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Parkinson's Disease 2nd Advanced Masterclass

12th September 2008 Holiday Inn Filton - Bristol



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Welcome Letter

On behalf of the faculty of the BGS Movement Disorders Section, I am very pleased to welcome you here to Bristol for our second 'Advanced Parkinson's Masterclass'. In the few years that have elapsed since the last one there have been several exciting developments, and our faculty has selected areas that we feel have progressed, developed, or in which new data is available since the Masterclasses that you have previously attended.

There have been several new drugs, new formulations and changed indications. The availability of DaTSCAN has extended, and we feel that it is worthwhile revisiting this area.

Guidelines have been published not only for Parkinson's disease (NICE) but also for PD Dementia, and we will have an opportunity to review these, and also subsequent developments since their publication. Finally, we welcome a neurologist who will be guiding us through the developments in functional neurosurgery.

In respect of the wider perspective now that we are the 'Movement Disorders Section', we have also extended the remit to include Essential Tremor, and since as a Section of the BGS, we feel it is important to reflect on the comorbidities that are so frequently found in our (older) patients.

In summary, we have assembled a glittering array of the top experts in their respective fields to share their knowledge and experience. We hope you enjoy the day, and use the refreshment breaks to review the commercial exhibition - our sponsors allow us to keep the cost of this event to a minimum, and I am sure you will extend every courtesy to them.

The faculty are always aware that the most important people at any meeting are the attendees, and so will be keen to receive and reflect on your own comments, and encourage you to feedback your views on the evaluation sheets provided.

Doug MacMahon
Faculty

2nd Advanced Masterclass Programme

Friday 12th September. Holiday Inn Filton, Bristol

- 09.30** **Coffee and Exhibition**
- 10.00** **Welcome & Introduction:**
After NICE – what's New?
(a review of new therapeutic modalities and where they fit with NICE recommendations)
Dr Doug MacMahon, Cornwall
- 10.45** **Co-morbidities and their importance in
older people**
Dr Graeme Macphee, Glasgow
- 11.30** **Using Duodopa - experience to date**
Dr Paul Worth, Norwich
- 12.15** **Discussion**
- 12.30** **Lunch & Exhibition**
- 13.45** **'DaTSCAN - capabilities and limitations'**
Dr Paul Kemp, Southampton
- 14.30** **Dementia in Parkinson's Disease – a review**
Dr Ira Leroi, Manchester
- 15.15** **Refreshments & Exhibition**
- 15.35** **Essential Tremor**
Dr David Stewart, Glasgow
- 16.15** **Surgical update**
Dr Alan Whone, Bristol
- 17.00** **Closing remarks**
Dr Doug MacMahon, Cornwall
- 17.15** **Depart**

Biographical Details

DR PAUL KEMP



Paul M Kemp is a Consultant and Honorary Senior Lecturer in Nuclear Medicine at Southampton University Hospitals Trust. His major interest is in functional brain imaging, in particular the role of SPECT and PET in movement disorders and the dementias.

Southampton is one of the 15 European 'centres of excellence' for functional brain imaging as selected by the European Association of Nuclear Medicine. In 2006, Paul was appointed as the national adviser (UK) on functional brain imaging by the British Nuclear Medicine Society.

DR IRA LEROI



Dr Leroi has been a consultant in Lancashire Care Foundation Trust and an Honorary Senior Lecturer with the University of Manchester since 2002. Her special interest lies in the psychiatric aspects of neurodegenerative movement disorders. In particular, she has done research in the neuropsychiatry of Huntington's disease and published seminal papers on the neuropsychiatry of spinocerebellar ataxias. Her main interest now is in the mental health aspects of Parkinson's disease (PD) and she is doing a trial of memantine in PD dementia. She was the lead author on the first RCT of donepezil in PDD and participated in the recently published NICE guidelines for the diagnosis and management of PD. She has also served on the guidelines' committees for the psychiatric treatment of Huntington's disease and the American Medical Directors' Association's PD guidelines for nursing homes. Dr Leroi received

