THE BRITISH NEUROPSYCHIATRY ASSOCIATION 27th AGM

www.bnpa.org.uk

The Institute of Child Health, Guilford Street, London

27/28 FEBRUARY 2014

Thursday 27 February

New Developments in Cognition
JNNP Guest Lecture
Members' platform presentations
Neuropsychiatry Research Update

Friday 28 February

Neuropsychiatric Manifestations/Encephalitis
BNPA Medal Lecture
BNPA AGM (members only)
The Wellcome Trust Debate

27/28 February
Poster Session

Welcome to the 27th annual meeting of the British Neuropsychiatry Association

British Neuropsychiatry Association Committee:

Dr Alan Carson
President

Professor David Skuse
Treasurer

Dr Hugh Rickards
Secretary

Dr Chris Butler
Director

Professor Peter Halligan
Director

Professor Markus Reuber
Director

Dr Kieran O'Driscoll
Director

Adam Zeman
Ex-officio

Professor Eileen Joyce
Ex-officio

BNPA Administration:

Jackie Ashmenall
Administrator

Tel/Fax: +44 (0)20 8878 0573
MB: 00 44 (0) 7940 591096
E-mail: admin@bnpa.org.uk
Website: www.bnpa.org.uk

Gwen Cutmore
Conference Secretary

Tel/Fax: 01621 843334
Email: gwen@gwen1.orangehome.co.uk

Speakers Short Biographies and Abstracts Day 1, Thursday 27 February

NEW DEVELOPMENTS IN COGNITION
Chair: Peter Halligan

Oxytocin and Social Cognition

Professor David Skuse is Professor of Behavioural and Brain Sciences at the Institute of Child Health, University College London, and honorary Consultant in Developmental Neuropsychiatry at Great Ormond Street Hospital. He has worked there since 1985, and in 1998 he established the UK's first Social Communication Disorders Clinic for Children with High Functioning Autism Spectrum Disorders. The clinic offers a national service for the clinical assessment and management of children in mainstream education who are suspected of having an autistic spectrum condition. Using 'state of the art' procedures, it is supported by a large multidisciplinary team. All children have comprehensive genetic investigations. Professor Skuse trained at the Maudsley Hospital and Institute of Psychiatry before coming to Great Ormond Street Hospital in 1985. He is a Fellow of the Royal Colleges of Physicians, Psychiatrists and Paediatricians. Specialisms include autism spectrum disorders, other neurodevelopmental conditions including ADHD and Tourette syndrome, and chromosomal disorders especially those involving sex chromosome aneuploides such as Turner and Klinefelter syndromes.

All families attending the National Clinic for High Functioning Autism are offered the opportunity to join the Autism Families Project. Our research aims to improve our understanding of autism spectrum conditions. The Autism Families research team at the Institute of Child Health is closely linked to the clinic. Current research interests focus on potential genetic causes, and on the most appropriate way to diagnose Autism Spectrum Disorders, including associated conditions such as ADHD, anxiety and conduct problems. We have developed methods of supporting children with ASD during their transition to secondary school, and provide psychoeducational groups for children to understand more about their condition.

Abstract
Individual differences in our capacity to read other people's emotions and to remember faces we have seen before are highly variable in the general population. Some people are super-recognizers; others have difficulty remembering their own family members. Such abilities are also highly heritable, implying our genetic makeup exerts an important influence. But what genes are involved in social perception? Where do they act when our brains process social signals? What happens if the social perception system malfunctions? How does it affect our social behaviour? These are questions I aim to answer in this lecture.

The neuropeptides (brain hormones) oxytocin and vasopressin are evolutionarily conserved regulators of social behaviour. Evidence is building that they are especially important for interpersonal bonding throughout mammalian species, from rodents through primates to humans. Oxytocin probably has a greater influence on the social perceptions and behaviour of females than males (influenced by female sex hormones such as oestrogen), and the impact of vasopressin may be rather greater upon male social perceptions and behaviour (influenced by male sex hormones or androgens). Recent research has suggested that, in people with autism, social motivation might be decreased because they do not find social interactions as rewarding as neurotypical individuals. The value we place on social reward appears to be influenced by oxytocin. Giving extra oxytocin to people with autism (for example, by nasal spray) improves the accuracy of their social perceptions (and potentially, the concomitant reward from a social encounter), at least temporarily.

If oxytocin in the brain impacts upon individual differences in sensitivity to social cues, the efficiency with which the hormone's receptor in the brain is activated might be important. We studied whether genetic variants of the oxytocin receptor affect face recognition memory in families where there was an autistic child. We discovered that possession of a single relatively common oxytocin receptor variant, of a type that could influence gene activity, accounted for up to 10% of their performance. Remarkably, our finding was replicated in both UK and in Finnish populations. About 35% of family members were homozygous for the risk genotype, meaning they possessed two copies of the gene variant that was associated with relatively less good facial memory than the population average. Our findings imply oxytocin and its receptor in the brain play a significant role in accounting for individual differences in our ability to remember the faces of unfamiliar people. On the whole, that influence is relatively subtle. Nevertheless, one in three of us possesses only the genetic version of the oxytocin receptor that is relatively inefficient. If we are in that large minority, it is unlikely we will ever be able to match the remarkable face memory skills of a Bill Clinton.

Speakers Short Biographies and Abstracts Day 1, Thursday 27 February

Neuropsychiatry of social knowledge and moral motivation

Since **Roland Zahn's** doctoral research training in neuropsychology and neuroimaging, he has been fascinated by the question of the neurocognitive underpinnings of psychiatric symptoms. Whilst training in neurology at the University of Aachen in Germany, he investigated the neural basis of post-stroke recovery of conceptual knowledge. On continuing his clinical training in psychiatry at the University of Freiburg, he became aware of the potential importance of the neuroscience of conceptual knowledge in understanding affective disorders and socially inappropriate behaviour, which have been classically studied from the perspective of emotion research. Whilst there was already a growing literature on the neural basis of general conceptual knowledge, the neural basis of conceptual knowledge of social behaviour (i.e. social concepts such as "stingy" or "honourable"), which is of eminent interest to psychiatry, was unknown. Funded by a fellowship from the German National Academy of Sciences, he pursued this question at the US National Institutes of Health. He continued this line of research at the University of Manchester whilst undertaking top-up training in old age psychiatry and subsequently working as an honorary consultant psychiatrist in cognitive assessment and affective disorders services. He has recently joined the newly founded Centre for Affective Disorders at the Institute of Psychiatry, King's College London. His current MRC clinician scientist fellowship aims at developing the first functional MRI biomarker of recurrence risk in major depressive disorder.

Abstract

Moral behaviour requires at least two components: 1) knowing about socio-cultural norms and the needs of others (i.e. social knowledge), and 2) being motivated to act on this knowledge (i.e. moral motivation and emotion). There is emerging evidence from neuroimaging in healthy participants as well as patients with frontotemporal dementia that abstract social knowledge (i.e. knowledge of social concepts such as "greed") is stored in the superior anterior temporal cortex, especially within the right hemisphere. Different moral emotions (guilt or indignation) are associated with the same social concepts (e.g. "greed") depending on the context (oneself or someone else behaving e.g. "greedily"). Interestingly, guilt and indignation shared activation within the anterior temporal cortex, whilst eliciting distinct activation patterns within frontal-subcortical networks. It is these distinctive neural signatures which are of interest to neuropsychiatry, because they could account for the clinical observation of selective disruption of particular types of moral emotions. The healthy experience of guilt was associated with septal, subgenual cingulate, and frontopolar activation. This was corroborated by showing that loss of guilt in patients with frontotemporal dementia correlated with neurodegeneration in septal and frontopolar areas. These neural signatures of guilt were distinctive when compared with indignation or anger towards others. Despite the theoretical importance of guilt and self-blame, first highlighted by Freud, their neural bases in major depressive disorder (MDD) were unknown. We recently addressed this question using fMRI. As predicted from our earlier work, overgeneralised self-blame (e.g. "feeling guilty for everything") and MDD were associated with functional disconnection between the anterior temporal cortex and a septal-subgenual-frontopolar network when patients with MDD experienced guilt. Ongoing clinical translation of these findings including development of an fMRI biomarker of MDD recurrence risk and real-time fMRI-based neurofeedback interventions to enhance adaptive moral emotions, as well as interventions tackling overgeneralised self-blame will be discussed.

Speakers Short Biographies and Abstracts Day 1, Thursday 27 February

Brain development in adolescence

Professor Sarah-Jayne Blakemore is a Royal Society University Research Fellow and Professor of Cognitive Neuroscience at UCL. She is Leader of the Developmental Cognitive Neuroscience Group at the Institute of Cognitive Neuroscience. Her group's research focuses on social cognition and decision-making in human adolescence. Professor Blakemore studied Experimental Psychology at Oxford University (1993-1996) and then did her PhD (1996-2000) at the Functional Imaging Lab (FIL) with Chris Frith and Daniel Wolpert, investigating the self-monitoring of action in healthy individuals and people with schizophrenia. She then took up a Wellcome Trust International Research Fellowship (2001-2003) to work in Lyon, France, with Jean Decety on the perception of causality in the human brain. This was followed by a Royal Society Dorothy Hodgkin Fellowship (2004-2007) and then a Royal Society University Research Fellowship (2007-2013) at the UCL Institute of Cognitive Neuroscience.

Professor Blakemore has been awarded a number of prizes including the British Psychological Society Doctoral Award 2001, the British Psychological Society Spearman Medal for outstanding early career research 2006, the Lecturer Award 2011 by the Swedish Neuropsychology Society and the Young Mind&Brain Prize from the University of Turin 2013. She is actively involved in Public Engagement with Science: frequently gives public lectures and talks at schools, has worked with the Select Committee for Education, and acted as scientific consultant on the BBC series The Human Mind in 2003. Professor Blakemore has an interest in the links between neuroscience and education and has co-authored a book with Professor Uta Frith called The Learning Brain: Lessons for Education. She is Co-Director of the Wellcome Trust's Four Year PhD Programme in Neuroscience at UCL. She is Editor-in-Chief of the journal Developmental Cognitive Neuroscience.

Professor Sarah-Jayne Blakemore is a member of the Vision for science and mathematics education 5-19 project committee.

Abstract
Adolescence is a period of formative biological and social transition. Social cognitive processes involved in navigating an increasingly complex social world continue to develop throughout adolescence. Research in the past 15 years has demonstrated significant functional and structural changes in the brain during adolescence. Areas of the social brain undergo both structural changes and functional reorganization during the second decade of life, possibly reflecting a sensitive period for adapting to one's social environment. The changes in social environment that occur during adolescence might interact with increasing executive functions and heightened social sensitivity to influence a number of adolescent behaviours. I will discuss the importance of considering the social environment and social rewards in research on adolescent cognition and behaviour.

Schizophrenia and Cognition

Professor Eileen Joyce obtained her first degree, PhD and medical degree from the University of Cambridge. She trained in psychiatry at the Bethlem and Maudsley Hospitals and spent several years as a research worker at the Institute of Psychiatry, where she was a Wellcome Trust Lecturer in Mental Health, and the USA National Institutes of Health. Before moving to UCL/UCLH, she was Professor of Neuropsychiatry at Imperial College London. Professor Joyce is a Consultant Neuropsychiatrist at the National Hospital for Neurology and Neurosurgery. Her research interests are, psychosis, deep brain stimulation, and functional neurological symptoms

Abstract
Cognitive impairment is generally considered and important facet of the schizophrenia syndrome but how fundamental is it? This presentation will argue that there is a limited general resource in schizophrenia that constrains the performance of a wide range of specific cognitive functions and underlies the development of psychotic symptoms as well as determining functional outcome. The possible neurobiological underpinnings will be discussed.

The prospects for a vaccine for Alzheimer's Disease

Prof James Nicoll BSc MBChB MD FRCPath, University of Southampton, UK.
I went to Medical School in Bristol and qualified in 1984, having undertaken an intercalated BSc in Neurophysiology, and decided to pursue a career in Neuropathology. After house jobs in Bristol I did pathology training jobs in Oxford and Cardiff and returned to Bristol to undertake specialist training in Neuropathology at Frenchay Hospital (1987-1992). I then obtained a clinical academic post at the Institute of Neurological Sciences in Glasgow as an Honorary Consultant and Senior Lecturer in Neuropathology, subsequently gaining promotion to a Readership and a Chair. While in Glasgow I developed interests in the parallels between the response of the brain to acute injury (e.g. head injury and stroke) and neurodegeneration (e.g. Alzheimer's disease), specifically that they share common cellular reactions (e.g. neuroinflammation), upregulation of certain proteins (e.g. APP, Aβ, APOE) and possibly share genetic influences (e.g. APOE and cytokine gene polymorphisms).

