

European Federation of Neurological Societies

30 August - 2 September, 2003; Helsinki, Finland

The 7th Congress of the EFNS was held at the Helsinki Fair Centre in Helsinki, Finland and large numbers of participants came from all over Europe, as well as Australia and the US.

It is always difficult to comment on the overall impact of the meeting in terms of new discoveries and changes in neurological practice given the large number of presentations and parallel sessions that are run. The various items that caught our particular eyes were:

- The work of Ken Smith (London) demonstrating cogently that axonal loss in multiple sclerosis may result from abnormalities in sodium loading which in turn leads to changes in intracellular calcium as a result of a reverse of a sodium calcium exchange pump. Blocking this influx of sodium may, therefore, have an impact upon axonal integrity and Ken Smith presented interesting experimental work not only *in vitro* but in the EAE model. This is leading to the possibility of a drug trial in MS looking at flecainide and its effect on axonal loss.

- Field of movement disorders. There was a very lucid account by Cristina Sampaio (Lisbon) on the evidence for the use of various drug therapies in Parkinson's disease. This was a sobering experience as it is quite clear that many of the drug therapies have no actual basis in terms of proper controlled trials. However, it emerged from this work that the efficacy of deep brain stimulation is probably very similar to that of apomorphine infusions and thus abandoning pharmacological therapies in favour of deep brain stimulation in advanced PD patients might not necessarily be always the right decision.

- Wolfgang Oertel (Marburg) gave a very comprehensive account of the incidence of sleep disorders including restless leg syndrome and its association with Parkinson's disease both pathologically and physiologically. The basic message from this talk was that these conditions are very common and that there is probably a degree of overlap between REM sleep disorder, Parkinson's disease and restless leg syndrome in terms of pathophysiological behaviour given their sensitivity to dopaminergic agents.

- There was also a fascinating satellite meeting, which looked at the evidence of neuroprotection with dopamine agonists in early Parkinson's disease. This explored not only possible *in vitro* mechanisms for this (stabilisation of mitochondrial membrane potential) but the recent clinical evidence that this can be seen in patients. This latter area relies heavily on functional imaging, and many intriguing questions were raised including whether levodopa interferes with dopamine uptake and by so doing influencing the scan result. If true, then the neuroprotection of dopamine agonists may turn out to be an artefact created by an apparent worsening in signal by L-dopa.

- At the Basal Ganglia Club meeting there was a superbly comprehensive and authoritative account on the aetiology and management of dystonia by Jo Jankovic. The major messages from his talk were: any child with cerebral palsy should be given a trial of levodopa (which many now adopt in their standard clinical practice already); polypharmacy for dystonia is often necessary and that surgical interventions are emerging as a very useful therapy in people with severe generalised dystonias especially if they carry the DYT1 gene.

- There was also a fascinating account by Mark Edwards (London) demonstrating that DYT1 gene carriers have abnormal neurophysiology within the cortex similar to that seen in the manifest patient. This suggests that people with

the DYT1 gene have abnormalities of cortical excitability but that some other trigger is necessary for this to become expressed clinically - possibly trauma. In this respect there was a most entertaining and illuminating lecture on the focal dystonia of Robert Schumann which also involved the playing of some of his pieces of music. This presentation by Eckart Altenmüller (Bern), also included a brief discussion on the thwarted solo career of Scriabin who appeared to have developed a myofascial syndrome of his right arm which prevented him from playing the piano and led to him increasingly composing pieces of music, for the left hand. This led on to discussions about the origin of such task specific dystonias, the consensus being that there are disturbances in sensory motor coupling within the somatosensory motor cortices and that the blurring of cortical representations of adjacent body causes the dystonia. This has led to the advent of constraint therapy as a way of trying to re-organise sensory information and mapping and by so doing should help treat some forms of dystonia. Some clearly believe this is useful (e.g. Priori - Milan), whilst others do not (Jankovic - USA).

- Martin Farlow (Indianapolis) presented the results of a placebo-controlled trial of the NMDA receptor antagonist memantine (Ebixa) in over 400 patients with moderate to severe Alzheimer's disease (AD; MMSE 5-14) already receiving stable doses of the cholinesterase inhibitor donepezil. The combination was well tolerated (better than placebo) and by 24 weeks there was significant improvement in cognition and less decline in function. Memantine/donepezil combination therapy may therefore be an option for AD in the future.

- Triau (Leuven) discussed some results from the AWARE ("Aricept washout and rechallenge") study in which patients with AD who seemed not to respond to 24 weeks of donepezil (single-blind treatment) were randomised to either withdrawal or continuation of therapy (double-blind). Cognitive, functional and behavioural benefit was found in 75% of those continuing as compared to the withdrawals. A small proportion of patients on continuing therapy did show further cognitive decline but had functional and behavioural improvement. Hence, patients showing an initial decline on donepezil treatment may still derive benefit from continued therapy.

- James Callaway (San Diego) discussed the past, present and future of vaccination therapy for AD, or, as the company (Wyeth-Elan) prefers to call it, immunotherapy. Immunological and neuropathological study of the cases of meningoencephalitis which brought the phase II trial of AN1792 to a premature close suggests that an aberrant T-cell response to amyloid beta-peptide was the cause of the side effects. Reduction in cortical amyloid plaques was seen in one patient who died after experiencing side effects from immunotherapy. Clinical trials using immunoconjugates or modified peptides which eliminate the T-cell response to amyloid beta-peptide are planned for the near future.

In addition to the intellectual fare, the congress also offered cultural stimulation. A judicious choice of sessions provided music by Schubert (arr. Liszt), in addition to Schumann and Scriabin ("Music and Neurology") as mentioned above, and by Prokofiev, as well as an historical review of Finnish contributions to neuroscience ("History of Neurology").

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Venue: Helsinki, Finland