her medical and psychiatric training in Canada and undertook fellowship training in neuropsychiatry at Johns Hopkins University in Baltimore, USA. She joined the psychiatry faculty at Johns Hopkins as Assistant Professor before moving to the UK. In 2000 she was awarded the American Neuropsychiatric Association's Young Investigators' Award. Dr Leroi was recently appointed a Senior Research Fellow with the Parkinson's Disease Society and has been funded to study motivation disorders in PD. She has also been awarded, in 2006 - selected to participate in NESTA's Crucible program for early career researchers and in 2007 awarded a TRAM mentorship award for research proposal development.

Biographical Details

DR DOUG MACMAHON



Dr. Doug MacMahon is a Consultant Physician with special responsibility for the elderly with the Royal Cornwall Hospitals NHS Trust. He has a particular interest and has specialised in the treatment of Parkinson's disease and other chronic neurological diseases for the past 25 years. Other interests include Intermediate and Community Care. He was founder chairman of the PD special interest group of the British Geriatrics Society (BGS) and has worked closely with the PD Society to develop the role of the specialist nurse (chaired the steering group). He is past chair of the BGS Policy Committee and previously Medical Director of a Community Trust for 5 years; member of the NICE Clinical Guidelines Development Group for PD; written over 100 publications and 20 book chapters. He has made many presentations at international and national meetings and is member of several editorial boards and founder chairman of the PD Academy faculty.

DR GRAEME MACPHEE



Dr. Graeme JA Macphee is Consultant and Honorary Clinical Senior Lecturer in the Department of Medicine for the Elderly at Southern General Hospital Glasgow. He runs a joint tertiary referral Movement Disorders Clinic in the Institute of Neurological Sciences with Dr Donald Grosset, Consultant Neurologist as well as local PD services. He is Chairman of the British Geriatrics Society PD Section and PD Academy faculty member. Among other publications, Dr Macphee contributed the chapter on Diagnosis and differential diagnosis in Parkinson's disease in 'Parkinson's disease in the Older Patient' eds Hindle and Playfer. Current interests include the use of FP CIT SPECT scanning in early diagnosis of clinically uncertain parkinsonism and evaluation of non motor features of Parkinson's disease. He is a member of the international PD Non Motor group. Dr Macphee is past chairman of the Geriatric Advisory committee at the Royal College of Physicians and Surgeons of Glasgow and is co chair of Greater Glasgow and Clyde Formulary and New Drugs committee.

DR DAVID STEWART



Dr. Stewart is a Consultant Physician in Medicine for the Elderly at the Victoria Infirmary in Glasgow and Honorary Clinical Senior Lecturer, University of Glasgow. He has a longstanding interest in Parkinson's disease and established a multidisciplinary PD clinic in 1993. In 2000 the clinic achieved Chartermark status. His interests include audit and database development. He has developed a Parkinson's disease database, now in use in a number of centres throughout the country. He is a founder member of the Glasgow Movement Disorders Group and an advisor to Greater Glasgow Health Board on PD services. He is a current committee member and past chairman of the BGS PD Section.

Biographical Details

DR ALAN WHONE



Dr Alan Whone is Burden Senior Lecturer at Frenchay Hospital Bristol. Prior to training in neurology in the South West he undertook a PhD in Parkinson's disease and other movement disorders at the Cyclotron Unit, Hammersmith Hospital, London, with Professor D J Brooks. Presently Dr Whone performs movement disorders and DBS clinics at Frenchay where his clinical role is to support the functional neurosurgery movement disorders programme. His current research projects include clinical investigations of novel target sights for DBS in PD (the pedunculo-pontine nucleus), which he is investigating with Professor Steve Gill and lab based investigations with Professor Neil Scolding to assess the potential utility of bone marrow stem cells as a cell based therapy in Parkinson's disease.