In 2001 I moved to Southampton to a Chair/Honorary Consultant in Neuropathology. Shortly after moving to Southampton we were privileged to be the first in the world to see the remarkable changes in the human brain of a person with Alzheimer's disease who had been immunised with Aβ, having enrolled in a clinical trial undertaken by Elan Pharmaceuticals. This showed that the immunisation had resulted in removal from the brain of the protein thought by many to be the root cause of Alzheimer's disease (Aβ). Following this, in collaboration with colleagues in Southampton and elsewhere, we have been attempting to perform a systematic clinical and neuropathological follow-up of the patients in that original Aβ immunisation trial. The results clearly show that it is possible to influence the abnormal processes that occur in the brain in Alzheimer's disease, although evidence for a beneficial effect on cognitive function was lacking in our study. Nevertheless, other studies have shown evidence of benefit to cognitive function and this gives rise to the hope that we have at least started on the road to finding an effective treatment or, preferably, prevention for Alzheimer's disease.

Abstract
Alzheimer's disease (AD) neuropathology is characterised by abnormal aggregation in the brain of amyloid-β (Ab) and hyperphosphorylated tau, associated with inflammation and loss of neurons and synapses. The amyloid cascade hypothesis places abnormal aggregation of Ab at an early point in the pathogenesis of the disease, upstream of tau aggregation, although how Ab and tau interact in disease pathogenesis is unclear. In numerous studies of transgenic mouse models of AD, Ab immunisation has resulted in Ab plaque removal with functional benefits. We have performed a long term clinical and neuropathological follow up of patients with AD who were actively immunised with Aβ42 (Elan Pharmaceuticals). A total of 80 patients were enrolled in the study which started in the year 2000: 64 received Aβ42 peptide plus adjuvant and 16 received adjuvant alone. Post mortem neuropathology has identified substantial changes in the AD process in immunised patients including a lower Aβ load and a reduction in hyperphosphorylated tau, particularly in neuronal processes. In addition pathological "side-effects" were also observed, including: (i) altered microglial activation (ii) increased Aβ in the cerebral vasculature (cerebral amyloid angiopathy) and (iii) increased microhaemorrhages. In demonstrating that Ab immunisation can influence tau pathology, the findings provide support for the amyloid cascade hypothesis. However, the pathological "side effects" identified may be relevant to current immunotherapy trials, indicating that at least in a proportion of patients removing amyloid from the brain may have complications that need to be surmounted, for example by earlier intervention or use of immunotherapy as a preventative measure.

Speakers Short Biographies and Abstracts Day 1, Thursday 27 February

Journal of Neurology, Neurosurgery & Psychiatry Guest Speaker
Chair: Alan Carson

Presymptomatic treatment for the dementias: plausibility and perils

Professor Nick Fox's first degree was in Physics and Physiology from Cambridge. He subsequently graduated with honours in medicine from the University of London and then specialised in cognitive neurology. Since 1993, he has held Research Fellowships from the Alzheimer's Society and the Medical Research Council (MRC). He is currently Professor of Neurology and MRC Senior Clinical Fellow at the Institute of Nerulogy, University College London. He co-chairs the Research Advisory Committee of the Alzheimer's Society and is a member of Alzheimer's Research UK's Scientific Advisory Board. His clinical interests are focussed on young onset and familial dementias. His research is aimed at improving diagnosis and treatment of cognitive problems.

He has been involved in research on MRI use in Alzheimer's disease and related disorders for the last ten years. He has shown that AD has a pre-symptomatic period where hippocampal and global cerebral atrophy are already established and accelerating, and that subtle cognitive deficits pre-date overt symptoms.

He has been an advisor to the NIH and an invited expert to the FDA and EMEA. He is the only non-US member of the MR imaging core of the Alzheimer's Disease Neuroimaging Initiative (ADNI).
Research interests: dementia, MRI, familial Alzheimer's disease, early onset dementia

Abstract
Dementia is arguably the most urgent public health challenge confronting the developed world. The overarching priority is to find treatments that slow or halt disease progression and to apply those treatments as early as possible so as to have maximum impact on loss of cognitive and neurological function. There is increasing recognition that the neurodegenerative dementias (notably Alzheimer's disease - AD) are characterized by a long prodromal period where molecular pathology gradually accumulates and is followed by a neurodegenerative cascade that leads to neuronal dysfunction and ultimately irreversible damage and loss. Advances in imaging and in cerebrospinal fluid biomarkers mean that we see, measure and track pathological effects at the prodromal stage *in vivo.* This opens up a window for presymptomatic treatment.
Although the exact sequence, relationships and time course of pathological and biomarker changes in AD are unclear autopsy, imaging and CSF studies suggest that the preclinical phase may extend over many years. Cerebral amyloid deposition may predate clinical decline by more than a decade while hippocampal and brain atrophy rates become abnormal much closer to symptoms.
Recent failures of large phase 3 trials in so called "mild to moderate AD" have led to concerns the most – and potentially the only – effective therapies for neurodegenerative diseases will be those applied at the *earliest* stages. A 'self-perpetuating' aspect to neurodegeneration that is difficult to slow once established, may account, at least in part, for trial failures. Our ability to identify individuals at high risk of a moleculary defined dementia makes earlier intervention possible. Designing such secondary "prevention" trials raises a number of challenges however including how best to identify subjects for inclusion, how to assess how near to symptoms they are and how to assess efficacy. I will discuss issues related to the potential and the difficulties associated with these studies.

MEMBERS' PLATFORM PRESENTATIONS
Chair: Chris Butler

TREM2 variants increase risk of typical early-onset Alzheimer's disease but not of prion or fronto-temporal dementia

Authors *(presenter first):* Slattery CF, Beck J, Harper L, Adamson G, Abdi Z, Uphill J, Campbell T, Druyeh R, Mahoney CJ, Rohrer JD, Kenny J, Lowe J, Leung KK, Barnes J, Clegg SL, Blair M, Nicholas JM, Guerreiro RJ, Rowe JB, Ponto C, Zerr I, Kretzschmar H, Gambetti P, Crutch SJ, Warren JD, Rossor MN, Fox NC, Collinge J, Schott JM, Mead S
Objective: Variants in TREM2, a gene expressed on microglia and involved in CNS innate immunity, have recently been reported as rare but strong risk factors for Alzheimer's disease. Microglial mediated inflammation is implicated in several dementias. It remains unresolved whether TREM2 variant effects are selective for Alzheimer's disease or generic to neurodegeneration, and whether there is a distinct clinical phenotype associated. This study sought to provide the first systematic characterisation and detailed clinical phenotyping of TREM2 associated neurodegeneration across multiple dementias.
Method: We used next generation sequencing of the entire coding sequence (n=700), targeted Sanger sequencing of exon 2 (n=2634), p.R47H genotyping (n=3518) and imputation from genome wide association study data (n=3383) to determine TREM2 variants in large independent cohorts of patients with clinical diagnoses of predominantly early-onset Alzheimer's disease (n=1002), frontotemporal dementia (n=358), sporadic Creutzfeldt-Jakob disease (n=2437) and variant Creutzfeldt-Jakob disease (n=115). We describe the demographics, disease duration, neuroimaging, neuropsychological profiles and pathological findings in Alzheimer's disease cases with p.R47H and perform a case-control study comparing the detailed clinical presentations, rates of mini mental state examination decline and volumetric neuroimaging characteristics.
Results: We confirm previous reports that p.R47H is a risk factor for Alzheimer's disease vs. unselected controls (OR=2.19; 95%CI=1.04-4.51; P=0.03) and show that it does not significantly alter the risk for frontotemporal dementia (OR=0.81, 95%CI=0.09-3.36, P=1.00), sporadic or variant Creutzfeldt-Jakob disease (ORs=0.97-1.54 in three populations). We found a total of 49 non-synonymous alleles in 47 Alzheimer's patients, including one homozygous p.R47H case. Where age of symptom onset was available, Alzheimer's disease individuals with p.R47H TREM2 variants (n=12) were significantly younger at symptom onset than individuals with no TREM2 variants (n=551) (p.R47H: 55.2 years vs. nil TREM2 variants: 61.7 years, P=0.024). 10/12 (83%) of the p.R47H Alzheimer's disease cases had age-at-onset below 65 years, and 4/12 below 50 years. Heterozygous p.R47H Alzheimer's disease typically had a memory led presentation with disease duration, rates of mini mental state examination decline, and neuroimaging and pathological features indistinguishable from 'typical' sporadic Alzheimer's. The one notable exception was the homozygous p.R47H individual who had a behavioural presentation. Despite having typical Alzheimer's disease pathology confirmed on histology, they had an unusual degree of frontal atrophy, and relative sparing of the hippocampi.
Conclusion: Heterozygous p.R47H variants confer specific risk for Alzheimer's disease, and result in earlier symptom onset in a disease that appears otherwise typical for sporadic Alzheimer's disease.

Post-Ictal Psychosis - A Case Control Study

Authors *(presenter first):* Dr Georgy Pius, ST6 Trainee, North West Deanery,
Dr R J Hackett, Consultant Neuro-Psychiatrist, Salford Royal NHS Foundation Trust

Objective: The aim of this research study was:
1. To identify the risk factors for Post-Ictal Psychosis (PIP)
2. To describe the phenomenology of PIP and how it compares with Schizophrenia.
Method: The study design is a case control study comprising 22 subjects with a diagnosis of Epilepsy and PIP (cases) and 44 subjects with Epilepsy (controls). The setting of the project is a tertiary centre neurology outpatient clinic. Demographic and clinical characteristics including risk factors were reviewed retrospectively and compared across both the groups. Categorical data were analysed using Chi-Square analysis while quantitative data were analysed using Wilcoxon Signed Rank and Mann-Whitney U tests.
Results: Post-Ictal Psychosis (PIP) followed a cluster of seizures with a mean frequency of 2.19 in the cases (PIP) group and this was statistically significant when compared to that in the week prior to last clinic in the control group ($z = 3.874$, N-Ties = 19, $p = 0.000$). The mean duration of epilepsy prior to onset of PIP was 18.82 years (N = 22, Std. Dev. = 11.742). Family history of psychiatric illness was similar in both the groups ($\chi^2 = 0.000$, df = 1, $p = 1.000$). There was a tendency towards association of focal epilepsy to PIP compared to the control group (df = 1, $p = 0.143$). Looking at the phenomenology of PIP, the duration of psychotic episode ranged from 12 hours to 2 weeks and the clinical features comprised mainly of auditory and visual hallucinations, religious and grandiose delusions and significant violent behaviour.
Conclusion: The author's study echoes previous studies' findings in areas of increased seizure frequency and chronicity of epilepsy prior to Post-Ictal Psychosis (PIP). However there is no association of family history of psychiatric illness to PIP and this along with the finding of a tendency towards an association of focal epilepsy to PIP suggests that epileptic factors play a more significant role than genetic factors in the emergence of PIP. Looking at the phenomenology of PIP, the duration of psychosis is of a transient and episodic nature. However symptoms of PIP are remarkable for its absence of 1st rank symptoms and negative symptoms which are characteristic of Schizophrenia. The other interesting aspect is the significance of religious delusions and violent behaviour both of which have been described as epileptic personality traits in the literature before.

MEMBERS' PLATFORM PRESENTATIONS
Chair: Chris Butler

Joint hypermobility and autonomic hyperactivity: relevance to neurodevelopmental disorders

Authors *(presenter first:* JA Eccles1, 2, V Iodice3,4, N G Dowell1, A Owens3,4 L Hughes2, S Skipper2, Y Lycette2,K Humphries2 , NA Harrison 1,2,5, C J Mathias3,4, HD Critchley1,2,5

1.Psychiatry, Brighton and Sussex Medical School, Brighton
2.Sussex Partnership NHS Foundation Trust, Brighton
3 National Hospital Neurology and Neurosurgery, UCL NHS Trust, London
4.Instiute of Neurology, University College London.
5.Sackler Centre for Consciousness Science, University of Sussex, UK

Objective: To test the hypothesis that Joint hypermobility and autonomic dysfunction are over-expressed within neurodevelopmental disorders. Joint hypermobility is a widespread poorly recognized connective tissue condition with affected individuals overrepresented among panic and anxiety disorders, irritable bowel syndrome, fibromyalgia, and chronic fatigue. The relevance of hypermobility to neuropsychiatric disorders of developmental origin is currently unknown, despite anecdotal case reports and clinical suspicion of a link. Autonomic nervous system dysregulation, typically postural tachycardia syndrome is often found in hypermobile individuals. Interestingly, differences in amygdala and superior temporal cortex anatomy have been reported in hypermobile populations and functional abnormalities in patients with autism.
Method:Thirty-seven adults with neurodevelopmental disorder, 205 patients attending general psychiatric clinics without neurodevelopmental diagnosis and 29 healthy controls were recruited. Hypermobility was assessed using the Beighton scale(BS) and autonomic symptoms using the Autonomic Symptoms and Quality of Life Score(ASQoLS: orthostatic, gastrointestinal, bladder, secretomotor, sudomotor and sleep domains.
Results:The neurodevelopmental cohort had a mean age of 34.6 years(27 male). Nineteen had Attention Deficit Hyperactivity Disorder(ADHD), 4 Autistic Spectrum Disorder(ASD), 1 Tourette Syndrome(TS) and the remainder combinations of ADHD, ASD and TS. Nine had co-morbid affective disorder. Eighteen patients(48.6%) were classified as hypermobile(BS>=4) compared to 67/204(32.7%) in the general psychiatric group(p=0.048) and 3/29(10.3%) in healthy controls(p=0.007) and this prevalence was also significantly higher that reported in a large general population cohort (1156/6022, 19.19%, p=<0.001). Mean autonomic dysfunction score was significantly higher in the neurodevelopmental cohort compared to controls (mean±SEM: neurodevelopmental disorder patients, 45.8 ±4.86; controls, 8.5±1.62). This effect was seen across all sub-scales of the ASQoLS. Total autonomic dysfunction score did not differ significantly between neurodevelopmental cohort and the general psychiatric group, however neurodevelopmental disorder patients had significantly higher scores on orthostatic and gastrointestinal disturbance subscales.
Conclusion:We demonstrate for the first time that rates of hypermobility and symptoms of autonomic dysfunction are particularly high in adults with neurodevelopmental diagnoses. It is likely that the importance of hypermobility and autonomic dysfunction to the generation and maintenance of psychopathology in neurodevelopmental disorders is poorly appreciated. Work underway(autonomic testing, fMRI) will test the hypothesis that autonomic reactivity and interoceptive sensitivity predispose to the expression of psychiatric symptoms, particularly anxiety. It is further hypothesized that inefficient neural co-ordination of efferent autonomic drive with imprecise interoceptive representations may be amplified in hypermobile individuals. In hypermobility, this mechanism might explain increased vulnerability to stress sensitive and developmental neuropsychiatric conditions.

