

# The Muscular Dystrophies

## A meeting organised by the Medical Genetics Section of the Royal Society of Medicine

19 November, 2007; London, UK.



Inclement weather and London's notorious public transport system did not deter over 100 clinicians and scientists from attending this meeting. The morning sessions were chaired by Lord Walton of Detchant, an inspired choice given his personal contribution in this field, including (the audience was reminded) the first description of Duchenne muscular dystrophy (DMD) in an individual with Turner's syndrome. He excused himself from the afternoon session, as he was speaking at the House of Lords on the Human Fertilisation and Embryology Bill – a reflection of the rapid expansion of knowledge and technologies in Medical Genetics, and the consequent ethical and legal ramifications. Indeed the ethical considerations surrounding the diagnosis and management of these disorders were highlighted by Professor Alan Emery (Oxford), the organiser of the meeting who also kicked off the talks. He talked movingly of the sometimes tragic implications of establishing these diagnoses to patients and families. Professor Emery gave an overview of the explosion of knowledge there had been in the two decades since the discovery of dystrophin. Currently, there are 40 or so genes associated with the muscular dystrophies, encoding proteins many of which are now known to interlink. He pointed out that questions remained, including the explanations for phenotypic heterogeneity associated with single genes (or even single mutations) and allelic heterogeneity (where mutations in different genes result in an identical clinical phenotype). He talked also of the possible interactions of particular environmental agents (especially pathogens) with specific proteins involved in certain muscular dystrophies. He left the audience with the salutary reminder of the dangers of following dogma (for example the current view that abnormal genes equal disease) in attempting to overcome any challenge, citing the explosion of the Hindenburg in 1937 and the subsequent abandonment of airships - which up to that time had been the preferred means of transatlantic air travel.

Professor Francesco Muntoni (London) in a scintillating talk described the congenital muscular dystrophies (CMDs), which generally present before six months of age. It was interesting to note the extent of central nervous system involvement in some forms, reflecting expression of some proteins in the brain during development. He highlighted three disorders. Ullrich variant and merosin deficient CMD are associated with deficiency of two extracellular matrix proteins (collagen VI and laminin  $\alpha 2$  respectively), and are phenotypically reasonably distinct. Glycosylation of alpha dystroglycan, a peripheral membrane protein, enables it to interact with extracellular matrix proteins,

and abnormal glycosylation (associated to-date with mutations in six separate genes) results in conditions such as Walker Warburg syndrome, Fukuyama muscular dystrophy and muscle eye brain disease. Whilst initially good correlation between specific gene mutations and clinical syndromes was suspected, Professor Muntoni's group has demonstrated that each of the six genes can result in a highly variable phenotype, including (confusingly) adult-onset limb-girdle muscular dystrophy (for example FKR1P).

In a commendably clinically orientated talk, Dr David Hilton-Jones (Oxford) covered Duchenne and Becker muscular dystrophies. Controversially he said that an Italian, Conte, should be credited with the first description of DMD in 1836, preceding even Meryon. This was firmly rebutted by Professor Emery at the end of Dr Hilton-Jones' talk, who maintained that the true credit should go to Meryon, as the latter's description in 1851 not only preceded Duchenne's but it included the key observations that it was maternally inherited and was primarily a disorder of muscle. DMD and Becker muscular dystrophy (BMD) are allelic disorders, and whilst 'out-of-frame' mutations resulting in truncated dystrophin cause DMD, 'in-frame' mutations cause BMD. Clinical 'gems' to take away included the observation that any boy with delayed motor, speech or general intellectual development should have his creatine kinase (CK) measured. Management with (non-invasive) ventilatory support can enable survival into the third and fourth decades. The use of drugs to manage cardiomyopathy and corticosteroids to prolong survival were mentioned. Clinical features associated with the milder BMD include exercise-induced cramp (resembling McArdle's disease) and asymmetric calf hypertrophy. It can be difficult to discriminate BMD from spinal muscular atrophy, and 10% of subjects are wheelchair-bound by 40 years. Cardiomyopathy again requires monitoring and treatment. Finally, Dr Hilton-Jones covered the specific issues of manifesting carriers (associated with non-random X inactivation), and the disorders associated with point mutations in dystrophin ranging from muscle pain and cramps without weakness to isolated cardiomyopathy and isolated 'hyperCKaemia'.