DR PAUL WORTH



Dr Paul Worth has been a consultant in neurology and lead clinician in Parkinson's disease and movement disorders at the Norfolk and Norwich University Hospital in Norwich since 2004. He trained in medicine at Cambridge and Oxford universities, and undertook his general postgraduate training at a number of London hospitals. His training in neurology took place at the National Hospital for Neurology and Neurosurgery, Queen Square, and at St Mary's and Atkinson Morley's hospitals in London.

Dr Worth undertook a 3 year programme of research as a Medical Research Council Clinical Training Fellow at the Institute of Neurology, London, with Professor Nick Wood, where his main research interest was the genetics of movement disorders including Parkinson's disease and the inherited cerebellar ataxias. He was awarded a PhD in Neurological genetics at University College London in 2002.

Dr Worth is at the Norfolk and Norwich University Hospital. He has developed a new clinic for patients with Parkinson's disease involving assessment by the multi-disciplinary team at the same visit. His team was shortlisted for the Hospital Doctor Parkinson's Team of the Year award in 2006. He is interested in ways of improving the delivery of health care services to patients with PD, including the appropriate use of coordinated community services, and is currently seeking funding for a research grant to investigate this. He is also interested in ways of improving diagnostic accuracy in Parkinson's disease, and is shortly to begin a collaborative research project on the use of DaTSCAN in the diagnosis of vascular parkinsonism with Dr Peter Bain at Charing Cross Hospital, London.

Dr Worth is lead clinician for Parkinson's Disease in the Dementia and Neurodegenerative Disease Research Network (DeNDRoN) East Anglia, and sits on the national DeNDRoN Parkinson's Disease Clinical Studies Group.

After NICE – what's New?

(a review of new therapeutic modalities and where they fit with NICE recommendations)

Dr Doug MacMahon

After a 2 year gestational period and much hard year work by the NICE Guidelines Development Group, charged with evaluating every available therapeutic and diagnostic option, the NICE guideline – CG035 - was born in June 2006. In the subsequent 2 years much has changed - or has it?

We have some anecdotes and audit evidence that at least some recommendations have been implemented but we await the roll-out to a National Implementation Audit to really assess how much has changed. This work is supported by the PDS and has been driven forward by Dorothy Robertson and Peter Fletcher for the BGS Section. It is no coincidence that the PD Academy has gone from strength to strength, and now can count well over 200 graduates from 13 courses held thus far, and most of these have completed Service Audits in their own areas.

Since the NICE publication, we have several new drugs launched including two with new routes of administration– e.g. Rasagiline (Azilect™), Rotigotine (Neupro™) transdermal patches, and Duodopa™ intra-jejunal levodopa system – and we have also seen a strategic move away from the ergot agonists towards the synthetic ones. All these will be discussed and their places in the therapeutic armamentarium will be reviewed.

Those clever neurosurgeons have found targets new, and a trial is currently in progress to assess the relative merits of this latest approach.

For our northern neighbours, the SIGN process is, I understand, making progress, and will be interesting to compare with the NICE ones when hatched.

On reflection, however, perhaps the greatest changes that have occurred since its publication are the widespread recognition of non-motor signs and symptoms, and amongst them the importance of cognitive changes and dementia and also the need for consideration of palliative care for people with this disease.

The importance of comorbidities in older patients with PD

Dr Graeme Macphee

AIMS:

1. To review and discuss the pattern and contribution of unrelated comorbidities to resource use and expenditure in older patients with PD
2. To review and explore the impact of comorbidities on the management of the older PD patient including some hazards of 'unscheduled care'.

ABSTRACT:

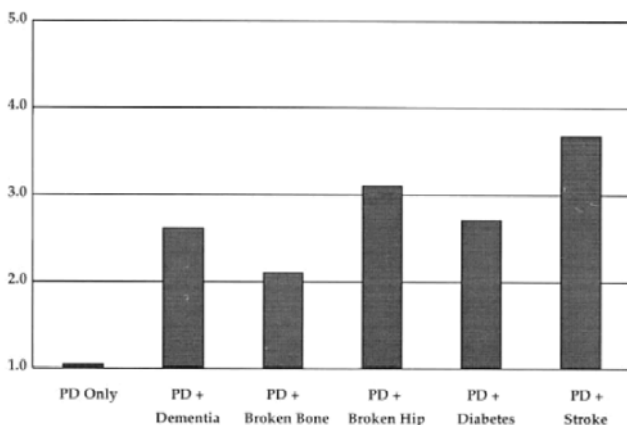
Identification and management of intrinsic non-motor features such as depression and autonomic dysfunction in PD are increasingly recognised as important determinants of quality of life for people with PD and their carers. In contrast, the impact of 'unrelated' medical comorbidities such as infection and vascular disease on chronic disease management in PD has received less attention .

A seminal study from the BGS PD SIG in 2000 reported that a cohort of older PD patients (mean age 76 years) had on average, 4 other active medical conditions. Such comorbidity may confound diagnosis and significantly influence prognosis and disease trajectory in PD. Studies from North America suggest that associated comorbidities in PD may contribute to two to threefold expenditure and resource use compared to PD patients without associated illnesses. Characterisation of comorbidity in PD is also crucial in ensuring clinical trials are robust and valid. Several instruments are reported as reliable and valid in measuring comorbidity generally but have not been subject to extensive clinimetric assessment in PD.

Acute insults or illness such as fractures or sepsis are common precipitants of hospital admission in PD patients. This session will use case based examples to highlight and discuss some hazards of inpatient episodes for conditions unrelated to PD. This will include a brief review of potential drug interactions with dopaminergic therapies and the rare but important complications of Parkinsonism – hyperpyrexia syndrome and akinetic crisis.

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1. Pressley JC, Louis ED, Tang M-X, Cote L, Cohen PD, Glied S, Mayeux R. The impact of comorbid disease and injuries on resource use and expenditures in Parkinsonism. *Neurology* 2003 Jan; 60: 87-93
2. de Groot V, Beckerman H, Lankhorst GJ, Bouter LM. How to measure comorbidity: a critical review of available methods. *J Clin Epidemiol* 2003; 56 221-29
3. Jost WH, Brück C. Drug interactions in the treatment of Parkinson's disease. *J Neurol* 2002 249(3): III/24–III/29
4. Woodford H, Walker R. Emergency hospital admissions in idiopathic Parkinson's disease *Mov Dis* 2005; 20 (9) 1104–8
5. Kipps et al *Movement Disorders Emergencies Mov Disord* 2004; 20: 322-334



Duodopa - Experience to Date

Dr Paul Worth

AIMS:

1. To describe the historical background to and the evidence base for intra-jejunal levodopa (Duodopa®) therapy
2. To discuss the indications for Duodopa™ therapy, and to describe the process of initiation and ongoing management of patients treated with Duodopa™

ABSTRACT:

Levodopa-induced motor complications in patients with advanced Parkinson's disease are common and present a significant management challenge. Levodopa is absorbed specifically from the jejunum. Oral therapies may contribute to the development of motor complications as a result of delayed gastric emptying. This results in gastric sequestration of oral medication, unpredictable absorption and ultimately fluctuating plasma drug levels with pulsatile stimulation of striatal dopamine receptors. Moreover, the management of these self same motor complications is itself dependent on reliable drug absorption, and poor bioavailability is thought to underlie the eventual failure of oral therapies adequately to manage these complications. In patients with severe motor fluctuations and dyskinesias, three main therapeutic options are available: apomorphine subcutaneous infusion, deep brain stimulation or intrajejunal levodopa infusion (Duodopa®).

The idea of infusing anti-parkinsonian medication intravenously or into the duodenum / jejunum for patients with advanced PD is not new and was first performed in the 1970s and continued experimentally into the 1980s and 1990s. Overall, the results were encouraging. However, the low solubility of Levodopa necessitated large volumes of fluid to be used and hence the procedure was not developed commercially. Duodopa® is the trade name for Levodopa / Carbidopa in the form of a gel containing Levodopa 2000mg and Carbidopa 500 mg suspended in carboxymethyl cellulose in a 100ml cassette that has been developed recently.