NEUROPSYCHIATRY RESEARCH UPDATE
Chair: Markus Reuber

Impulse Control

David Okai is currently a Locum Consultant in Psychological Medicine in Oxford, and a Clinical Research Associate in Neuropsychiatry at the Institute of Psychiatry. He trained in psychiatry at the Bethlem and Maudsley Hospital, where he also undertook a diploma in Cognitive Behavioural Therapy (CBT). He has spent the last 4 years involved in the evaluation of a CBT-based randomised-control trial dealing with the management of Impulsive Compulsive behaviours in Parkinson's disease. David is a member of the Movement Disorders Task Force on Rating Scales in ICBs, and is also a member of the Clinical Advisory Panel of Parkinson's UK. In 2012 he was awarded a Movement Disorders Society, Junior Investigator award for current contribution to the field of ICBs in PD. He has published book chapters on neuropsychiatry and psychology has a particular interest in factors such as the ecological validity of psychological tests of dysexecutive syndrome. He has his viva for an MD(res) in the assessment and management of PD-ICBs three days after this presentation!

Abstract Impulsive Compulsive Behaviours (ICBs) in Parkinson's disease (PD) are motivation-based behaviours that involve repetitive occurrences of impulsive and uncontrolled activity. There is established recognition of an association with the dopaminergic medication, used to treat the motor disability, in some sufferers of PD. This increased risk has sparked a decade of research into the epidemiology, biological, and psychosocial mechanisms associated with PD-ICBs. Nonetheless (neuro)psychiatric classification is currently inconsistent and unclear. Additionally little is known regarding the best evidence-based clinical approach to management of these behaviours. The talk will address new developments in the understanding of assessment and management of PD-ICBs including:

1. Conceptual and methodological problems underlying ICB diagnoses.
2. Existing methods of assessment and rating of ICBs.
3. Discussion of new scales for ICB severity assessment.
4. Evaluation of the effects of a novel cognitive behavioural therapy based intervention for ICB management.
5. Demographic and clinical variables predictive of ICB outcome.

There will also be comparison of the biopsychosocial aspects of PD-ICB presentation to Impulse Control Disorder presentations in the general population and those who suffer from Substance Use Disorders.

Do Cholinesterase inhibitors work?

Dr Hugh Rickards
Consultant Neuropsychiatrist, Department of Neuropsychiatry, The Barberry, Birmingham. His main interests are the neuropsychiatry of motor disorder (in particular, Tourette syndrome and Huntington's disease). He also runs an MSc in clinical neuropsychiatry from the University of Birmingham.

Abstract Acetylcholinesterase inhibitors (ACHEI) are used for a range of brain disorders, particularly dementia. I reviewed the available literature to find an answer to the question of their effectiveness and efficacy based on the question "if a patient were sitting in front of me with this disorder, how best could I inform the discussion about whether or not to prescribe these medications". The evidence from schizophrenia is that they have no effect. The evidence from Parkinson disease and related Lewy body disorders suggests a mild beneficial effect on cognition of doubtful clinical significance with a higher chance of adverse events. This was better in the purer Parkinson and dementia (PDD) group. There was a similar, but less marked, cognitive benefit in vascular cognitive impairment. Trials in MCI showed a trend to benefit but no difference in "conversion to dementia" rate. In Alzheimer dementia the evidence was strongest and showed a mean difference between treatment and placebo of 1.4 points on the MMSE with no effect on behaviour. The DOMINO trial showed, surprisingly, that patients on ACHEI were less likely to withdraw from treatment than those on placebo. Although patients in this trial taking ACHEI did better in cognition and function than those who weren't, all the groups deteriorated to some extent. The CALM-AD study showed that ACHEI did not affect behaviour in a group of patients who had BPSD. Finally, a recent meta-analysis calculated the probability of a randomly chosen patient with dementia achieving significant benefit on treatment when compared to placebo (with 1.0 being "certain of benefit" and 0.0 being "certain of no benefit"). For ACHEI in AD the probability of cognitive benefit was 0.58 and for global functioning 0.55. The probability of harm, leading to withdrawal was 0.56. In summary, "probability of benefit" was roughly equal to "probability of harm". However, patients and carers may have specific viewpoints on these probabilities in terms of their own values and these should be discussed.

Speakers Short Biographies and Abstracts Day 2, Friday 28 February

Neuropsychiatric Manifestations/Encephalitis
Chair: Eileen Joyce

Recognising and Diagnosing inflammatory brain disease

Professor Neil Scolding trained in neurology in Cambridge and in London (National Hospital for Neurology & Neurosurgery); he was a Consultant Lecturer in Cambridge before coming to Bristol in 1999. His research centres on multiple sclerosis & related diseases, and on developing stem cell repair treatments. He has published over 200 research papers and three Neurology textbooks.

Abstract

In contrast to 'conventional' neuro-inflammatory or neuro-immune diseases like multiple sclerosis, CNS involvement in systemic inflammatory disorders can be very difficult to recognise, and even more difficult to confirm. Vasculitis, lupus, sarcoidosis and other disorders all may involve the brain and/or spinal cord, and in each instance, presentation with exclusively neurological features is far from unknown. In most such disorders, there are no diagnostic clinical features; many of these diseases mimic each other. Once suspected, it is commonly the case that no tests are available that categorically prove the disease to be present – other than cerebral biopsy, and even this has limited sensitivity and clearly carries a certain risk. A number of investigations often considered valuable are neither sensitive nor specific, and are prone to over-interpretation. CNS inflammatory diseases may be aggressive, seriously disabling or even fatal – and yet most are highly treatable.

Clinical nomenclature and definitions have not helped a complex field – many are at best confusing, and often wholly misleading. Vasculitis, for example, is neither a diagnosis nor a disease, but rather a histopathological description (intramural inflammation, often but not invariably with additional perivascular infiltrates, accompanied by necrosis of the blood vessel wall). Lupoid sclerosis, a term firmly embedded in the literature, probably does not exist; lupus vasculitis is a 'diagnosis' in rather commonly usage, but in fact very rarely is it a useful or accurate description of CNS lupus-related disease.

Nonetheless, progress in this difficult area is being made. Clinical scenarios when such disorders ought to feature in a differential diagnosis are better defined, and symptoms or signs that help raise suspicion of individual disorders may be putatively put forward (based in the main on lessons from internal medicine rather than neurology). Approaches to confirming CNS inflammatory diagnoses can be suggested, with increasing recognition of the limitations of investigative techniques. Finally, and admittedly again almost wholly informed by clinical research in systemic inflammatory disorders, rather than good quality clinical trials in patients with neurological disease, treatment regimes are improving. A brief overview of these changes will be presented.

Speakers Short Biographies and Abstracts Day 2, Friday 28 February

Paraneoplastic neurological disorders

Dr Jeremy Rees qualified in 1988 from University College and Middlesex Medical School with distinctions in Medicine, Surgery and Therapeutics and numerous prizes including the Broderip Scholarship. General professional training in various postgraduate London teaching hospitals. Awarded an MRC Clinical Training Fellowship at Guy's Hospital, investigating the association between C jejuni infection and Guillain-Barre syndrome towards a PhD. Completed clinical neurology training at the Royal Free Hospital, National Hospital for Neurology and Neurosurgery and St Thomas' Hospital. Subspecialty in neuro-oncology with a period of training at Memorial Sloan Kettering Cancer Center, New York. Appointed consultant neurologist in 1999 at the National Hospital for Neurology and Neurosurgery and Senior Lecturer at UCL Institute of Neurology, Holds Consultant posts at Royal Marsden Hospital, Mount Vernon Cancer Centre and Royal National Orthopaedic Hospital, Stanmore. He is the Clinical Lead for the Neuro-oncology service in the UCLH Trust. His research interests are: low-grade gliomas and paraneoplastic neurological syndromes.

Abstract

Paraneoplastic Neurological Disorders (PND) are uncommon but important because they frequently present before a cancer is diagnosed and because they cause severe neurological disability. Current thinking is that they are caused by an autoimmune response to 'onconeural' antigens, shared by the tumour and the nervous system, although the precise immunopathogenic mechanism is unknown. It is likely that there is an important cellular immune response as evidenced by the presence of lymphocytic infiltration and activated cytotoxic T lymphocytes, found in the CSF of affected patients.

PND may affect any part of the nervous system either focally (e.g. cerebellar degeneration) or diffusely (e.g. encephalomyelitis). Both the Central and Peripheral Nervous System may be affected, with antigenic targets being either intracellular (both nuclear and cytoplasmic) or extracellular (receptors and ion channels). As a general rule, PND associated with antibodies against intracellular targets cause predominantly CNS disorders while those associated with antibodies against extracellular antigens cause predominantly neuromuscular disorders.

PND affecting the CNS are commonly associated with specific anti-neuronal antibodies, which are present in both serum and CSF. A suspected diagnosis of PND should prompt a search for what may be a very small tumour, sometimes not visible with conventional imaging techniques. Fluoro-deoxyglucose - Positron Emission Tomography (FDG-PET) scanning is helpful in this regard as it can visualise tumours down to a resolution of 6-8 mm anywhere in the body and is sometimes positive when chest X-ray and CT are negative. Current recommendations are that the search for cancer should continue for up to 5 years after diagnosis, except for Lambert Eaton Myasthenic Syndrome (LEMS) associated with Small cell Lung Cancer, where 2 years are sufficient.

Most CNS syndromes respond poorly to immunomodulatory treatment although occasional improvement is seen when the underlying tumour is treated. In contrast, disorders affecting the Neuromuscular Junction e.g. LEMS do improve with treatments that remove the relevant antibodies, directed against Voltage-Gated Calcium Channels.

Traditonally, PNS affect older patients with malignant tumours. Recently the spectrum of paraneoplasia has been broadened to include younger patients with benign tumours e.g. ovarian teratoma presenting with prodromal flu-like symptoms, psychiatric disturbance progressing to coma, movement disorders, autonomic instability and respiratory failure. These disorders are associated with antibodies directed against NMDA receptors in the hippocampi and improve with removal of the teratoma and plasma exchange.
The prognosis for the majority of PND is poor, even if the tumour is detected and treated, and patients may live in a severely disabled state for many years.

Speakers Short Biographies and Abstracts Day 2, Friday 28 February

Infectious encephalitis and other CNS infections with psychiatric presentations

Professor Tom Solomon

Director - Institute of Infection and Global Health; Chair of Neurological Science; Head - Brain Infections Group; MRC Senior Clinical Fellow, University of Liverpool and Walton Centre for Neurology and Neurosurgery. Tom studied medicine in Oxford, before working on brain infections in Vietnam, with a Wellcome Trust Training Fellowship. He then became Clinical Lecturer at the University of Liverpool, and was awarded a Wellcome Trust Career Development Fellowship, with two years in the USA. Returning to Liverpool, he became Senior Lecturer, MRC Senior Clinical Fellow, then Chair of Neurological Science, and Director of the Liverpool Institute of Infection and Global Health. Tom's group studies brain infections of major global importance with collaborations in Asia, Africa, the UK and the US, close work with WHO. The group has more than £8M in funding principally from MRC, Wellcome Trust, Gates Foundation, and NIHR. Facebook.com/RunningMadProf Twitter: @RunningMadProf

Abstract

Central nervous system infections can cause both acute and chronic presentations with neuropsychiatric symptoms. Acute CNS infections, such as encephalitis not infrequently have behavioural disturbances as part of the presenting syndrome, and indeed some patients are initially thought to have psychiatric illness. Other infections that have more insidious presentations, such as tuberculous meningitis, may initially present as a dementing illness. In this talk some of the more common CNS infections that present with neuropsychiatric disease will be considered through a series of interactive case presentations; in addition there will be one or two two rarities for reference. Some of the key red flags that should make one consider infection will be highlighted, as well as essential early steps in patient management. In addition the latest relevent research from the www.BrainInfectionsUK.org portfolio will be presented, along with pertinent studies from elsewhere.