Professor Kate Bushby (Newcastle) talked on the limb-girdle muscular dystrophies (LGMD). Autosomal dominant and recessive forms exist, and whilst clinically (and genetically) heterogeneous all share a predominant pattern of proximal myopathy. It was explained that determining the exact type (if possible) was important for genetic counselling and determining prognosis, as different forms were associated with variable involvement of the cardiac and respiratory systems. The exact diagnosis could only be

achieved by the synthesis of accurate clinical data, muscle biopsy findings and selected genetic studies, and the need for close collaboration with national centres of expertise (such as Professor Bushby's) was readily apparent.

The afternoon session began with another authority in his field, Professor Padberg (Nijmegen) talking on fascioscapulohumeral muscular dystrophy (FSHD). This autosomal dominant disorder is the third most common muscular dystrophy (after myotonic dystrophy and DMD/BMD), but more than 30% of gene carriers (females more than males) may be asymptomatic. (Age of onset is older in females too.) There is often asymmetrical and frequently unrecognised facial weakness first. At clinical presentation, however, shoulder-girdle weakness is common (80%), while foot extensor (10%), pelvic-girdle (5%) and facial muscle (5%) weakness are less so. Disease progression slows in the sixth decade, but at 60 years two-thirds have foot extensor weakness, half have pelvic-girdle weakness and 20% are wheelchair dependent outdoors. Coats' disease, a retinal vasculopathy (often subclinical), can be present, and hearing loss and epilepsy have been described. Professor Padberg next considered the molecular genetic basis of FSHD. It is associated with a reduction in the number of 3.3 kb repeats (normally more than 11) in the D4Z4 locus on 4q35. Whether a transcript (DUX4) or an effect on an upstream gene (FRG1) mediates disease is a matter of debate, but epigenetic mechanisms including hypomethylation of the repeat unit seem to be implicated in the pathophysiology.

Professor Glen Morris (Oswestry) next talked about Emery-Dreifuss muscular dystrophy (EDMD), which is caused by mutations affecting the genes encoding emerin (X-linked), lamin A/C, or the nesprins. Crucially, all three proteins are co-located in the nuclear membrane. Clinically EDMD is characterised by early contractures (Achilles tendons, elbows and neck), muscle wasting and weakness initially in proximal upper limbs and distal lower limbs, and cardiac conduction defects. The importance of the insertion of cardiac pacemakers and defibrillators in management was stressed. Lamin A/C mutations (associated with dominant EDMD) can cause dilated cardiomyopathy necessitating cardiac transplantation, and provides a further example of phenotypic heterogeneity - being associated with a form of CMD and one type of LGMD, both complicated by cardiac disorders too.

Professor Bjarne Udd (Tampere) covered the confusing area of the distal muscular dystrophies. Once again, rapid growth in knowledge has taken place and more than 16 genetically distinct types are now recognised. About half cause distal disease exclusively, whilst the other

half can be associated with scapuloperoneal, proximal or generalised phenotypes. Most of these genes appear to encode sarcomeric proteins, as do the recently identified genes associated with distal arthrogryposis (which is probably a manifestation of congenital distal myopathy). The highly selective nature of muscle involvement is striking, and is even better appreciated by magnetic resonance studies.

Finally, in this section, Dr Gurman Pall (Glasgow) spoke on myotonic dystrophy (DM1). He explained that the triplet-repeat expansion characteristic of the disease produced mRNA containing expanded CUG repeats which are 'toxic' to cells. Dr Pall and colleagues have identified short (CUG)<sub>n</sub>-RNA fragments in cells expressing mRNA containing expansions characteristic of DM1, which may represent an intermediate degradation product of the 'toxic' repeat-containing transcripts. Characterisation of this potential decay pathway may obviously yield opportunities for ther-

apeutic interventions in DM1.