Evidence for the superiority of Duodopa® over conventional management was obtained in a randomised crossover study. This showed that patients treated with Duodopa spent less time with dyskinesia and 'off' than those treated with conventional therapy. However, this study was short, not placebo-controlled and there are limited published long-term data. Safety issues identified to date include peri-operative infection, tube blockage or accidental displacement.

Duodopa® has recently been introduced to the UK and has been used in approximately 60 patients to date with 1200 overall in Europe. However, owing to high cost (approximately £30,000 per annum), obtaining funding for the treatment is not straightforward in the UK and usually requires application to the local Drugs and Therapeutics Committee or PCT on an 'exceptional' basis. Subcutaneous apomorphine therapy is unavailable in some countries and there are a number of relative and absolute contraindications to deep brain stimulation which exclude a significant proportion of patients from receiving this therapy. To date, there are no comparative studies on the efficacy, cost-effectiveness, adverse event profile and long term outcomes with these three treatment modalities which might help to inform the decision as to which of these therapies is appropriate in a given patient. Hence, the choice of therapy is made somewhat arbitrarily by the treating clinician. Patients deemed suitable for Duodopa® therapy are admitted to hospital and a temporary naso-jejunal tube is inserted allowing Duodopa® to be infused directly into the jejunum from a dedicated pump. The dose of Duodopa® is then titrated according to the patient's response. Once the response has been ascertained and the optimal doses (morning bolus and continuous infusion rate) have been determined, a dedicated PEG tube with an inner tube is placed endoscopically in the jejunum.

DaTSCAN - Capabilities and Limitations

Dr Paul Kemp

AIMS:

To appreciate the contribution of dopamine imaging in the clinically uncertain patient with:

- 1) Suspected Parkinson's disease/parkinsonian syndrome
- 2) Suspected drug induced movement disorder
- 3) Suspected cognitive impairment associated with movement disorder.

ABSTRACT:

This presentation will review the clinical role of dopaminergic imaging in the uncertain patient with suspected IPD, parkinsonian syndrome, drug induced movement disorder and Lewy body dementia. Case examples illustrating the clinical usage of imaging will be presented in conjunction with a critical up-to-date review of the literature. Finally, the relevant recent recommendations from NICE on neuro-imaging will be covered.

Dementia in PD

Dr Ira Leroi

AIMS:

At the end of the session, participants will understand:

- a) The prevalence, clinical presentation and aspects of etiology of PDD
- b) Differences between PDD and DLB, highlighting the recommendations from the international PDD/DLB working group
- c) New diagnostic criteria for PDD
- d) Prevalence of mild cognitive impairment in PD (MCI)
- e) Prevalence and assessment of behavioural disturbances associated with PDD
- f) Up to date aspects of risk factors for conversion to PDD
- g) New rating scales for assessing PDD
- h) A review of PDD treatment

ABSTRACT:

Dementia associated with Parkinson's disease (PDD) is a common and debilitating complication of PD and can occur in more than 40% of PD sufferers. There are several risk factors that have been identified for conversion from PD to PDD including older age, weight loss, poor response to levo-dopa, emergence of visual hallucinations and clinical phenotype, which may include excessive daytime sleepiness and REM sleep behaviour disorder. Diagnostic criteria for PDD have now been established (Emre et al, 2007) and newer cognitive assessment tools, such as the SCOPA-Cog and PD-Cognitive Rating Scale (Pagonabarraga et al, 2008), have been developed. Mild cognitive impairment in untreated, incident PD is almost 20%, which is almost twice as common as age-matched controls. MCI-PD can be divided into various subtypes including amnesic-MCI and non-amnesic MCI (Aarsland et al, 2008). Behavioural disturbances in PDD are common and assessment can be undertaken using the Neuropsychiatric Inventory (NPI)(Aarsland et al, 2007).