References

1. Kneen R, Michael BD, Menson E, Mehta B, Easton A, Hemingway C, Klapper PE, Vincent A, Lim M, Carrol E, Solomon T, National Encephalitis Guidelines D, Stakeholder G. Management of suspected viral encephalitis in children - Association of British Neurologists and British Paediatric Allergy, Immunology and Infection Group national guidelines. The Journal of infection. 2012;64:449-77.

2. Solomon T, Michael BD, Smith PE, Sanderson F, Davies NW, Hart IJ, Holland M, Easton A, Buckley C, Kneen R, Beeching NJ, National Encephalitis Guidelines D, Stakeholder G. Management of suspected viral encephalitis in adults--Association of British Neurologists and British Infection Association National Guidelines. The Journal of infection. 2012;64:347-73.

3. Thwaites G, Fisher M, Hemingway C, Scott G, Solomon T, Innes J. British Infection Society guidelines for the diagnosis and treatment of tuberculosis of the central nervous system in adults and children. The Journal of infection. 2009;59:167-87.

4. Letendre S. Central nervous system complications in HIV disease: HIV-associated neurocognitive disorder. Topics in antiviral medicine. 2011;19:137-42.

Speakers Short Biographies and Abstracts Day 2, Friday 28 February

BNPA Medical Lecture
Chair: Adam Zeman

Autoimmune encephalitis

Angela Vincent qualified as a doctor at Westminster Hospital Medical School but after one year post qualification residence, she enrolled to do an MSc in Biochemistry at University College London. Subsequently, working with Ricardo Miledi FRS, she became involved in some of the earliest studies on acetylcholine receptors in myasthenia gravis and congenital myasthenic syndromes, and began a long partnership with John Newsom-Davis (later FRS), first at the Royal Free Hospital in London and then at the newly-established Weatherall Institute of Molecular Medicine in Oxford. Since Newsom-Davis' retirement in 1998 (and unexpected death in 2007), she has led the neuroimmunology research in Oxford. She is an honorary consultant in immunology and has established a national and international referral centre for the diagnosis of immune-mediated neurological diseases. From 2005-2008, she was Head of Department of Clinical Neurology. She is now Emeritus Professor of Neuroimmunology in Oxford and an Emeritus Fellow of Somerville College and continues to actively run the clinical service and to lead the Neuroimmunology Group.

She has an Honorary degree from the University of Bergen (2004), and is a Fellow of the Academy of Medical Sciences (FMedSci, 2002) and a Fellow of the Royal Society (FRS, 2011), as well as Honorary Member American Association of Neuromuscular & Electrodiagnostic Medicine, and Honorary Fellow of the American Neurological Association. She has received the Duchenne-Erb Award, German Muscle Society, Darmstadt (2009), and the Medal of the Association of British Neurologists (2009). She was previously President of the International Society of Neuroimmunology (2001-2004). She has co-edited four books including one with Russell Dale on pediatric neuroimmunology (2010). Her major clinical interest is in the role of auto-antibodies to ion channels and receptors in peripheral and central disorders, and in advancing the diagnosis of these immunotherapy-responsive conditions. Her research interests include neuromuscular junction disorders, models of immune-mediated CNS diseases, and the influence of maternal antibodies in neurodevelopmental disorders.

Abstract
It is now accepted that there are antibody-mediated diseases of both the peripheral and central nervous systems. Myasthenia gravis remains the prototype autoimmune disease of the neuromuscular junction, but subsequent studies have revealed antibodies to other peripheral and autonomic targets. In the 1990s, antibodies to voltage-gated potassium channel complexes were identified in acquired neuromyotonia, a condition caused by peripheral nerve hyperexcitatibility that leads to muscle fasciulations, muscle cramps and pain. Somewhat surprisingly, the same antibodies were identified in relatively acute-onset central nervous system disorders such as Morvan's syndrome and limbic encephalitis. It turned out that the potassium channel antibodies were mainly directed at other proteins that are complexed with the channels in situ, such as LGI1 and CASPR2. These proteins help localise (CASPR2) and modify (LGI1) potassium channel function, and the antibodies bind to extracellular epitopes and are pathogenic in vitro. Tumours can be found in a proportion of each of these conditions, but the proportion varies from <10% to around 50%. Thymomas are the most common. In 2007, antibodies to NMDA receptors (NR1 principally) were identified and subsequently found quite commonly in younger patients, often women and small children, who have a very complicated form of encephalitis that results in psychiatric and movement disorders. Ovarian teratomas are common in the adult females but rare in children. Other antibodies have now been discovered, each one directed at a specific receptor or ion-channel related associated protein, although so far the associated diseases are fairly rare. Antibodies to glycine receptors are associated with a form of stiff person plus, usually termed progressive encephalomyelitis with rigidity and myoclonus (PERM), a condition which is well described in the literature and can be life threatening. Now it is recognised in more patients with a greater breadth of clinical symptoms. Each of these diseases shows a very good respond to immunotherapies such as steroids, plasma exchange, intravenous immunoglobulins. If the response is poor, second line therapies such as rituximab and/or cylclophosphamide are tried. Some require longer term immunosuppression with azathioprine or mycophenolate. Altogether there is a growing field of immunotherapy-responsive neurological diseases which need to be recognised by the clinicians and treated appropriately. There are now many neurological presentations in which the possibility of an autoimmune disease needs to be considered, and this is beginning to apply to those that are less clearly "organic".

Speakers Short Biographies and Abstracts Day 2, Friday 28 February

THE WELLCOME TRUST DEBATE
Chair: Peter White

This house believes that neurology and psychiatry should be one medical discipline

Debate Chair: Peter D White, BSc, MBBS, MD, FRCP, FRCPsych Professor of Psychological MedicineI am Professor of Psychological Medicine, honorary consultant liaison psychiatrist at St Bartholomew's hospital and co-lead the chronic fatigue syndrome (CFS) service there. My clinical work involves assessing and caring for patients who have both a physical and mental health problem, such as cancer and depression, as well as co-leading an assessment and treatment service for patients suffering from chronic fatigue syndrome (CFS). I qualified in medicine at St Bartholomew's Hospital Medical College, and then trained in general medicine in Southampton, after which I received my psychiatric training at the Maudsley and St Bartholomew's Hospitals.

Geraint Rees is a Professor of Cognitive Neurology at University College London, where he directs the Institute of Cognitive Neuroscience. His research interests focus on understanding the neural basis of human consciousness in health and disease, using functional neuroimaging techniques in combination with other methodologies. Recently he has pioneered new approaches to analysing functional brain images to individuate the contents of consciousness, and has written and spoken on the potential moral and ethical implications of such techniques. His work has been internationally recognised by award of the Young Investigator Medal of the Organisation for Human Brain Mapping, the Experimental Psychology Prize; and he has given the Francis Crick lecture at the Royal Society and the Goulstonian lecture at the Royal College of Physicians. In 2010 he was elected a Fellow of the Academy of Medical Sciences.

In addition to his research interests, Geraint has a track record of personal and professional commitment to improving clinical academic training both at UCL Partners and throughout the UK. He oversees the NIHR Academic Clinical Fellow and Clinical Lecturer programmes across UCL Partners and is the lead for postgraduate education & training within the UCL/UCLH Comprehensive Biomedical Research Centre and the UCL Faculty of Biomedical Sciences. Nationally, he is a member of the Medical Programme Board for England, Deputy Chair of the BMA's Medical Academic Staff Committee, and a member of the MRC Neurosciences & Mental Health Board. He actively contributes to development of national policy on clinical academic training.

Professor Sir Simon Wessely is Professor of Psychological Medicine at the Institute of Psychiatry, King'sCollege London and Head of its department of psychological medicine, Vice Dean for Academic, Teaching and Training at the Institute of Psychiatry, as well as Director of the King's Centre for Military Health Research. He is also honorary Consultant Psychiatrist at King's College Hospital and the Maudsley Hospital, as well as Civilian Consultant Advisor in Psychiatry to the British Army. He was knighted in the 2013 New Year Honours for services to military healthcare and to medicine More recently, Wessely's work was the first to show that service in the 1991 Gulf War had had a significant effect on the health of UK servicemen and women. Other work suggested a link to particular vaccination schedules used to protect against biological warfare, and also a link with psychological stress. His group also confirmed that classic psychiatric injury, post-traumatic stress disorder (PTSD), was not a sufficient explanation for the observed health problems.

Members' Posters

1. Title: **Research trends in British neuropsychiatry over the last decade**
Authors: Andrea E. Cavanna, The Barberry National Centre for Mental Health, Department of Neuropsychiatry, 25 Vincent Drive Birmingham B15 2FG, United Kingdom

2. Title: A systematic review and meta-analysis of the efficacy and safety of sodium valproate for "off-label" indications in mental health
Authors: Piriyankan Ananthavarathan, Faculty of Medicine, Imperial College London, Verity Leeson, Faculty of Medicine, Imperial College London Thomas Barnes, Faculty of Medicine, Imperial College London

3. Title: Pharmacological interventions for depression in people with traumatic brain injury: Systematic Review
Authors: JJ Vattakatuchery, N Lathif, J Joy, A Cavanna, H Rickards, 5 Boroughs NHS Foundation Trust, Wakefield House, Guardian Street, Warrington, WA51GG

4. Title: An audit of the diagnosis and costs associated with inpatients with functional neurological symptoms in a district general hospital
Authors: Adjei M, Coebergh JA, Email: m0901784@sgul.ac.uk

5. Title: Is there a link between Non-Epileptic Attack Disorders (NEAD) and Personality Disorders (PD)? - A Systematic Review.
Authors: Dr Obiajulu Chudi Okoye, Email: objmichael@hotmail.com

6. Title: Identifying the shared neurobiological underpinnings of negative urgency and neuroticism
Authors: Muhlert N, Lawrence AD, Cognitive Neuroscience, Cardiff University, United Kingdom

7. Title: The prevalence of tic disorder in an electoral district of Mauritius
Authors: Vinu Chummun, Vanisha Seetaram, Hugh Rickards. Email: vinu@doctors.org.uk

8. Title: The role of alexithymia in the development of functional motor symptoms (conversion disorder)
Authors: Benedetta Demartini, Panayiota Petrochilos, Lucia Ricciardi, Gary Price, Mark J EdwardS, Eileen Joyce, Sobell Department, UCL Institute of Neurology, Queen Square, London WC1N 3BG

9. Title: Autoantibodies in Alzheimer disease;
Authors: 1- Ramin Nilforooshan: Brain Science research Unit, ACU, Holloway Hill, Chertsey, KT16 0AE, UK. ramin.nilforooshan@sabp.nhs.uk
2- Angela Vincent Neurosciences Group, Department of Clinical Neurology, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DS, UK angela.vincent@sabp.nhs.uk
3- Jessica Eccles, Brighton and Sussex Medical School, BN1 9RR, UK. J.eccles@bsms.ac.uk
4- Rosie Pettingill Neurosciences Group, Department of Clinical Neurology, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DS, UK R rosie.pettingill@imm.ox.ac.uk
5-Philippa Pettingill Neurosciences Group, Department of Clinical Neurology, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DS, UK philippa.pettingill@imm.ox.ac.uk
6- Lamia Ali Brain Science research Unit, ACU, Holloway Hill, Chertsey, KT16 0AE, UK lamia.ali@sabp.nhs.uk
7- Naji Tabet: Cognitive Treatment and Research Unit, Sussex Partnership NHS Foundation Trust, Brighton and Sussex Medical School, BN1 9RR, UK N.T.Tabet@brighton.ac.uk

10. Title: Do prenatal and perinatal complications influence tic severity in patients with Gilles de la Tourette syndrome?
Authors: Taylor KA, Stern JS, Williams D, Simmons HS, Robertson MR , Department of Neurology, Atkinson Morely's Wing, St. George's Hospital, Blackshaw Road, London. SW17 0QQ

11. Title: Psychosocial characteristics of Tourette syndrome in older adults attending a specialist clinic
Authors: Man CHA, Stern JS, Gharatya A, Williams D, Simmons HS, Robertson MM, Department of Neurology**,** Atkinson Morley's Wing, St George's Hospital, Blackshaw Road, London SW17 0QQ

Members' Posters

12. Title: Suicidality in patients with Tourette's syndrome
Authors: Gharatya AK, Stern JS, Man CHA, Williams D, Simmons HS, Robertson MM**,** Department of Neurology, Atkinson Morley's Wing, St George's Hospital, Blackshaw Road, London, SW17 0QQ

13. Title: Role of catecholaminergic and cholinergic drugs in management of cognitive deficits in adults with Traumatic Brain Injury: a systematic review
Authors: Inderbir Singh Sidhu, ST6 Psychiatry of Intellectual Disabilities, South Essex Partnership University NHS Foundation Trust, Clinical Base 3 Heath Close, Billericay, Essex , CM12 9NW, United Kingdom