In the last part of the meeting, Professor Kay Davies (Oxford) and Dr Jennifer Morgan (London) tackled the treatment of DMD, addressing gene and stem cell therapies respectively. There seem to be numerous (some very ingenious) interventions capable of correcting or ameliorating the absence of dystrophin, and it may be that a combination of approaches will need to be utilised eventually. It was heartening to note the phase I/II trials in planning or progress, and Professor Davies' view that there was "great promise...(for the successful treatment of DMD)...in the next decade".

All in all, this was an outstanding meeting, bringing together a faculty of true opinion leaders in their respective specialities. For workers in the field there were unrivalled opportunities for networking and sharing recent knowledge behind the scenes, and in this regard it resembled an international symposium. I found it of enormous educational value, and felt that

the field (with only a few exceptions, such as oculopharyngeal muscular dystrophy) had been comprehensively covered. Neurologists in training, especially those with an interest in the genetic aspects of disease are encouraged to join the Medical Genetics Section of the RSM, which is one of its youngest Sections and, on the evidence of this meeting, also one of its most dynamic.

I am grateful to Professor Emery for reviewing the manuscript.

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#### References

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## Society for Research and Rehabilitation Winter Meeting 2008

15 January, 2008; Oxford, UK.

The SRR Winter conference was held on the 15th January 2008 at Oxford Brookes University, hosted by Dr Helen Dawes. Despite only being a one day conference there was a wide range of material presented: free research papers, poster presentations, work in process posters and three symposia. The day's proceedings were started by Professor Paul Matthews' interesting symposium on the utility of different brain imaging techniques to explore the relationship between brain plasticity and rehabilitation.

After a quick coffee and the first chance to view the posters, Professor Cath Sackley gave the first of her presentations on a cluster randomised controlled trial of physiotherapy and occupational therapy intervention to enhance mobility and activity in care home residents. The first session also included a qualitative paper examining experiences of an Exercise Referral Scheme from the perspective of people with chronic stroke presented by Helen Sharma, a randomised single blind trial of the use of multi-sensory stimulation to improve functional performance in older people with dementia, by Dr Lesley Collier, and a survey of the circumstances surrounding falls among people with Parkinson's disease by Professor Ann Ashburn.

Following lunch, Professor Derick Wade gave a thought-provoking symposium exploring how, paradoxically, rehabilitation may prolong disability and illness behaviours, which generated an interesting discussion. This was followed by an interesting and entertaining symposium by Dr Tom Manly on how investigating variability within impairment measures can be utilised in rehabilitation for examining attention and executive function.



During the coffee break delegates had their final chance to view both the posters presentations and the work in progress posters: Poster presentations included: lower limb muscle weakness in Huntington's disease by Dr Monica Busse; walking and wheelchair navigation in stroke patients with left sided visual neglect by Mrs Kelly O'Leary; the 'Dark Art' of physiotherapy: experiences of people with cerebellar ataxia by Elizabeth Cassidy; modulating performance on wheelchair navigation in patients with unilateral neglect following stroke by Dr David Punt; self-optimisation of walking speed following stroke by Dr Johnny Collett; modeling recovery after stroke, Ms Shweta Malhotra; can botulinum toxin, administered in the early stages following a stroke help the recovery of arm function; estimating effect size from a phase II pilot study by Ms Elizabeth Cousins. Work in progress posters included: examining outcome measures of inpatient care in profound brain injury; economic analysis of return to work after traumatic brain injury; reintegration outcomes following spinal cord injury; and the feasibility of NIRS in detecting neural activation in the DLP cortex

during cognitive tasks.

The day concluded with final free research presentations. Dr John Saxton presented his work on the physiological responses to treadmill walking with Nordic poles in patients with intermittent claudication, Mr Atzori his study of concurrent validity of the IDEEA activity monitor to quantify mobility related activities among people with stroke in free-living conditions, Dr Eimear Smith on examination of the prevalence of low bone mineral density in patients at a national rehabilitation centre and we finished where we started with Dr Cath Sackley talking about a phase II randomised controlled trial of bilateral limb movement exercise in chronic hemiparetic stroke patients.

