

The 2nd World Congress of the International Society of Physical and Rehabilitation Medicine

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Transcranial magnetic stimulation in rehabilitation medicine

Transcranial Magnetic Stimulation (TMS) is beginning to be used for therapeutic purposes in rehabilitation, as well as for assessment.

S. Wolf *et al* (USA) and M. Lissens (Belgium) reviewed the latest developments regarding the clinical applications of TMS. S Wolf *et al* presented TMS applications relevant to stroke rehabilitation. The emergence of TMS for mapping cortical motor areas and Motor Evoked Potentials has made it possible to identify marked plasticity in the motor cortex and to evaluate the extent of motor recovery in stroke patients after rehabilitation. Using this technique, the group reported the effect of intense physical therapy and Constraint Induced therapy on cortical plasticity. Furthermore, TMS is useful in predicting the prognosis of neurologic recovery and thus helping in patient selection for therapy to improve outcome with stroke rehabilitation.

The Belgium group presented an application of TMS, not only in motor recovery and prediction of prognosis, but also in predicting the presence of reflex sympathetic dystrophy and measuring significant defect in central conduction for neurogenic respiratory failure.

A group from Japan presented a case study of 47 year old man with chronic right hemiplegia following left putaminal haemorrhage. He had an improvement in hand function after therapy with TMS.

Neuropathic pain

Dr A A Fisher *et al* from the USA presented an improved technique for the treatment of neuropathic pain. They suggested paraspinal block (PSB) as a specific therapeutic modality in treating patients with neuropathic pain who failed to respond to other conventional therapies. Spinal segmental sensitisation (SSS) is a consistent phenomenon present in patients with neuropathic pain. SSS was diagnosed by the presence of dermatomal, myotomal hyperalgesia and sympathetic abnormalities. They proposed that PSB alleviated pain by reversing SSS to normal sensitivity.

Their prospective study comprised 36 patients with chronic neuropathic pain but with different underlying causes. Half had CRPS and the rest suffered one of the diagnoses of phantom and stump pain, spinal cord injury pain, traumatic brain injury, post herpetic neuralgia or incision neuroma. Patients received weekly intervals of PSB with 1% Lidocaine aiming into the posterior primary ramus at the level of SSS. The mean number of PSB needed was 2.5+/-1.6 and dermatomal and myotomal hypersensitivities were markedly reduced. The authors concluded that pain reduction was still significant 5.9 months follow-up after the treatment and allowed patients to increase participation in rehabilitation programmes.

Psychopharmacology in the treatment of agitation after acquired brain injury

Controlling psychomotor agitation can be extremely difficult in patients with brain injury, either at the acute or chronic phase. Trying to balance between using sedative medication and their negative effects on cognition is a challenge in this patient group. A group from Italy presented the use of olanzapine in agitated behaviour after traumatic brain injury. Due to its widespread receptor

blocking mechanism on dopamine, serotonin, muscarinic, alpha adrenergic receptors, and its atypical neuroleptic action with less extrapyramidal side-effects, olanzapine was chosen to control post traumatic (brain injury) psychomotor agitation.

Six young male patients (mean age 35 +/- 15 yrs) with GCS <7 had variable dose of olanzapine (range between 2.5 – 12.5 mg/day) at least for six months. Among them, five patients had post traumatic brain injury and the one subarachnoid haemorrhage. All needed olanzapine once they regained consciousness due to an immediate presentation of psychomotor agitation. The extent of agitation was assessed by ABS (Agitated Behaviour Scale).

The authors concluded that despite the need of further higher evidence based research like RCT, olanzapine proved to be effective and tolerable in severe brain injury of both traumatic and non-traumatic origin. Olanzapine did not cause a marked sedation, although it was started at a low dose of 2.5 mg/day and increased slowly later on. It also restored a better sleep/wake pattern to the patients during early PTA (Post Traumatic Amnesia).

Scientific basis for spasticity outcome measures

Mr Johnson from the UK presented a paper aimed at increasing reliability and validity in measuring spasticity. The author combined clinical Modified Ashworth Scale (MAS) and biomechanical techniques. It was pointed out how difficult it is to measure pure reflex component of spasticity without passive soft tissue visco-elasticity properties which might be involved in the development of contractures clinically. MAS is not completely reliable and valid due to inconsistencies in the hierarchy of its points, and it cannot solely measure reflex component of spasticity. The author's biomechanical principle was based on reflex excitability and measuring reflex amplitude, latency and duration. It was tested in 14 stroke patients and 17 normal subjects. The reduced latency indicated an increased motor neurone excitability but the amplitude reduction was an unexpected result. The author concluded by pointing out the lack of clinical scales and benefit of adding biomechanical techniques in measuring "true" spasticity.

The 2nd World Congress of the ISPRM presented a variety of topics in rehabilitation medicine. Innovative diversities, outcome measures and specific interventions were presented in branches of rehabilitation medicine such as brain and spinal cord injuries, stroke rehabilitation, orthopaedic and musculoskeletal disorders as well as special groups such as geriatric and paediatric populations. I look forward to the next congress of the ISPRM in Brazil in 2005.

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