14. Title: Prescribing practices in adults with Tourette syndrome
Authors: Graham Blackman, Andrea E. Cavanna, Department of Neuropsychiatry, BSMHFT and University of Birmingham, The Barberry National Centre for Mental Health, 25 Vincent Drive Birmingham

15. Title: Multidisciplinary inpatient programme for functional neurological symptoms: a prospective study assessing efficacy and predictors of good outcome.
Authors: Benedetta Demartini, Panayiota Petrochilos, Mark J Edwards, Eileen Joyce, Sobell Department, UCL Institute of Neurology, Queen Square, London WC1N 3BG, UK

16. Title: Habenula responses during appetitive and aversive conditioning in Major Depressive Disorder
Authors: Rebecca P. Lawson, Camilla L. Nord, Ben Seymour, David L. Thomas, Raymond J. Dolan, Peter Dayan, Nikolaus Weiskopf & Jonathan P. Roiser, Wellcome Trust Centre for Neuroimaging, 12 Queen Square, London, WC1N 3BG, United Kingdom

17. Title: Clinical features of depression in Huntington's disease - A cross sectional study comparing the clinical features of depression in patients with Huntington's disease and in patients without Huntington's disease
Authors: Dr Joby Scaria, Dr David Craufurd, ST4 General Adult Psychiatry, NorthWestern Deanery

18. Title: Emotional Processing in Adults with Functional Neurological Symptoms
Authors: Hannah R Reynolds, Lisa A Jones, Hugh E Rickards, Email: HRR929@bham.ac.uk

19. Title: EEG Biofeedback Therapy for ADHD: A Systematic Review
Authors: Dr Ashley Liew, Email: ashleyliew@gmail.com

20. Title: Severe Refractory Tourette Syndrome
Authors: Colquhoun M, Stern JS , Collicott N, Williams D , Grabecki K, Simmons H , Robertson MR, Department of neurology, Atkinson Morley's Wing, St. George's Hospital, Blackshaw Road, London

21. Title: Facial emotion expressiveness and facial emotion recognition in Parkinson's disease: how much does alexithymia count?
Authors: Lucia Ricciardi, Matteo Bologna, Diego Ricciardi, Bruno Morabito, Francesca Morgante, Daniele Volpe, Davide Martino, Alessandro Tessitore, Massimiliano Pomponi, Anna Rita Bentivoglio, Roberto Bernabei, Alfonso Fasano. Department of Clinical and Experimental Medicine, University of Messina, via Consolare Valeria, 98125 Messina, Italy.

22. Title: Neural Bases of Musical Hallucinations
Authors: Sukhbinder Kumar, William Sedley, Gareth R Barnes, Sundeep Teki, Karl J Friston, Timothy D Griffiths, Auditory Group, Institute of Neuroscience, Newcastle University Medical School, Newcastle University, Newcastle upon Tyne,NE2 4HH,United Kingdom

23. Title: Misophonia: a Disorder of Emotion Processing of Sounds
Authors: Sukhbinder Kumar, Olana Hancock, Thomas Cope, William Sedley, Joel Winston, Timothy D Griffiths, Auditory Group Institute of Neuroscience, Newcastle University Medical School, Newcastle upon Tyne, NE2 4HH, United Kingdom.

24. Title: Interoceptive sensitivity and sense of body ownership in patients with functional neurological symptoms
Authors: Lucia Ricciardi, Benedetta Demartini, Laura Crucianelli, Mark J. Edwards, Aikaterini Fotopoulou. Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology, University College London

Members' Posters

1. Title: **Research trends in British neuropsychiatry over the last decade**

Authors: Andrea E. Cavanna, The Barberry National Centre for Mental Health, Department of Neuropsychiatry, 25 Vincent Drive Birmingham B15 2FG, United Kingdom

Objective: Since its establishment in 1987, the British Neuropsychiatry Association (BNPA) has gathered clinicians and researchers with special interest in neuropsychiatry and behavioural neurology. The association regularly invites abstract submissions on neuropsychiatry research topics to its yearly Annual General Meeting (AGM). These spontaneous contributions mainly reflect research interests of the BNPA members and their analysis can provide an overview on the recent research trends of British neuropsychiatry.
Method: The author reviewed the BNPA-AGM Programmes and Abstract Books, as well as the Proceedings published in the Journal of Neurology, Neurosurgery and Psychiatry over the last ten years (2004-2013), in order to identify the research topics of all abstracts accepted for oral or poster presentation.
Results: A total of 186 research abstracts were presented at BNPA-AGM over the last decade. The mean number of abstracts was 19 per year. The year with the highest number of abstracts was 2011 (n=26), whilst the year with the lowest number was 2004 (n=7). The vast majority of abstracts (n=173, 93.0%) were presented by a corresponding author based in the United Kingdom. The overall distribution of research topics in these abstracts was as follows: Tourette syndrome (n=36, 20.4%), epilepsy (n=26, 14.8%), functional neurological symptoms (n=19, 10.8%), dementias (n=11, 6.2%), acquired brain injury (n=10, 5.7%), Parkinson disease (n=9, 5.1%), amyotrophic lateral sclerosis (n=8, 4.5%), Huntington disease (n=6, 3.4%), multiple sclerosis (n=5, 2.8%), attention-deficit and hyperactivity disorder (n=3, 1.7%), catatonia (n=2, 1.1%), autistic spectrum disorders (n=2, 1.1%), autoimmune limbic encephalitis (n=2, 1.1%), sleep disorders (n=2, 1.1%), plus systemic lupus erythematosus, chronic fatigue syndrome, headache and addiction (all n=1, 0.6%). Three of these abstracts focused on more than one neuropsychiatric conditions: n=2 on epilepsy and functional neurological symptoms; n=1 on epilepsy and Tourette syndrome. The remaining abstracts (n=31, 17.6%) did not focus on a particular neuropsychiatric condition: these abstracts reported investigations of neurobiological mechanisms/cognitive processes in patients with common psychiatric disorders (n=10) and healthy subjects (n=10), neuropsychiatry service evaluations (n=5), single case reports/case series (n=3) and psychometric studies on the development of scales for behavioural/cognitive symptoms (n=2).
Conclusion: The last decade has seen a considerable increase in the number of scientific abstracts presented at the BNPA by researchers based in UK, along with a diversification in the research topics. Specifically, in recent years there has been an increased interest in movement disorders, epilepsy and functional neurological symptoms, reflecting important advances in clinical research focusing on these neuropsychiatric conditions.

2. Title: A systematic review and meta-analysis of the efficacy and safety of sodium valproate for "off-label" indications in mental health

Authors: Piriyankan Ananthavarathan, Faculty of Medicine, Imperial College London, Verity Leeson, Faculty of Medicine, Imperial College London. Thomas Barnes, Faculty of Medicine, Imperial College London

Objective: Valproate is currently licensed for use in epilepsy and, in mental health practice, for the treatment of acute mania. It is also commonly used for the maintenance treatment of bipolar disorder, though on an unlicensed basis. Though few guidelines exist to support its wider "off-label" use, clinicians have extrapolated from licensed indications to a range of others that encompass mood instability or problems with impulse control at their core.
We aimed to assess available evidence on the efficacy and risks of valproate in adult psychiatric practice, either as monotherapy or adjunctive treatment, in unlicensed indications, aside from bipolar disorder.
Method: A systematic review was conducted on 193 published paper (31 randomised control trials) using the MEDLINE, EMBASE and Cochrane databases to identify the various unlicensed uses of valproate in mental health care, and analyse extracted data on valproate's efficacy and safety. A meta-analysis was undertaken on extracted data where appropriate.
Results: Relevant literature was identified considering the unlicensed uses of valproate in aggression, alcohol-associated disorders, borderline personality disorder, dependence disorders (including cannabis, cocaine and nicotine), depressive disorders and schizophrenia.
Conclusion: Analysis of results depicts a limited efficacy of valproate in schizophrenia, acute alcohol withdrawal, depressive disorders, pathological gambling, suicidal behaviour, and benzodiazepine, cannabis and cocaine dependence. Nevertheless, evidence exists to suggest efficacy in hostility amongst patients with acute alcohol-associated hallucinosis or schizophrenia, and in aggressive behaviour, either alone, or in the context of comorbid bipolar disorder or personality disorder.
Common side effects of valproate include elevated liver enzymes, thrombocytopenia and weight gain. The documentation of adverse events and side effects considering valproate is inconsistent. It would improve our understanding of the risk-benefit balance of valproate if future studies reported "common" and treatment -emergent side effects in a more standardised manner.

3. Title: Pharmacological interventions for depression in people with traumatic brain injury: Systematic Review

Authors: JJ Vattakatuchery, N Lathif, J Joy, A Cavanna, H Rickards, 5 Boroughs NHS Foundation Trust, Wakefield House, Guardian Street, Warrington, WA51GG
United Kingdom

Objective: To undertake a systematic review and meta-analysis of pharmacological interventions for depression in people with traumatic brain injury.

Method: Searches were undertaken for randomised controlled trials of pharmacological interventions in people with depression and traumatic brain injury. Searches were carried out as per our protocol and studies that fulfilled our inclusion criteria were included in the meta-analysis.

Results: Four studies were identified that fulfilled our inclusion criteria. Sertraline, desipramine, methylphenidate and modafinil were investigated in these studies. There were 72 participants in total in the intervention arm and 57 participants in the placebo arm. Meta-analysis showed favourable results for sertraline, desipramine and methylphenidate on outcomes measures for depressive symptoms. Only results for methylphenidate were statistically significant. Sertraline, methylphenidate and modafinil showed favourable results on quality of life indicators but results were not statistically significant. Sertraline showed favourable results on outcome measures for anxiety symptoms but results were not statistically significant.

Conclusion: Some pharmacological interventions appear to improve depressive symptoms, anxiety symptoms and quality of life in people with depression and traumatic brain injury. However, the evidence is limited. There is a paucity of evidence for the effectiveness of other pharmacological interventions used in depression in this particular patient group.

4. Title: An audit of the diagnosis and costs associated with inpatients with functional neurological symptoms in a district general hospital

Authors: Adjei M, Coebergh JA, Email: m0901784@sgul.ac.uk

Objective: Functional symptoms are signs of illness which are not associated with any organic cause. Patients with functional symptoms cost the NHS three billion pounds in 2008-9.1 Within neurology alone, functional symptoms represent as much as nine percent of all inpatient episodes.2 Patients with functional neurological symptoms may be difficult to manage and often have multiple hospital admissions. This may contribute to the high costs associated with their care.1

The aims of this audit are twofold. Firstly to assess the hospital stay and investigations undertaken by patients with functional neurological symptoms between 2009 and 2012 at Ashford St Peters Healthcare Trust (ASPH). The total cost of these patients between 2010 and 2012 incurred in one hospital were calculated

Method: This audit was carried out using patient notes and the hospital information system at ASPH. Of the 150 patients admitted to ASPH between February and September 2012, nineteen patients with functional symptoms were identified.

Information regarding basic demographics as well as A&E admissions for neurological disease in the last three years (September 2009 to September 2012) was collected. Length of stay in hospital, both in total and specifically due to their neurological symptoms was calculated along with the number of scans of the nervous system each patient had undergone in the last three years. Cost to the Primary Care Trust of each patient between April 2010 and August 2012 was calculated by a financial accountant at ASPH using the internal income software of the trust**.**

Results: Patients with functional neurological symptoms experienced an average of five ward admissions, six A&E admissions and 24 days hospital stay between 2009 and 2012. On average, seventeen days of hospital stay were specifically due to neurological disease and patients had up to eleven scans within this period. The average cost of patients to ASPH was £13 288 in just two years and four months and the total cost of all nineteen patients was £262 975.

Conclusion: Patients with functional neurological symptoms are associated with considerable hospital admissions, length of stays as well as high costs.

Additional Information: 1. Bermingham S.L, Cohen A, Hague J. Ment Health Fam Med 2010; 7(2): 71-84, 2. Reuber M, Mitchell A.J, Howlett S.J et al. J Neurol Neurosurg Psychiatry 2005; 76: 307-314

5. Title: Is there a link between Non-Epileptic Attack Disorders (NEAD) and Personality Disorders (PD)? - A Systematic Review. **Authors:** Dr Obiajulu Chudi Okoye, Email: objmichael@hotmail.com
Objective: This systematic review determined whether there are specific types of Personality disorders (PD) cluster(s) and/or specific PD disorder(s) that is more likely to occur in people with Non-epileptic attack disorders (NEAD) without epileptic seizures (ES) comorbidity, compared to those with NEAD and comorbid ES, or compared to people with ES only? An ancillary question is: is any such association characteristically affected by comorbidity of the sister axis-1 mental disorders?
Method: A systematic search of 4 major databases and reference lists identified 15 studies comprising of a mix of prospective -, controlled-cross-sectional -, non-controlled cross-sectional -, retrospective(of which 2 were controlled studies)-, and 2 case- studies. The main eligibility criteria were that study participants had diagnoses of NEAD and PD made with video-EEG and DSM (III to IV-TR) - respectively, and the relationship between the 2 aforementioned disorders was primarily or secondarily investigated.
Results: The result of this systematic review suggests that people with NEAD-only are more likely to have Cluster-B personality disorders and cluster-C PD (as shown by 9 and 4 studies - respectively), compared to people with comorbidity of NEAD and ES (2 studies and 1 study showing predominance of cluster B and C- respectively). People with NEAD-only and NEAD with ES are much more likely to have cluster-B PD than people with ES. Borderline PD, followed by histrionic PD are the most frequent cluster-B, PDs in NEAD-only and NEAD plus ES patients. People with ES are much more likely to have cluster-C PD than people with NEAD-only or NEAD with ES. Axis-I disorders did not appear to have any pattern of association or influence in regards the link between NEAD and PD.
Conclusion: Though the results suggest some specific association between NEAD and PD which is, in the main, in keeping with findings of previous studies, the quality of the individual studies examined in this review was too poor for any firm conclusions to be drawn.