Many thanks to all the presenters and delegates for an interesting, informative and thought-provoking day. The Summer Meeting will be held in Preston, hosted by Professor Caroline Watkins of the University of Central Lancashire.

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# The Parkinson's Disease Non-Motor Group 3rd Annual Meeting

8 March, 2008; London, UK.

The Parkinson's Disease Non-Motor Group (PDNMG) 3rd annual meeting was held at the Royal Society of Medicine, London, on 8th March 2008 and was attended by 170 health care professionals. It provided an opportunity to review the past year's progress in the field of non-motor symptoms (NMS) and another informative and invaluable day of learning.

Attendees were welcomed by PDNMG Chairman, K Ray Chaudhuri, who briefly discussed the prevalence of non-motor symptoms (NMS) including dribbling saliva, constipation, depression, sleep disorders, apathy, hallucinations and dementia which complicates the lives of people with Parkinson's disease (PD). Professor E Tolosa (Barcelona) described the pre-clinical phase of PD. He examined the correlation between Braak staging with clinical manifestations, imaging of the substantia nigra, and non-motor symptoms including olfactory disturbances, depression and autonomic system disorders. Professor Tolosa suggested that NMS are not prodromal but in fact part of PD. Professor A Schapira (London) then delivered a talk on when to start treatment for PD. He discussed the 'pre-clinical' markers including depression and olfactory disturbances, the pathological clues, neuroimaging assessment of progression, and genetic linkages. He highlighted results from the DATATOP, TEMPO and QE2 studies. Professor Schapira reviewed the advantages of early monotherapy, the issue of neuroprotection and the conflicting evidence surrounding the concept. He concluded that the eventual decision to treat early must be made after a patient orientated discussion, balancing the side effects of treatment with the reported improvements in quality of life, symptom control and disease progression.

Professor D Burn (Newcastle) then examined the similarities and differences between dementia with Lewy bodies (DLB) with PD dementia (PDD), and discussed diagnostic criteria, management algorithms and drug treatments for PDD. He briefly reviewed cholinesterase inhibitors, and memantine as possible therapies, then proceeded to talk about drugs with multiple modes of action (including ladostigil and adenosine receptor antagonists). Finally, various anti-amyloid strategies for PDD, such as statins, muscarinic-M1 receptor agonists, anti-inflammatory agents and amyloid immunisation therapy were discussed.

Professor D Brooks (London) outlined the uses of FDG-PET, FP-SPECT, F-Dopa PET, Acetylcholinesterase Imaging and PET amyloid plaque imaging in PDD, DLB and Alzheimer's disease. PDD patients demonstrated decreased parieto-temporal metabolism levels, decreased mesocortical dopamine levels and globally decreased acetylcholinesterase levels. Professor Brooks talked briefly on depression and concluded that depressed PD patients were more likely to demonstrate frontal lobe hypometabolism and



limbic dopamine dysfunction.

The afternoon session began with a presentation by Professor P Barone (Milan) on the epidemiology, treatment and management of hallucinations. He highlighted the facts that atypical anti-psychotics were recommended, long term treatment is the rule, patients with concomitant dementia with visual hallucinations should probably start a cholinesterase inhibitor, and that testing cognitive function is an essential part of the clinical examination.

Professor C Clarke (Birmingham) offered an appraisal of drug therapy for motor and non-motor symptoms. He summarised the shortfalls of previous trials in symptomatic early PD treatment and then introduced the ongoing PDMED trial, which looks at both the patient-related quality of life and health economics. He then discussed the surgical options for PD including bilateral subthalamic stimulation. Finally, he discussed the future of drug therapy in PD, including prolonged release ropinirole, safinamide, and antidyskinesia agents such as istradefylline, an adenosine A2a receptor antagonist.

