THE BRITISH NEUROPSYCHIATRY ASSOCIATION 26th AGM

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The Institute of Child Health, Guilford Street, London

7/8 FEBRUARY 2013

Thursday 7 February

members' platform

NEUROPSYCHIATRY RESEARCH UPDATE

Friday 8 February

7/8 February
Poster Session

Welcome to the 26th annual meeting of the British Neuropsychiatry Association

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British NeuroPsychiatry Association

British Neuropsychiatry 26th AGM PROGRAMME

BNPA

BNPA 26th AGM - 7th and 8th February 2013

Institute of Child Health, Guilford Street, London

DAY 1 Thursday 7th February
STRESS AND THE BRAIN
Chair: Eileen Joyce

0830	***Registration and refreshments***
0930	**The impact of stress on the social brain – Psychopathological implications and neurobiological mechanisms** Carmen Sandi (Ch)
1005	**Equipped to Survive: Comprehensive Response to Threat Enables Optimal Behaviour** Guillén Fernández (Nl)
1040	***Refreshments***
1110	**Posttraumatic stress disorder and the brain** Chris Brewin (UK)
1145	**Inflammation and mental illness** Neil Harrison (UK)
1225	**Childhood stress and risk for later mental disorder** Jeremy Hall (UK)
1300	***Lunch and poster viewing***
1400	**JNNP Guest Speaker Chair: Alan Carson** **Stress and war: the limits of neuropsychiatry** Neil Greenberg (UK)
1500	***Refreshments***
1530	**MEMBERS' PLATFORM PRESENTATIONS** **Temporal Lobe Epilepsy & Affective Disorders: The Role of the Subgenual Prefrontal Cortex** Cleary RA, Stretton J, Winston G, Symms M, Sidhu M, Thompson PJ, Koepp M, Duncan JS, Foong J. **Effects of early childhood Posterior Fossa Tumours on IQ** Carroll C, Clare I, Watson P, Hawkins MM, Spoudeas H, Walker D, Holland A, Ring HA **The neural correlates of Freudian "repression" in Conversion Disorder** Aybek S, Nicholson TR, Zelaya F, O'Daly OG, Craig TJ, David AS, Kanaan RA *Prize Giving*
1630	**BNPA - Neuropsychiatry Research Update** **Deep Brain Stimulation for mental illness** Eileen Joyce (UK) **Autoimmunity and Neuropsychiatry** Tim Nicholson (UK)
1730	***Close Day 1***
1900	***BNPA Evening Reception - Cartoon Museum***

BNPA 26th AGM - 7th and 8th February 2013

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DAY 2 Friday 8th February
EPILEPSY
Chair: Markus Reuber

0830	***Registration and refreshments***
0930	**Cellular mechanisms of epilepsy** John Jefferys (UK)
1000	**Brain networks in human epilepsy** Mark Richardson (UK)
1030	***Refreshments***
1100	**The impact of epilepsy on cognitive function** Christoph Helmstaedter (D)
1130	**BNPA Plenary** **Epilepsy, Depression and Anxiety Disorders: A complex relation with significant therapeutic implications for the three conditions** Andres Kanner (US)
1220	***BNPA AGM 1220-1300 (Members only) Lunch and poster viewing***

CONSCIOUSNESS
Chair: Peter Halligan

1400	**Decoding consciousness** Geraint Rees (UK)
1500	**Psychedelic drugs, magical thinking and psychosis** Robin Carhart-Harris (UK)
1600	***Close***

Speakers Short Biographies and Abstracts Day 1, Thursday 7 February

STRESS AND THE BRAIN
Chair: Eileen Joyce

The impact of stress on the social brain – Psychopathological implications and neurobiological mechanisms

Carmen Sandi is Professor and Director of the Brain Mind Institute, at the Swiss Federal Institute of Technology in Lausanne (EPFL), Switzerland, where she leads the Laboratory of Behavioral Genetics. She obtained a PhD in Neuroscience at the Cajal Institute, CSIC Madrid, and had postdoctoral appointments at the University of Bordeaux II-INSERM and the Open University, UK. After a sabbatical stay at the University of Bern, she joined the EPFL in 2003. Her main interest is to understand how stress affects brain function and behavior. Her work has been pioneering in identifying the neurobiological mechanisms whereby stress affects memory and psychopathology. Currently, the main focus of her lab is to investigate the impact of stress in psychopathology, with a main emphasis in the social brain. She has been the Coordinator of the FP7 EU Project MemStick and the President of the European Brain and Behavior Society (EBBS). She holds several editorial and board commitments, including being Chief Editor of the journal Frontiers in Behavioral Neuroscience. E-mail: carmen.sandi@epfl.ch