6. Title: Identifying the shared neurobiological underpinnings of negative urgency and neuroticism
Authors: Muhlert N, Lawrence AD, Cognitive Neuroscience, Cardiff University, United Kingdom
Objective: Negative urgency, or the tendency to act rashly when experiencing negative affect, is a transdiagnostic risk factor for vulnerability to a number of psychopathologies. When combined with high levels of neuroticism, the two factors predict high rates of externalising behaviours. In this study we examined whether there are overlapping neurobiological markers of both urgency and neuroticism in the healthy population. **Method:** One hundred and fifty-two participants underwent T1-weighted MRI (1 x 1 x 1mm) at 3T. Voxel-based morphometry using diffeomorphic anatomical registration through exponentiated lie algebra in SPM8 was used to examine grey matter volumes. Self-report measures of urgency were acquired using the UPPS impulsivity scale. General linear models were used to examine associations between urgency and grey matter volumes in brain regions previously linked to neuroticism (the dorsomedial prefrontal cortex, medial temporal lobe and precentral gyrus). Age, gender and intracranial volumes were included as covariates of no interest. **Results:** Individual variability in urgency was negatively associated with individual variability in grey matter volumes within the dorsomedial prefrontal cortex (P).
Conclusion: Grey matter volumes within the dorsomedial prefrontal cortex, a region strongly implicated in emotion regulation, are linked to individual variability in both negative urgency and neuroticism. Normal inter-individual variation in grey matter volumes within this region may impact upon the risk and resilience of developing psychiatric disorders, particularly those associated with externalising disorders.

7. Title: The prevalence of tic disorder in an electoral district of Mauritius
Authors: Vinu Chummun, Vanisha Seetaram, Hugh Rickards. Email: vinu@doctors.org.uk
Objective: The aim of this project was to ascertain the minimum prevalence of tic disorders and Tourette syndrome, in school children aged 9-11 in all the mainstream schools of an electoral district of Mauritius, an island in the Indian Ocean with a 1.2 million population. Most tic disorder studies have been conducted in countries in the northern hemisphere. So far, there are only 3 studies that have been done in the southern hemisphere countries and they were directed at Tourette syndrome explicitly.
Method: This study, first of its kind in the southern hemisphere, consisted of a 2 stage process, screening followed by a face to face clinical interview. Screening questionnaires were sent to all the children in standard 4 to 6 (9-11 years of age - a total of 2003) in the 8 schools of the constituency. The tic positive patients were interviewed and the diagnoses were ascertained in accordance to the DSM IV-TR criteria.
Results: Out of the 1287 children screened, 53 (35 males,18 females) were diagnosed with a tic disorder yielding a prevalence of 4.1%. 8 children (7 males and 1 female) fulfilled the criteria for Tourette Disorder (0.6%). Another 0.8% had chronic tic disorder (6 boys and 4 girls). The majority presented with transient tic disorder at 2.5% with a male to female ratio of 19 to 13 respectively. The distribution of non-specific tic disorder was 0.2% (3 males). **Conclusion:** Tic disorder estimates were congruent with studies performed in the northern realm of the globe. The study was inaugural in suggesting that Tourette disorder may be common in people of Indian descent but is less frequent in African descent.

8. Title: The role of alexithymia in the development of functional motor symptoms (conversion disorder)

Authors: Benedetta Demartini, Panayiota Petrochilos, Lucia Ricciardi, Gary Price, Mark J EdwardS, Eileen Joyce, Sobell Department, UCL Institute of Neurology, Queen Square, London WC1N 3BG, UK, United Kingdom

Objective: The mechanisms leading to the development of functional motor symptoms (FMS) are of pathophysiological and clinical relevance, yet are poorly understood. The aim of the present study was to evaluate whether impaired emotional processing at the cognitive level (alexithymia) is present in patients affected by FMS. We conducted a cross-sectional study in a population of patients with FMS and in two control groups [patients with organic movement disorders (OMD) and healthy volunteers].
Method: Fifty-five patients with FMS, 33 patients affected by OMD and 34 healthy volunteers were recruited. The assessment included: the 20-item Toronto Alexithymia Scale (TAS-20), the Montgomery-Asberg Depression Rating Scale (MADRS), the Reading the Mind in the Eyes' Test and the Structured Clinical Interview for Personality Disorders (SCID II).
Results: Alexithymia was present in 34.5% of patients with FMS, 9.1% with OMD and 5.9% of the healthy volunteers, which was significantly higher in the FMS group (Ï‡ square (2)=14.129
Conclusion: Alexithymia, a personality construct denoting the inability to identify emotions at a cognitive level, may explain why some patients misattribute autonomic symptoms of anxiety, e.g. tremor, paraesthesiae, paralysis, to that of a physical illness.

9. Title: Autoantibodies in Alzheimer disease;

Authors: 1- Ramin Nilforooshan: Brain Science research Unit, ACU, Holloway Hill, Chertsey, KT16 0AE, UK. ramin.nilforooshan@sabp.nhs.uk
2- Angela Vincent Neurosciences Group, Department of Clinical Neurology, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DS, UK angela.vincent@sabp.nhs.uk
3- Jessica Eccles, Brighton and Sussex Medical School, BN1 9RR, UK. J.eccles@bsms.ac.uk
4- Rosie Pettingill Neurosciences Group, Department of Clinical Neurology, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DS, UK R rosie.pettingill@imm.ox.ac.uk
5-Philippa Pettingill Neurosciences Group, Department of Clinical Neurology, University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DS, UK philippa.pettingill@imm.ox.ac.uk
6- Lamia Ali Brain Science research Unit, ACU, Holloway Hill, Chertsey, KT16 0AE, UK lamia.ali@sabp.nhs.uk
7- Naji Tabet: Cognitive Treatment and Research Unit, Sussex Partnership NHS Foundation Trust, Brighton and Sussex Medical School, BN1 9RR, UK N.T.Tabet@brighton.ac.uk

Aims: There is increasing evidence to support the relevance of immune system in the pathogenesis of Alzheimer Disease (AD) and also there is growing evidence for importance of specific antibodies in some neurological disorders. Antibodies against Voltage Gated Potassium Channels (VGKC) and Glutamic Decarboxylase (GAD) are of interest for AD. The presence of these antibodies is thought to be related to cognitive impairment and memory problems. In our study we attempted to find a relationship between these antibodies in individuals with AD compared with healthy controls.
Method: Twenty two patients with diagnosis of mild to moderate AD aged 65 and above were recruited from a memory clinic. Controls (22) were partners/carers of recruited patients who had no complaints of memory impairment and had a Mini Mental State Examination (MMSE) of 29 and above. Antibodies were measured by radioimmuo-precipitaion assay
Results: AD and control group did not differ significantly in age (p=0.593) and gender (0.674) but as expected the median MMSE scores (p≤0.001) did. We could not find any statistically significant difference between level of VGKC-ab (p=0.490) or GAD (p=0.330) between patients and controls. NMDA antibodies were negative in both groups. One of the patients (84 year old, female, MMSE 20/30 none smoker with no medical history and only 6 months history of memory decline) had strong positive Hippocampal Neurons (G) antibodies. This was observed on the strong immunocytochemical staining of the hippocampal neurons.
Conclusion: It is still unclear for how long these antibodies are being identified in acute onset of many neurological diseases including amnesia or psychosis. We know that pathophysiology of AD starts many years before the clinical presentations. It may be the case that positive antibodies can be found in a very early stage of mild cognitive impairment.

10. Title: Do prenatal and perinatal complications influence tic severity in patients with Gilles de la Tourette syndrome?

Authors: Taylor KA, Stern JS, Williams D, Simmons HS, Robertson MR , Department of Neurology, Atkinson Morely's Wing, St. George's Hospital, Blackshaw Road, London. SW17 0QQ, United Kingdom

Objective: Evidence for the role of complex genetics in the clinical expression of Gilles de la Tourette (GTS) is widespread. Streptococcal autoimmunity as another aetiology is under intense investigation but there is relatively little evidence for other environmental factors. It has long been suggested that perinatal problems increase vulnerability to Tourette syndrome. The aim of this project was to investigate whether prenatal or perinatal complications are associated with an increased tic severity in patients who develop GTS.

Method: 193 patients with GTS attending St George's Hospital between 2004-2011 were retrospectively reviewed for exposure to prenatal and perinatal complications (Mean age 20.3 Â± 13.55; age range 3-76; 145 males: 48 females)

The Yale Global Tic Severity Score (YGTSS) was used to assess current tic severity, giving each patient a score out of 25 for phonic and motor tics, and a score out of 50 for total tic severity. An additional score was given for overall impairment caused by GTS ranging from 0 (no impairment) to 50 (severe impairment). Records were also reviewed for the presence of current co-morbidity associated with GTS (ADHD/OCB/OCD); and assessed for any family history of GTS, tics, ADHD, OCD or OCB. The mean tic severity and impairment scores for the group who reported prenatal or perinatal complications were assessed in comparison to those of the group reporting no complications.

Results: Perinatal complications were reported in 92 out of 193 patients (48%). The mean total tic score compared to patients with a history of complications was 25.94 v. 24.88 and the mean impairment scores were 29.01 v. 25.24 respectively.

Conclusion: Previous multivariate analyses have correlated perinatal factors with tic severity e.g. maternal smoking. In this cohort, prenatal and perinatal complications were not associated with increased motor, phonic or total tic severity or increase in impairment and there was no increased level of comorbidity or family history. Limitations include recall bias for perinatal events, a univariate approach and the measure of severity in all studies which applies to one time-point in a fluctuating condition.

11. Title: Psychosocial characteristics of Tourette syndrome in older adults attending a specialist clinic

Authors: Man CHA, Stern JS, Gharatya A, Williams D, Simmons HS, Robertson MM, Department of Neurology**,** Atkinson Morley's Wing, St George's Hospital, Blackshaw Road, London SW17 0QQ

Objective: As Tourette syndrome (TS) is a childhood-onset disease with symptoms which are expected to decrease beyond adolescence, older adults who attend specialist clinics are less common. TS is associated with impaired social functioning and a lowered quality of life. The aim of this study is to investigate the psychosocial health of these patients.

Method: Older adults, defined as aged 40 and over, who have attended the specialist clinic at St. George's Hospital in London and diagnosed with TS were included in this study. These patients were divided into three age sub-groups: 40-49 years old, 50-59 years old, and 60+ years old. Comparisons were made with younger adult patients (aged 25-39).

Using patient's clinical letters, the mean Yale Global Tic Severity Score (YGTSS) and percentage of patients categorised into each clinician-rated severity impression were calculated. Rates of co-existing psychopathology, coprophenomena, forensic history, alcohol abuse, employment and marital status, and highest level of education achieved were recorded.

Results: 524 patients in total have attended the clinic, of which 46 were younger adults. 48 older adults were identified in total: 35 aged 40-49, 8 aged 50-59 and 5 aged 60+. Older adults present with greater tic severity compared to younger adults (mean YGTSS of 28.05 vs. 24.95 and a "severe" clinician-rated severity in 22.0% vs. 12.2%). Greater rates of social impairment were noted in 50-59 year olds than younger adults with respects to: unemployment (50% vs. 23.4%) and lack of a formal educational qualification (37.5% vs. 9.1%). A high proportion of patients 40-49 and 50-59 are either single or divorced- 50% and 25% respectively. Rates of an eventful forensic history (26.5% vs. 14.4%) and past alcohol abuse (32.4% vs. 9.1%) were greater in 40-49 year olds than younger adults. Rates of anxiety (10.4% vs. 4.5%) and depression (29.2% vs. 16.4%) were greater in older adults than younger adults. Rates of coprophenoma are 31.4% in ages 40-49, 37.5% in ages 50-59, and 20.0% in ages 60+.

Conclusion: In this clinical cohort, older adults attending a specialist TS clinic have more severe tics and are often in poor psychological health, with a history of past and current social impairment. There is an element of referral bias but the findings indicate the significant psychosocial burden in cases with an atypical evolution with ageing.