Professor F Stocchi (Rome) discussed the management of respiratory dysfunction and sleep problems in PD. He reported the early results of his own study in 30 patients (all non-smokers) with respiratory dysfunction and PD. Assessments of maximal inspiratory pressure, pulmonary function tests and dyspnoea perception in patients in 'on' and 'off' phases showed that 90% of patients had difficulties when 'off' compared to 78% when 'on'. He postulated that the results can be explained by bradykinesia of the diaphragm and intercostal muscles, and postural problems. He then went on to review sleep problems which are highly prevalent in PD, and their effect on quality of life. He suggested treatments for nocturnal akinesia, rigidity and dystonia (prolonged release levodopa and COMT-I), sleep initiation problems (benzodiazepines) and sleep maintenance problems (amitriptyline, clonazepam and clozapine).

Professor R Brown (London) emphasised the importance of screening and detection of symptoms such as depression which are frequently stigmatised, and mentioned the various assessment tools for measuring these symptoms. For mild depression, he recommended anti-depressants and general measures such as anxiety man-

agement and exercise. For more severe depression, he suggested SSRIs, but called for more research into PD and depression since there is currently no established treatment algorithm. Ongoing trials suggest the use of combined serotonin-noradrenaline therapies such as venlafaxine and D2/D3 agonists such as pramipexole, for patients with treatment resistant depression with PD. However all have limited evidence of safety and tolerability, and cognitive behavioural therapy may therefore also have an important role.

Mrs J Johnson (London), a clinical specialist speech and language therapist in progressive neurological disorders, outlined the main motor features of dysarthria. She reviewed the drug treatments, therapeutic devices and the management programmes (including the Lee Silverman Voice Treatment) available to patients. Mrs Johnson concluded by calling attention to the paucity of drug trials that use speech assessment as end points.

Professor P Odin (Bremerhaven) discussed sexual dysfunction and highlighted previous studies which showed that 68.5% of patients with PD suffered from such problems, compared to 33% in a healthy group of the same mean age. While a combination of autonomic dysfunction and psychological anxiety was blamed for hyposexuality, Professor Odin recommended the involvement of gynaecological and urological teams, psychosocial counselling, anti-anxiety medication, the use of sildenafil and related agents, and a reduction in PD medication (if possible).

Dr G MacPhee (Glasgow) discussed gambling, impulsive and compulsive behaviour, outlining the progression from 'recreational', 'problem' and 'pathological' gambling (PG), drawing parallels with obsessive compulsive disorder. Dr MacPhee ended with a review of management. Whilst the evidence base remains poor, an individualised approach utilising neuroleptics, mood stabilisers and psychological counselling, including CBT and Gambler's Anonymous, was advised.

Dr M Visser (Netherlands) discussed quality of life determinants in PD. Sexual, urinary, gastrointestinal and thermoregulatory problems appear to be most predictive of low Health Related Quality of Life scores. Dr Visser suggested that future treatment should target improvement in activities of daily living, psychosocial problems such as depression, and the autonomic nervous system.

The valuable questions which followed many of the presentations made a significant contribution to the proceedings and ensured that lively discussion continued in-between sessions. A fourth meeting is already at an advanced stage of planning.

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# The MS Society Convention for People with MS – MS Life 2008

29-30 March, 2008; Manchester, UK.

**M**S Life 2008 was held in Manchester and was Europe's biggest ever event for the MS community. The convention was a massive achievement, attended by more than 3,500 people touched by multiple sclerosis, including over 350 wheelchair users and 450 visitors with mobility aids.

Every part of the UK was represented and there were many European and international visitors who were drawn to the event by the research presentations, workshops and over 80 exhibition stands aimed at offering information on improvements in quality of life for people affected by MS. Highlights of the weekend also included live cookery demonstrations, Kinky Myelinky – the exclusively inclusive club night and the My Style fashion show featuring models from the MS community in front of a sell out crowd of 400.