Abstract

Carmen Sandi, Brain Mind Institute, Swiss Federal Institute of Technology Lausanne (EPFL), Switzerland
In addition to its well-known impact on cognitive function, stress has prominent effects in social behaviors. Epidemiological data in humans indicates that early life stress can have long-term consequences in individuals' personality, including increased aggression and anti-social behaviors. Moreover, other stress-related pathological conditions, such as anxiety and depression are frequently associated with alterations in both the motivation and the actual way to interact with other conspecifics. I will present different animal models developed in our lab in which stress affects the nature of social interactions in rats, their social motivation, dominance hierarchy, and aggression levels. I will briefly discuss some of the neural mechanisms that were observed to be altered by stress and linked to the deficits in social behaviors. Among others, these mechanisms include changes in the expression of synaptic cell adhesion molecules and genes of the serotonin family, as well as in the dynamics of interactions between different brain regions. Evidence for the implication of epigenetic mechanisms will be presented. In addition, I will show evidence indicating that individuals scoring high in anxiety trait show a higher probability of becoming subordinate in a social contest between two males of otherwise equivalent characteristics. The same effect is observed when individuals interact upon the influence of stress. I will, then, speculate about potential neurobiological mechanisms linking trait anxiety with subordination. Overall, I will discuss the different findings within a broader context implying stress as a strong modulator of social interactions.

Equipped to Survive: Comprehensive Response to Threat Enables Optimal Behaviour

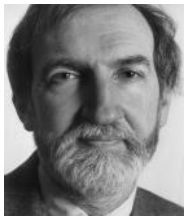
Guillén Fernández is Professor for Cognitive Neuroscience and director of the Donders Center for Neuroscience at the Radboud University Nijmegen Medical Center. He obtained his medical degree, doctorate, and habilitation at Bonn University. He received full training in clinical neurology and cognitive neurosciences in Bonn, Magdeburg, and Stanford. In 2002, he became a founding principal investigator of the Donders Center for Cognitive Neuroimaging in Nijmegen. His area of research is human cognitive neuroscience in which he studies the brain basis of memory, emotion, and their interactions. He applies an interdisciplinary approach integrating cognitive neuroimaging, genetics, pharmacology and diverse clinical disciplines. He is elected member of the Memory Disorder Research Society. He received the Richard-Jung Award of the German Society for Clinical Neurophysiology, the Vici Award of the Dutch Organization for Scientific Research, the Radboud Science Award, and an Advanced Investigator Grant from the European Research Council. Email: g.fernandez@donders.ru.nl

Abstract

In response to acute environmental adversity, organisms rapidly shift into a state that is optimal to detect and react to imminent threat. To identify underlying neural network dynamics and neuromodulatory mechanisms we combined in a series of studies fMRI with thread based stress induction procedures and pharmacological manipulation. Our data show that acute psychological stress increases responsiveness and interconnectivity within a salience related network as a function of stress response magnitudes. Beta-adrenergic receptor blockade, but not cortisol synthesis inhibition, diminished this increase. These findings reveal that noradrenergic activation during acute stress results in coupling within a distributed network that integrates information exchange between regions involved in autonomic-neuroendocrine control and vigilant, Cont../..

Cont../… attentional reorienting. This reorientation causes tonic amygdala activity and phasic responses to biologically salient stimuli while the functional connectivity to the locus coeruleus, the medial prefrontal cortex and the anterior insula is enhanced. This response goes along with higher amygdala sensitivity to threatening stimuli, but lower specificity. This heightened sensitivity is critical for survival when an individual is threatened, but cognitive elaboration would slow down appropriate reactions to threat. Accordingly, further experiments show that acute stress leads to elaboration-related working memory impairments and down regulation of associated processes in the prefrontal cortex. This instantaneously occurring reorganization affecting amygdala processing, prefrontal processing, and the connectivity between these regions is normalized by corticosteroid-related mechanisms. Most interestingly, medial temporal and prefrontal effects of cortisol appear to have different time courses, suggesting dynamically changing processes with different underlying molecular mechanisms. This pattern of results reveals basic principles of how we respond when our survival is at stake. It provides a mechanistic account for an acute central nervous stress response and its normalization. Such an account might become instrumental when investigating the pathophysiology of stress-related mental disorders, studying the mechanisms of diverse treatment approaches, and when predicting risk or outcome. Already, our approach to study the neural underpinnings of the acute threat response is capable of delineating mechanisms of how known genetic and environmental factors cause stress vulnerability.