12. Title: Suicidality in patients with Tourette's syndrome

Authors: Gharatya AK, Stern JS, Man CHA, Williams D, Simmons HS, Robertson MM**,** Department of Neurology, Atkinson Morley's Wing, St George's Hospital, Blackshaw Road, London, SW17 0QQ

Objective: To investigate the clinical characteristics of patients with suicide attempts or suicidal ideation in a cohort of Tourette (TS) patients

Method: We reviewed the clinical notes of 524 patients diagnosed with TS attending the St. George's Hospital Tic Disorder Clinic. A control group used an existing database of 141 to 469 patients, according to availability of relevant items.

The following features were examined: history of suicide attempts (SA) or suicidal ideation (SI), disease severity, prevalence of co-morbidity and psychopathology, family history of suicide/ suicide attempt and depression, history of substance abuse, employment status, forensic history and self-injurious behaviour (SIB). **Results:** Of the total cohort of 524 patients suicide attempts were recorded in 25, mean age 26.9, M:F 1.8:1 and suicidal ideation only in 30, mean age 21.4, M:F 2.3:1. SA patients recorded the highest disease severity compared to SI and control group with an average Yale Tic Severity Score of 30.4 vs. 28.3 vs. 24.6. Clinician-rated severity was "severe" in 40.9%* vs. 21.4% vs. 8.4%. SA patients also had the highest prevalence of co-morbidities and psychopathology including depression (75%* vs. 81.5%* vs. 12.3%), anxiety (80%* vs. 70.8%* vs. 12.9%), SIB (20% vs. 26.7% vs. 16.4%), ADHD (81.8%* vs. 69.00% vs. 53.92%,), OCD (73.9%* vs. 51.7%* vs. 28.2%), retrospective diagnosis of oppositional defiant disorder (40%* vs. 33.3%* vs. 16.5%), retrospective diagnosis of conduct disorder (24%* vs. 6.7% vs. 4.6%.). Family history of suicide/suicide attempts were more common in SA patients (36.8%* vs. 26.7%* vs. 4.70%,) while family history of depression was greatest within the SI population (60.9%* vs. 66.7%* vs. 28.2%). (* $p<0.05$). Unemployment rates were also highest among SA patients compared to employment-aged members of the control group (33.3% vs. 16% vs. 15.6%). Additionally SA and SI patients had high levels of drug abuse (50% and 19.1%), alcohol abuse (43.5% and 13.6%) or a forensic history (56.5% and 13.8%) but have not been compared with an age-appropriate or matched group.

Conclusion: This is the first study to examine suicidality in a clinical TS cohort. These 55 patients had severe tics, high rates of comorbidity, substance abuse, forensic history and family history of suicide attempt. Limitations include referral bias and an age-unmatched control population, hence we have not yet given a prevalence figure for suicide attempts as it is important for this to be valid.

13. Title: Role of catecholaminergic and cholinergic drugs in management of cognitive deficits in adults with Traumatic Brain Injury: a systematic review

Authors: Inderbir Singh Sidhu, ST6 Psychiatry of Intellectual Disabilities, South Essex Partnership University NHS Foundation Trust, Clinical Base 3 Heath Close, Billericay, Essex , CM12 9NW, United Kingdom

Objective: Pharmacological therapies aimed at modulating catecholaminergic and cholinergic neuronal dysfunction caused by brain trauma have been tried to improve cognitive function post TBI. This systematic review aims to evaluate effectiveness of catecholamingeric and cholinergic drugs used to improve cognitive deficits in adults with TBI.

Method: Medline, EMBASE and PsycINFO were searched for randomised control trials (RCTs), open label, case control studies or case series with 10 or more subjects, assessing effectiveness of catecholaminergic or cholinergic drugs or both, in adults who had TBI at least 2 weeks ago (post-acute phase). All eligible studies required at least one outcome that assessed attention or memory using standardised test.

Any study involving children, non-TBI, adults with minimal conscious states or neurodegenerative diseases, less than 10 subjects or single dose treatment was excluded.

Results: The studies were pooled using narrative synthesis with results grouped according to the type of medication used. 19 studies (n=823) were selected: 12 RCTs (n=434), 1 single blind randomised study (n=115), 1 case control study (n=36), 3 open label prospective studies (n=175), 1 case series (n=10) and 1 retrospective study (n=53). Catecholaminergics (9 studies): Methylphenidate showed significant improvement in speed of information processing and attention in survivors of TBI in three RCTs as compared to placebo, while no significant difference was found between the two groups in other RCT. One RCT showed possible role of amantadine in cognitive recovery in early stages of TBI, while the other two studies did not support these findings. Bromocriptine in low dose was shown to improve executive function in one RCT, but its effects on attention and memory improvement were less promising. Cholinergics (10 studies): All but one (case control study) of the 6 studies (1 RCT, 3 open label and 1 case series) reported positive effect on sustained attention and short term memory with donepezil. The level 1 evidence based on one large multicentric RCT does not support use of rivastigmine for cognitive augmentation post-TBI. Results for the use of choline and physotigmine on cognitive recovery after TBI from two RCTs are inconsistent

Conclusion: Heterogeneity of TBI population, methodological difficulties, variation in the dose, duration and timing of the medication and use of various psychometric tests to measure cognitive changes confound the results of these studies. Pharmacotherapy of cognitive symptoms following TBI remains a common practice although robust evidence to support this practice is limited. Further research in this area is warranted.

14. Title: Prescribing practices in adults with Tourette syndrome

Authors: Graham Blackman, Andrea E. Cavanna, Department of Neuropsychiatry, BSMHFT and University of Birmingham, The Barberry National Centre for Mental Health, 25 Vincent Drive Birmingham B152FG,United Kingdom

Objective: Tourette syndrome (TS) is a developmental condition characterised by tics and associated with other psychiatric diagnoses, in particular obsessive-compulsive disorder (OCD) and attention-deficit hyperactivity disorder (ADHD). Despite the availability of behavioural and surgical options, pharmacotherapy remains the most common management option for patients with TS. The possible effects of psychiatric co-morbidities upon prescribing practices in adults with TS have not been examined in the literature to date.
Method: We conducted a retrospective chart review of all consecutive adult patients with TS who agreed to take part in clinical research conducted at the specialist TS clinic between 2009 and 2013. Types of pharmacological agents, as well as DSM-validated diagnoses and clinician-rated measures of tic severity (Yale Global Tic Severity Scale), were examined across different patient groups defined by the presence of psychiatric co-morbidities.
Results: Of 162 adults with TS, 88 (54.4%) had "pure" TS, 34 (21.0%) had a co-morbid diagnosis of OCD, 26 (16.0%) had co-morbid ADHD and 14 (8.6%) had both co-morbidities. Overall, 106 patients (65.4%) were on pharmacological treatment. The presence of co-morbid ADHD or OCD was significantly associated with use of pharmacotherapy (p=.02), more strongly than total tic severity (p=.04). Although there was no significant association between co-morbidity type and pharmacological class used, serotonergic agents were most commonly prescribed in patients with TS+OCD (41.2%) and alpha-2 agonists in patients with TS+ADHD (23.1%).
Conclusion: The majority of adult patients attending specialist TS clinics require pharmacotherapy. The presence of co-morbid OCD and ADHD, followed by tic severity, are the main factors associated with use of pharmacotherapy and can play an important role in the choice of medication class.

15. Title: Multidisciplinary inpatient programme for functional neurological symptoms: a prospective study assessing efficacy and predictors of good outcome.

Authors: Benedetta Demartini, Panayiota Petrochilos, Mark J Edwards, Eileen Joyce, Sobell Department, UCL Institute of Neurology, Queen Square, London WC1N 3BG, UK

Objective: Although functional neurological symptoms (FNS) are often very disabling, little is known about their long-term prognosis and about the best approaches to treatment. Inpatient treatment programmes combining different approaches have been described but the lack of controlled studies and the expensiveness of the intervention have limited their use. Here we aimed to assess the short- and long-term efficacy of an inpatient multidisciplinary programme for patients with FNS. We also sought to determine baseline predictors of good outcome. Finally we aimed to assess the responsiveness of different psychological and physical scales administered at admission, discharge and follow-up.
Method: Sixty-six consecutive patients affected by FNS treated at a specialised multidisciplinary inpatient programme were included. The assessment at admission, discharge and at 1-year follow-up included: the Health of the Nation Outcome Scale (HoNOS), the Hospital Anxiety and Depression Scale (HADS), the Patient Heath Questionnaire-15 (PHQ-15), the Revised Illness Perception Questionnaire (IPQ-R), the Common Neurological Symptom Questionnaire, the Fear Questionnaire (FQ) and the Canadian Occupational Performance Measure (COPM). At discharge and at 1-year follow-up patients were also asked to complete a five-point self-rated scale of Clinical Global Improvement (CGI).
Results: Respectively 68.1% and 66.6% patients rated their general health such as "better" or "much better" on the CGI at discharge and at 1-year follow-up. Good outcomes were predicted by the IPQ-R timeline dimension and by low score on the HoNOS and on the HADS-depression. The HoNOS, the IPQ-R and the COPM performance were responsive to change indexed by self-reported CGI.
Conclusion: Our data suggest that a specialized multidisciplinary inpatient programme for FNS can provide long-lasting benefits (measured by CGI) in the majority of patients. Although we identified some interesting findings, further studies are needed to better clarify the role of baseline predictors of good outcomes and the responsiveness of psychological and physical scales.

16. Title: Habenula responses during appetitive and aversive conditioning in Major Depressive Disorder. Authors: Rebecca P. Lawson, Camilla L. Nord, Ben Seymour, David L. Thomas, Raymond J. Dolan, Peter Dayan, Nikolaus Weiskopf & Jonathan P. Roiser, Wellcome Trust Centre for Neuroimaging, 12 Queen Square, London, WC1N 3BG, United Kingdom

Objective: The lateral habenula (LHb) has been shown to respond to cues that predict aversive stimuli in non-human primates (Matsumoto & Hikosaka, 2009, Nat Neurosci, 12(1), 77-84) and has been implicated in reinforcement processing and the pathophysiology of major depression (MDD) (Roiser et al, Biol Psychiatry, 66(5),441-450), possibly via reciprocal connections with monoaminergic nuclei. We report the first high-resolution fMRI investigation of haemodynamic responses during appetitive and aversive conditioning in the LHb in unipolar MDD. Additionally, we report the first assessment of tonic habenula function using quantitative Arterial Spin Labelling (ASL) in MDD.

Method: Unmedicated MDD patients (n=26) and matched controls (n=26) performed a Pavlovian conditioning task where they were exposed to conditioned stimuli (CSs) that preceded reinforcing outcomes (win £1; lose £1; and painful electric shock) in a probabilistic manner. Neural responses were monitored using high-resolution T2* echo-planar imaging (1.5mm isotropic), and T1-weighted (0.77mm isotropic) images were obtained to accurately identify the habenula (Lawson, et al., 2013, NeuroImage, 64(0), 722/727). Breathing and heart-rate data were collected to correct for pulse-related artefacts. Using a temporal difference learning algorithm, trial-by-trial values for win, loss and shock predicting CSs were derived for each subject and used as parametric modulators in the fMRI analysis. Additionally, to investigate tonic habenula function, high-resolution ASL images were acquired to quantitatively assess baseline blood flow in the habenula.

Results: We replicated our previous result that habenula responses were positive to shock-predicting CSs in healthy volunteers (Lawson et al, submitted). We found no significant group differences in habenula responses to win and loss CSs (Ps>0.7), but surprisingly habenula responses to the value of shock CSs were negative in MDD subjects (P<0.05) and significantly different to controls (p<0005). However, there were no significant group differences in baseline blood flow (P>0.6) or habenula volume (P>0.5), ruling out tonic habenula function or grey matter volume differences as an explanation for our functional results.

Conclusion: These surprising results, which cannot be accounted for by group differences in tonic habenula activity or habenula volume, reflect a difference in how depressed patients process aversive stimuli and have important implications for understanding the constructs of anhedonia and learned helplessness.

17. Title: Clinical features of depression in Huntington's disease - A cross sectional study comparing the clinical features of depression in patients with Huntington's disease and in patients without Huntington's disease

Authors: Dr Joby Scaria, Dr David Craufurd, ST4 General Adult Psychiatry, NorthWestern Deanery

Objective: Huntington's Disease (HD) is an inherited progressive neurodegenerative disorder, characterised by a triad of motor, cognitive and psychiatric symptoms. Depression is common in patients with HD, but does not correlate well with motor or cognitive measures of disease progression, suggesting that different neuropathological process may be involved. However clinical experience suggests that depressive symptoms in HD are variable and tend to fluctuate from day to day and in response to environmental factors and mental stimulation, suggesting that HD related cognitive changes and impaired motivation might contribute to the aetiology of depressive symptoms. The aim of the present exploratory pilot study was therefore to compare the clinical features of depression in HD with depression in general population.

Method: A cross sectional comparison study was completed with 15 depressed Huntington's Disease patients and 15 depressed patients from general population. Recruitment of the participants was based on specific inclusion and exclusion criteria and selected patients with moderate depression. The comparison group was matched for age, sex and Visual Analogue Mood Scores. All subjects in both groups completed the Hospital Anxiety and Depression Scale, Beck Depression Inventory, Test for Anhedonia questionnaire and Montgomery Asberg Depression Rating Scale; the latter was modified for this study with 3 extra questions about environmental susceptibility of low mood.