In response to delegate feedback from previous years the research sections of the convention were expanded and improved. The Meet the Scientist zone, where delegates could link up with research scientists to gain an insight into basic laboratory projects in the field of MS, was developed to become the Meet the Expert zone. As well as scientists, there was representation from many parts of the multi-disciplinary team involved in the care of a person with MS, including MS nurses, clinicians and clinical psychologists. The zone also boasted information stands focusing on magnetic resonance imaging (MRI) and the UK MS Tissue bank.

In parallel with around 30 workshops on varied topics including social care, living with primary progressive MS, managing fatigue, care services and starting a family, seven international research speakers gave talks on a diverse range of topical subject areas in the field of MS throughout the two day conference. Dr Alasdair Coles, an academic neurologist in Cambridge working on experimental therapies for MS, began the sessions with a discussion of risks, placebos and myth busting. He presented some of the trends and conclusions which can be drawn from looking at longitudinal data from people with MS and discussed the balance which needs to be achieved between potential benefits and risks associated with treatment of a long term condition such as MS. He also reviewed data on the effectiveness of current therapies for MS and discussed the need for open and candid information to be provided on the benefits of these treatments.

Prof David Miller, Head of the Department of Neuroinflammation at the Institute of Neurology followed this with an update on research using MRI methods to improve diagnosis, identify prognostic markers, understand disease mechanisms and monitor new treatments in MS. He showed how a new and more powerful generation of MRI scanners is now being manufactured which are able to detect the damage to nerve fibres that causes disability as well as the repair of myelin that enables



recovery to take place.

Dr Brenda Banwell, Director of the Paediatric Multiple Sclerosis Clinic in Toronto, Canada, discussed why MS is increasingly being recognised in children and showed why the diagnosis of MS in a child or teen may be complicated and delayed. Dr Banwell's talk highlighted the fact that research into the causes of MS may be particularly important in the youngest people diagnosed with the condition because environmental triggers may be more readily detected in people closer to the onset of the condition. Dr Banwell also focused on the impact of MS on the lives and activities of children and teens.

Saturday closed with an open debate chaired by broadcaster Nicholas Owen which explored the issue of patient choice. A panel comprising MS Society Chief Executive, Simon Gillespie, people with MS, a representative of MS Therapy Centres and a neurologist hosted a lively discussion on many topics including new MS treatments and how best to balance the benefits against harmful side effects.

Prof George Ebers, a clinician at the Wellcome Trust Centre for Human Genetics, opened the research presentations on Sunday with a talk on genetics in MS and the inheritance of MS susceptibility. He summarised his work investigating the genetic epidemiology of MS as well as his primary interest in studying gene environment interactions in MS.

The audience then heard from Prof Charles ffrench-Constant, whose session described how

two MS Society funded centres, The Cambridge Centre for Myelin Repair and the Edinburgh Translational Research Centre, would address the issues of prevention of nerve fibre loss, which causes chronic progressive disease and the promotion of myelin repair using new technologies such as stem or precursor cell reactivation or transplantation in the search for new therapies for MS.

Prof David Bates, a clinical neurologist at the University of Newcastle upon Tyne followed with a discussion on the future of disease modifying therapy. He focused on potential therapies which are currently in phase III trials, assessing their effectiveness in treating MS. He also touched on key contemporary issues, questioning how to treat those for whom traditional MS therapies are not effective and future possibilities for treating MS progression.

The research talks, one of the most popular elements of the MS Life weekend, ended with a fascinating presentation from Prof Carolyn Young, who heads a Neurological Rehabilitation Unit at the Walton Centre in Liverpool. Prof Young's talk, entitled 'If I had MS...', covered topics such as exercise, diet, medication, stress and rehabilitation for people with MS. She gave expert advice on the best way to make the most of clinical consultations and MS teams as well as information on how trials are run and her views on the benefits of participation in research.

The conference was an excellent opportunity for people affected by MS to hear about the latest advances in research and provided the opportunity for people from every area of the MS community to share experiences and their ideas about the many aspects of living with MS. Interviews with the scientists and a full conference breakdown are available on the MS Society website.