The clinical presentation of PDD overlaps with DLB. A recent position statement on the overlap between DLB and PDD has recently been published and suggests that the conditions, while phenotypically distinct should be considered as single spectrum disorder at the pathophysiological level (Lippa et al, 2007). The conclusions from this consensus meeting will be outlined.

Treatment of PDD will be reviewed. The mainstay of treatment is the recently licensed cholinesterase inhibitor, rivastigmine, which was shown to be effective and well tolerated in the EXPRESS study (Emre et al, 2004). Other treatment options, including memantine will be reviewed (Leroi et al, 2006) and treatment guidelines for PDD outlined.

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1. Aarsland D et al. Neuropsychiatric symptoms in patients with Parkinson's disease and dementia: frequency, profile and associated care giver stress. *JNNP* 2007;78:36-42
2. Aarsland D et al. Mild cognitive impairment in incident, untreated Parkinson's disease: The Norwegian ParkWest study. Abstract. Movement Disorders Society Annual Meeting, Chicago, 2008.
3. Emre et al. Rivastigmine for dementia associated with Parkinson's disease. *NEJM* 2004. 351;24:2509-2518
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5. Lippa et al. DLB and PDD boundary issues: Diagnosis, treatment, molecular pathology and biomarkers. *Neurology*. 2007;68:812-819
6. Pagonabarraga J et al. Parkinson's Disease-Cognitive Rating Scale: A new cognitive scale specific for Parkinson's disease. *Mov Dis* 2008;23:998-1005

Essential Tremor

Dr David Stewart

AIMS:

1. To review current thinking as to the aetiology and pathophysiology of essential tremor
2. To explore the diagnosis and differential diagnosis of essential tremor.
3. To review the treatment options.

ABSTRACT:

Essential tremor (ET) is the most common tremor disorder. Prevalence rises with age with over 4% of those over 65 years affected. The aetiology is unknown although genetic factors may be less important than previously thought. A number of environmental risk factors including beta-carboline alkaloids and lead have been implicated. The view of ET as a benign monosymptomatic condition has been challenged with the suggestion that it may be neurodegenerative in nature. Pathophysiology is poorly determined but is likely to be heterogeneous.

ET is commonly misdiagnosed: 30-50% of patients are erroneously diagnosed as having Parkinson's disease (PD) or another tremor disorder. Diagnosis is clinical and based on the finding of a 4-12 Hz postural and kinetic tremor without significant rigidity or bradykinesia. The main differential diagnosis lies between PD and dystonic tremor. In younger patients it is important to exclude Wilson's disease. In cases of diagnostic uncertainty, FP-CIT SPECT scanning (DaTSCAN) can help to differentiate ET from degenerative parkinsonism.

Treatment is often disappointing and tremor is rarely abolished. Indications for treatment include significant functional impairment or embarrassment. Intermittent therapy tailored to specific occasions may be appropriate for some patients. Propranolol and primidone are the first line drugs and are effective in between 30-70% of cases. Patients should be started on low doses and slowly titrated upwards thereafter. Primidone is poorly tolerated in a substantial proportion of patients even at low dose. Alternative drug therapy includes topiramate, gabapentin and benzodiazepines. In severe cases functional neurosurgery may be indicated with thalamic deep brain stimulation the procedure of choice.

Surgical Update

Dr Alan Whone

AIMS:

At the end of the session participants will:

1. Understand the history, background and developments of neurosurgical approaches to the treatment of Parkinson's disease.
2. Appreciate which patients to consider referring for DBS, understand what the symptomatic benefits may be and appreciate the potential side effects and complications.

ABSTRACT:

The role of functional neurosurgery in the treatment of PD has long been considered. In past decades we have moved from lesioning, to DBS, to foetal cell transplants, to infusions of growth factors. This session will embrace all of these developments as well as highlight potential future developments. Particularly, the role of conventional DBS surgery in the armourmentarium of PD therapy will be considered as well as novel DBS target sites for hitherto untreatable symptoms in PD. Putative future neuroprotective and neurorestorative strategies including approaches such as viral vectors, stem cell based therapies and convection enhanced delivery will be discussed.

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