Results: The results of the study indicate that there was no significant difference between groups in the clinical features of depression, based on the total score of all the questionnaires used. Depressed patients without HD had significantly higher scorers on the lack of enjoyment (anhedonia) item from the Hospital Anxiety and Depression Scale; however, this was the only significant difference between the groups, and this finding did not survive Bonferroni Correction. There was a trend for concentration difficulties and improvement in mood based on changes in environmental factors to be more pronounced in HD patients, but this difference was not statistically significant based on p values and confidence intervals.

Conclusion: We conclude that there is no significant difference in the clinical features of depression in Huntington's disease compared to depressed patients from general population. The overall effect size for the whole study was low to medium and based on the calculations more significant results could be obtained with more than 100 participants in each group in future studies. We plan a further study looking at the pervasiveness of low mood over time in the two groups.

18. Title: Emotional Processing in Adults with Functional Neurological Symptoms
Authors: Hannah R Reynolds, Lisa A Jones, Hugh E Rickards, Email: HRR929@bham.ac.uk
Objective: Functional neurological symptoms may develop as a result of altered patterns of emotional processing. We aimed to determine whether emotional processing differs between adults with functional neurological symptoms (seizures, n=30 and sensorimotor symptoms, n=18) and healthy controls (n=44).
Method: Emotional processing was measured via two self-report questionnaires: the Beliefs about Emotions Scale (BES) and the Emotional Processing Scale (EPS-25). The severity of current anxiety and depression symptoms was measured by the Hospital Anxiety and Depression Scale (HADS).
Results: Patients with functional seizures, but not those with functional sensorimotor symptoms, reported significantly more difficulty with emotional processing than healthy controls, particularly with emotional suppression (p=0.045) and avoidance (p=0.003) and impoverished emotional experience (p=0.004). These differences did not remain significant after controlling for anxiety and depression.
Conclusion: Although some aspects of emotional processing differ between patients with functional seizures and healthy controls, these differences may be attributable to anxiety and depression. Further longitudinal studies are required to fully explore these associations.

19. Title: EEG Biofeedback Therapy for ADHD: A Systematic Review
Authors: Dr Ashley Liew, Email: ashleyliew@gmail.com
Objective: To examine systematically the evidence for the application of electroencephalographic (EEG) biofeedback, otherwise called neurofeedback, in the treatment of children and young people with Attention Deficit Hyperactivity Disorder (ADHD).
Method: A literature search of 8 electronic databases was conducted to identify articles published between February 1985 and February 2013. Furthermore, researchers in the field and professional organisations were contacted for access to unpublished data. The review was restricted to intervention studies, although they did not have to be randomised controlled trials. For each study, the quality of the methods and the strength of the evidence were assessed using the evidence based guidelines devised by Sackett.
Results: A total of 112 studies were identified, of which 26 met the inclusion criteria. As some of these included follow-up data, a total of 20 separate studies were identified. 8 studies were randomised controlled trials. All the studies demonstrated significant improvements for neurofeedback across a range of cognitive and behavioural measures. However, several important weaknesses were identified in the methodology of the majority of the studies, namely, absence of blinding, lack of randomisation and small sample sizes. More recent studies have overcome some of these weaknesses through novel designs, but important limitations remain.
Conclusion: There is growing evidence for neurofeedback as a non-pharmacological alternative in the treatment of ADHD, but the existing literature displays a range of methodological weaknesses. Further and more convincing research is required.

20. Title: Severe Refractory Tourette Syndrome
Authors: Colquhoun M, Stern JS , Collicott N, Williams D , Grabecki K, Simmons H , Robertson MR, Department of neurology, Atkinson Morley's Wing, St. George's Hospital, Blackshaw Road, London.
Objective: The worldwide prevalence of Tourette's syndrome (TS) is well established at around 1% of school children in community studies, but little is known about the frequency of severe cases resistant to medical treatment. This subset of patients has taken on new significance due to the emergence of deep brain stimulation (DBS). We screened a specialist clinical cohort to identify this group.
Method: Data was acquired from the case records of 329 patients of all ages attending the St. George's Tic Disorder clinic. Disease severity and resistance to pharmacological treatment were defined as per published European Society for the Study of Tourette Syndrome guidelines, i.e. Yale Global Tic Severity Score (YGTSS) > 35 and resistance to three drug treatments including a typical and an atypical neuroleptic. Clinical records were reviewed for adherence 13 points in the guideline.
Results: 14 out of 329 patients (4.3%) were deemed both severe and refractory to pharmacological treatment and had a mean YGTSS of 38.7. The small number in this group precluded statistical analysis of patient characteristics compared to the control group. No patients fulfilled all ESSTS guideline criteria although two patients in the cohort had already had DBS and one other had been assessed for DBS but improved substantially with a further medication choice (Topiramate).
Conclusion: Patients in this group are the public face of TS. Their population prevalence is unknown but they are likely to over-represented in clinical cohorts although other individuals may have withdrawn from society and medical care. Even in this tertiary specialised clinic cohort fewer than 5% would be likely to be suitable for DBS given it remains a procedure for the severest cases. Current guidelines for patient selection have been drawn up in the era of a semi-experimental technique and are likely to evolve. Published criteria that that have been waived on compassionate grounds in this and other cohorts are minimum age recommendations (>25 years) and full access to behavioural therapies.

Members' Posters

21. Title: Facial emotion expressiveness and facial emotion recognition in Parkinson's disease: how much does alexithymia count?

Authors: Lucia Ricciardi, Matteo Bologna, Diego Ricciardi, Bruno Morabito, Francesca Morgante, Daniele Volpe, Davide Martino, Alessandro Tessitore, Massimiliano Pomponi, Anna Rita Bentivoglio, Roberto Bernabei, Alfonso Fasano. Department of Clinical and Experimental Medicine, University of Messina, via Consolare Valeria, 98125 Messina, Italy.

Objective: Background and aims: It is recognized that emotional deficits are part of the non-motor features of Parkinson's disease but scant attention has been paid to specific aspects such as emotional facial expression, subjective emotional experience (alexithymia) and recognition of facial emotion expressions. This study aimed to investigate the relationship between alexithymia, emotion facial recognition, and emotion facial expression in PD patients.

Method: Forty-one PD patients and seventeen healthy controls, matched for demographical characteristics, were enrolled in the study.

Alexithymia was assessed by means of Toronto Alexithymia Scale (TAS-20), emotion facial recognition was tested by means of the Ekman 60 Faces Test, emotion facial expression was investigated with a video protocol encompassing of a static expression recording (subject watching the camera in silence for 30 seconds), a dynamic expression recording (subject recorded while talking for 30 seconds) and emotion expression (subject was asked to express with his/her face the six main human expressions: happiness, sadness, anger, fear, surprise, disgust).

Six blind raters evaluated the patients' video recordings.

Results: No difference in alexithymia was detected between PD patients and HC. PD patients performed significantly worse than HC in recognizing Surprize ($p=0.03$) and showed significant poorer global facial expression than HC (in static, dynamic and emotion facial expression).

There was a significant negative correlation between the factor F3 of TAS (externally orientated thoughts) and the patient's capability to express disgust (-.447, $p=0.007$). Elkman total score positively correlates with the patient's capability to express disgust with his face (.325, $p=0.006$,).

Conclusion: These results suggest that PD patients have difficulties with emotional recognition and expression in a contest of a unimpaired subjective emotional experience. These deficits need to be targeted in clinical practise for rehabilitation purposes.

22. Title: Neural Bases of Musical Hallucinations

Authors: Sukhbinder Kumar, William Sedley, Gareth R Barnes, Sundeep Teki, Karl J Friston, Timothy D Griffiths, Auditory Group,
Institute of Neuroscience, Newcastle University Medical School, Newcastle University, Newcastle upon Tyne,NE2 4HH,United Kingdom

Objective: The brain bases of musical hallucinations (MH) is not understood. A major problem in this effort is the lack of a suitable method to experimentally manipulate MH during the course of experiment. In this work we present a method to systematically manipulate MH using an external masker stimulus. Furthermore, we make use of the manipulation of MH to compare brain activity as a function of intensity of hallucination. We then combine our results with the computational theories of brain function to propose a model of how the brain generates and maintain MH

Method: We used a residual inhibition (RI) paradigm to manipulate MH. In the RI, an external masking stimulus (music in the current work) is presented which suppresses hallucinations. The intensity of hallucinations remain low after the offset of masking stimulus before the hallucinations come back to normal level. We make use of the RI to manipulate MH while brain activity being acquired using Magnetoencephalography (MEG).

Results: Our results show that (i) MH can residually suppress the MH for up to a minute(ii) Source-space analysis capable of single-subject inference defined left-lateralised power increases, associated with stronger hallucinations, in the gamma band in left anterior superior temporal gyrus, and in the beta band in motor cortex and posteromedial cortex.

Conclusion: The data indicate that these areas form a crucial network in the generation of MH, and are consistent with a model in which MH are generated by persistent reciprocal communication in a predictive coding hierarchy.

23. Title: Misophonia: a Disorder of Emotion Processing of Sounds

Authors: Sukhbinder Kumar, Olana Hancock, Thomas Cope, William Sedley, Joel Winston, Timothy D Griffiths, Auditory Group Institute of Neuroscience, Newcastle University Medical School, Newcastle upon Tyne, NE2 4HH, United Kingdom.

Objective: Sounds of chewing, breathing, keyboard typing are considered by most people as 'normal' sounds and are ignored as background sounds in everyday listening. However, for a group of people these sounds are not only distracting but also evoke a strong feeling of anger accompanied by an urge to escape from the situation producing these sounds. This condition, marked by sensitivity to a selective group of sounds, was given the name "misophonia" (hatred of sounds) almost a decade ago. Since the sounds that act as "triggers" in this condition are quite common at home, work place and in social gatherings, misophonia has devastating effects on social, family and personal life of the sufferer.

Presently misophonia is not featured in any official medical /psychiatric classifications. People with misophonia suffer in silence because they do not share their problem with others for the fear of being called "crazy". The objective of the current work was to check the consistency of profile of symptoms and trigger sounds in a population of subjects with misophonia.

Method: A group of subjects were assessed by a group of neurologists in a neurology clinic.

To further assess the symptoms/triggers in a bigger population, a questionnaire was designed asking questions about symptoms/triggers etc which was then posted on a website dedicated to disseminate information about misophonia. People were requested to fill-in the questionnaire and send back by email.

Results: The assessment in the neurology clinic confirmed a striking similar profile of symptoms and triggers in the individuals. A group of 195 subjects filled-in the questionnaire posted on the website. Analysis of 157 questionnaire showed:

Mean Age of Onset =12.08 years [5-50]

146/157 (93.0 %) describe eating sounds as triggers

135/157 (86.0 %) describe anger as the dominant emotion (other emotions include panic/anxiety)

132/157 (84.0 %) describe leaving the situation which produce trigger sounds.

Conclusion: Our data indicate that misophonia is a disorder of emotion processing of sounds which need to be investigated further.

24. Title: Interoceptive sensitivity and sense of body ownership in patients with functional neurological symptoms

Authors: Lucia Ricciardi, Benedetta Demartini, Laura Crucianelli, Mark J. Edwards, Aikaterini Fotopoulou. Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology, University College London,

Objective: Background and aims.

Patients with functional (psychogenic) neurological symptoms are commonly seen in neurological practice. Though emotional/psychological causes are often proposed to underlie their symptoms, patients characteristically deny such problems, even when objective evidence for (for example) anxiety or panic is present. Interception is the perception of sensations from inside the body and includes the perception of physical sensations related to internal organ function. Heartbeat perception is considered a standard method for the assessment of interceptive sensitivity and it could be considered as a measure of self-awareness of internal stimuli which may have relevance for determining emotional state. The aim of our study was to evaluate interceptive awareness in patients with functional (psychogenic) movement symptoms (FMS). In addition we assessed the sense of body ownership using the rubber hand illusion (RHI).

Method: We included in the study 17 patients with FMS according to Fahn and Williams criteria. Eighteen healthy controls (HC), matched for age and gender served as a control group. Patients and HC were asked to complete the Toronto Alexithymia Scale (TAS-20) and the self-consciousness scale (self-objectification questionnaire), also we administered the Montgomery depression scale. Heart beat perception task: heart rate was recorded by means of a commercial heart rate monitor and subjects were asked to count their heart beats (only by concentration on their body and not by taking their pulse) during a signalled time interval. The reported number of beats was then compared to the actual number of beats. All subjects were tested before and after a stress-induction task. RHI: illusionary experience was measured by self-report and by proprioceptive alteration.

Results: FMS patients showed a poorer interceptive sensitivity than HC in the pre stress condition ($p=0.048$), but no difference was seen between groups in the post stress condition. No significant differences were revealed between patients with FMS and HC in the RHI on both perceptual (i.e. proprioceptive drift) and subjective (i.e. self-report questionnaire) measures.

Conclusion: Patients with FMS have poor interoceptive sensitivity. This could relate to impairments of assessment of emotional state in these patients.