*Dr Laura Bell,  
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Multiple Sclerosis Society.*

## PREVIEW: British Neuro-Oncology Society (BNOS) Annual Conference 2008

25-27 June, 2008; Lancashire, UK

The University of Central Lancashire together with the Lancashire NHS Trust Royal Preston Hospital are hosting this year's BNOS conference from June 25th to the 27th. BNOS is the modern development of the British Neuro Oncology Group, formed in 1980, and includes UK leaders in basic science research together with a clinical membership drawn from consultant neuropathologists, neurosurgeons, oncologists, nurses and other health professionals who all have a common interest in malignant CNS tumours in both adults and children.

The conference venue will be in the Darwin and Foster buildings at the centre of the University campus a few hundred yards from Preston city centre. Accommodation will be at the nearby Holiday Inn and Travel Lodge. Preston is well served for transport routes and there is a direct rail link with Manchester International Airport.

This year it is estimated that the conference will attract approximately 280 delegates, and as well as the scientific sessions, will also include an extensive trade exhibition, 'education afternoon' and postgraduate forum. Consistent with the Society's original aims of promoting a dialogue on the management and biology of primary malignant brain tumours between basic scientists and clinicians, the meetings are kept as informal as possible.

Scientific sessions will be inter-disciplinary

and consist of short presentations and posters in addition to key note addresses. This year the conference is pleased to include talks from Professors Vescovi (Milan, Italy) and Lamzus (Hamburg, Germany) upon the involvement of stem cells in brain tumour and Professors Burnet (Cambridge) and Stummer (Dusseldorf, Germany) on novel advances in radiotherapy and neurosurgery. BNOS actively collaborates with the UK brain tumour charities and with the IBTA (International Brain Tumour Alliance) who have generously given their financial support towards hosting these eminent speakers.

Peer reviewed abstracts, including extended short papers from the main speakers, will be published in the British Journal of Neurosurgery. Copies of the edition, which will have a particular emphasis on brain tumour, will be available for delegates at the conference.

A full social programme is planned which includes a reception evening with the mayor of Preston in attendance at the Harris Museum, a stunning Grade I listed neo-classical building and the conference banquet at the National Football Museum, one of the UK's most original venues, with the opportunity for interactive participation and cabaret entertainment. Further details and appropriate registration and abstract submission forms may be found on the website: [www.uclan.ac.uk/bnos2008](http://www.uclan.ac.uk/bnos2008)



Charles Davis, Consultant Neurosurgeon, RPH, Vice-president of BNOS and Co-organiser BNOS2008.



British Neuro-Oncology Society



The Darwin Lecture Theatre. UCLan.

## PREVIEW: ABN Meeting

10-12 September, 2008; Aviemore, UK

Where better to enjoy a bracing academic programme than the Scottish Highlands in the early Autumn? The conference will be held at the Hilton Coylumbridge which has ample on-site accommodation (<http://www.hilton.co.uk/coylumbridge>), and is surrounded by the beauty of the Cairngorms National Park. Why not stay on for the weekend, and bring your partner or family, to enjoy a wild adventure or two around Aviemore? You can choose from archery, gorge walking (or swimming, if you're brave), canoeing, hill walking, sled dog tours, fishing, mountain biking, pony trekking, or even golf. See <http://www.visitaviemore.com> and <http://www.rothiemurchus.net> for more details.

As well as the usual scientific presentations, case reports and the traditional CPC, there will be an educational symposium on functional problems in neurology, and a scientific symposium on multiple sclerosis sponsored by the MS Society Scotland. On the Thursday evening there will be

a highland dinner and ceilidh. The ideal way to travel if you are coming from or through London is on the sleeper to Aviemore from Euston. There are potentially 36 first class single berths, and up to 84 places in standard class double berths, which can be booked 12 weeks in advance of the date of travel. You can fly to Inverness (we will lay on coaches) from most UK airports. Deadline for abstracts will be the end of May 2008 (tbc).

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