

Association of British Neurologists, Spring Meeting

14-16 April, 2004; London, UK

The ABN spring meeting 2004 was held at the impressive Church House Conference Centre, tucked away near Westminster, London. The opening lecture was from Professor Pamela Shaw on the latest developments in Motor Neurone Disease. In a nicely structured talk, Professor Shaw beautifully encapsulated how genetic advances and animal models may improve our understanding and treatment of this devastating disorder, "sandwiched" between a focused and relevant clinical overview.

The remainder of the conference comprised scientific sessions and posters, covering a wide variety of specialist areas. The first afternoon session was dedicated to epilepsy, myasthenia gravis and motor neurone disease. An interesting trial of a novel therapeutic compound in myasthenia gravis was presented by D McKee (Manchester). The antisense oligodeoxynucleotide, EN101, binds to acetylcholinesterase mRNA to prevent its translation into protein. The agent appeared to improve symptoms with few side effects in 14 of 16 patients with myasthenia gravis, in whom pyridostigmine had been discontinued for the trial. This drug obviously needs further testing in a randomised controlled trial to evaluate its role in the management of myasthenia but early data seem promising.

Thursday morning presentations covered multiple sclerosis (MS) and movement disorders. Interferon and glatiramer are available on the NHS, conditional on long-term data collection by the MS monitoring study. Currently 4,000 people with MS in the UK are prescribed disease modifying therapies (approximately 10% of patients), although there is significant variability between centres. 3,500 of these are in the current study. Issues that have arisen from the initial clinical and demographic data were presented (C Cooper). The development of antibodies to interferon reduces the efficacy of the drug and occurs in approximately 10-15% of treated patients. Inter- and intra-rater reliability for the EDSS was assessed and showed moderately good consistency. There are no natural history controls in the study and it would be unethical to deny suitable patients this treatment, so unfortunately historical controls will be used. More data is needed on cost effectiveness and progress post-treatment. These drugs are effective against the inflammatory component which causes acute events in MS. Axonal degeneration also causes significant disability and to date has been resistant to treatment. Some research was presented from Kings College, London (DA Bechtold) looking at the effects of anticonvulsants on axonal protection in rats with experimental autoimmune encephalomyelitis (an animal model of MS). Carbamazepine showed no positive effects and phenytoin caused only a modest improvement, while lamotrigine appeared to show significant protection from axonal degeneration. Clearly, these promising results need to be replicated in human subjects. On a related theme, E Lim (London) presented her work in MS patients, suggesting that the release of neurofilament heavy chains in the CSF associates with axonal loss, while higher levels of neurofilament following an acute relapse may be predictive of a poor outcome in clinically definite MS cases. Dr L Teare (Plymouth) also presented intriguing preliminary data in this session to hypothesise that cannabinoids may have neuroprotective effects in MS.

The second session covered movement disorders and included a fascinating presentation on punding in Parkinson's disease (PD) (A Evans, London). Punding is a prolonged complex purposeless stereotyped behaviour, originally described in chronic amphetamine users. Punding PD patients were more likely to be taking higher doses of dopaminergic treatment, suffer from insomnia, and take nocturnal doses of medication, compared with non-pun-

ders, suggesting a relationship with the dopamine dysregulation syndrome. The pathophysiology underpinning dyskinesias in PD was elegantly discussed by M Silverdale (Manchester), who proposed a central role for over-activity of AMPA receptors in mediating this motor complication, via increased receptor trafficking into the post-synaptic membrane. Other interesting developments included the prospect of diffusion tensor MRI being of diagnostic utility in differentiating PSP and MSA (C Blain, London) and the intriguing observation of a very high frequency of anti-basal ganglia antibodies in adults with atypical dystonia and tics, when compared with a number of control groups, including primary torsion dystonia (M Edwards, London).

The afternoon session was short to allow time for the annual general meeting (or shopping, in the case of those drawn in by the bright lights and big city). The proposal of intradetrusor botulinum toxin injections to treat the symptoms of severe detrusor muscle instability may be a promising therapeutic option (C Fowler, London). The Leeds study of dementia in PD, impressive for its lengthy follow up of the original cohort, emphasised that the main predictors for cognitive decline are age at entry to the study, lack of tremor and presence of gait disorder (E Dunn, Leeds).

Those who managed to drag themselves out of bed on the morning of the last day, after the excesses of the multi course dinner at the Savoy, were treated to some interesting talks on vascular disease, including the identification that treating TIA as a medical emergency is an economically viable consideration, with medical therapy administered within 14 days proving highly cost effective (S Alder, Sheffield). The proposal that endovascular carotid artery stenosis treatment is as safe as surgical intervention (L Coward, London) requires further supportive evidence, on the basis of a Cochrane review. Anti-phospholipid antibodies can cause a variety of movement disorders, including chorea, often affecting the mouth, tics and myoclonus. Two of six patients improved with oral anticoagulation, which should be considered in the treatment of this disorder (D Martino, London).

A study of the effectiveness of liaison neurology in an acute medical admissions unit (R Forbes, Belfast) identified benefit in terms of shorter hospital stays, while significant changes in diagnosis were made in 34% of cases assessed by the neurologist. The importance of recognising tetanus in intravenous drug abusers was highlighted (K Gormley, Devon & Exeter). Phil Smith (Cardiff) gave an entertaining resume of that elusive strategy to be adopted in creating an award winning poster at scientific meetings (Blackpool here we come!).

An extra presentation was included, documenting a mitochondrial mutation responsible for PARK 6, a hereditary form of Parkinson's Disease described in a large consanguineous family from Sicily (M Muqit, London). The PINK 1 gene encodes for a mitochondrial protein, thought to protect against stress-induced mitochondrial dysfunction. A causative mutation has been found in this gene which leads to disruption of this protective effect. PINK 1 is the first mitochondrial gene to be directly implicated in the pathogenesis of PD.

Overall the meeting was well attended and the standard of presentations was very high. It was useful to catch up, not only with colleagues, but also with the wide range of interesting research being undertaken in the UK. The organisation was superb, and thanks must go to the local organisers, headed by Professor LJ Findley.

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Church House Conference Centre, Westminster, London.