATROPHY**

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We report the case of a 44-year-old male patient, affected by the syndrome first described by Gastaut as “HHE”, that is hemiconvulsion, hemiplegia, epilepsy. The syndrome starts with febrile convulsive seizures, which are so severe as, by themselves, to cause brain damage. As a consequence, an hemiplegia and a new form of secondary epilepsy arise.

A brain MR scan was performed, which demonstrated an astonishing atrophy of the left cerebral hemisphere. An interesting MR finding was a remarkable right cerebellar atrophy; furthermore, an MR T2-elongation of the gray matter of the cortex and dentatus nucleus of the right cerebellar hemisphere, consistent with the diagnosis of gliosis, was evident. The observation of contralateral cerebellar atrophy in cerebral hemiatrophy has been reported in pathological papers, and it is presumed to be due to retrograde transsynaptic degeneration within the cerebello-rubro-thalamic tract. Only a few neuroradiological communications have notified a crossed cerebellar hemiatrophy, but never an MR signal alteration of the cerebellar cortex.

We considered it would be interesting to verify whether the described images could actually correlate with an objective deficit. Considering the patient’s severe right spastic hemiplegia due to the left cerebral damage, the standard clinical tests to assess cerebellar functions could not be applied, as an eventual homolateral cerebellar syndrome would not be recognised. To overcome this problem, we decided to test the patient’s saccadic ocular movements, which demonstrated an accuracy impairment of cerebellar origin. So, although not clearly asserted in literature so far, this degeneration did provoke a functional deficit, clearly proved by neurophysiological tests.