Posttraumatic stress disorder and the brain



Chris Brewin is a Consultant Clinical Psychologist at the Traumatic Stress Clinic and Professor in the Research Department of Clinical, Educational & Health Psychology, University College London. He is a joint author of the dual representation theory of posttraumatic stress disorder, was centrally involved in shaping and overseeing Camden & Islington's lead role in the mental health response to the 2005 London bombings, and is the author of "Posttraumatic Stress Disorder: Malady or myth?" (Yale University Press 2003).

Abstract

A substantial number of structural and functional neuroimaging studies have been conducted with individuals suffering from PTSD. Many of them, however, have produced strikingly similar sets of findings to studies of depression and schizophrenia, for example involving reduced hippocampal volume and deficits in prefrontal control over limbic structures, particularly the amygdala. Investigations typically include only one psychiatric diagnosis and overlook the considerable symptom overlap between disorders. Also frequently overlooked is the presence of childhood adversity in patients' backgrounds, which is associated with neural changes in its own right. Hence previous research has not succeeded in distinguishing the correlates of generic aetiological or psychopathological factors from the specific processes that differentiate one disorder from another. This is essential if viable and well-targeted neurobiological models are to be developed that have clinical applications. Most imaging research has drawn on a limited set of basic science models such as fear conditioning. Despite its important contribution, the fear conditioning model of PTSD and paradigms such as script-driven imagery do not address important features such as the conscious reexperiencing of trauma and the distinction between voluntary and involuntary memory. The revised dual representation theory of PTSD (DRT) focusses on the difference between voluntary trauma memories and involuntary flashbacks, vivid images that are experienced in the present. Flashbacks are an important treatment target and are addressed in trauma-focussed cognitive-behaviour therapy. According to DRT, flashbacks are primarily supported by sensation-based representations created by activity in the dorsal visual stream, insula, and amygdala that have become disconnected from corresponding contextualised representations created by activity in the ventral visual stream and medial temporal lobe. Structural and functional studies will be described that focus on flashbacks and attempt to distinguish how the neural signature of PTSD is different from depression. Understanding the underlying processes and neural basis of these therapeutic target symptoms promises to generate more precise treatments, whether psychological or biological.

Speakers Short Biographies and Abstracts Day 1, Thursday 7 February

Inflammation and mental illness

Dr Neil Harrison is a Consultant Neuropsychiatrist and Head of the Psychoneuroimmunology Lab at Brighton & Sussex Medical School. His research investigates how infection/ inflammation in the body interacts with the brain to modulate emotion, motivation and cognition and contribute to common mental illnesses such as depression, chronic fatigue and Alzheimer disease.

Abstract

Once considered an immune privileged site, it is now clear that immune actions in the brain play a critical role in many fundamental neural processes and are increasingly implicated in the aetiology of mental illness. For example, cytokines, innate immune system proteins responsible for coordinating bodily responses to infection, are also critical to fundamental learning processes such as long term potentiation (LTP) in the brain. Microglial cells, the brains equivalent of macrophages, appear central to dendritic pruning and neural plasticity while MHC proteins central to self/non-self distinctions play a critical role in early neural development. Perhaps unsurprisingly given these roles aberrant immune responses are also increasingly implicated across the range of mental illnesses. In this talk I shall review mechanisms of immune modulation of neural function, summarise evidence implicating aberrant immune responses in common mental illnesses then finally present compelling new data demonstrating anti-depressant properties of a commonly used immuno-modulatory therapy. Together illustrating that psychoneuroimmunology has rapidly become an exciting new frontier for psychiatry.

Childhood stress and risk for later mental disorder

Jeremy Hall is Professor of Psychiatry and Scottish Senior Clinical Fellow. He is an Honorary Consultant Psychiatrist in General Adult and Liaison Psychiatry, and works both at the Royal Edinburgh Hospital and the New Royal Infirmary of Edinburgh. He studied Biology as an undergraduate at Oxford University before completing pre-clinical medicine at the University of Edinburgh. He then joined the MB/PhD programme at Cambridge University, graduating MB/BChir with a PhD in Experimental Psychology in 2000. Following his house jobs he completed clinical training in Psychiatry in South East Scotland. After a six month Stanley Foundation Research Fellowship he joined the Division of Psychiatry as a Clinical Lecturer in 2005. He then gained an MRC Postgraduate Research Fellowship in 2007 and was awarded a Scottish Senior Clinical Fellowship in 2010.

Abstract

Childhood stress has been associated with increased risk for later psychiatric disorders. One condition that is strongly associated with childhood stress is borderline personality disorder. Here I will present neuropsychological and imaging studies of borderline personality disorder that show evidence of a link between childhood maltreatment and emotional/social brain function and impulse control.

Speakers Short Biographies and Abstracts Day 1, Thursday 7 February

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

Stress and war: the limits of neuropsychiatry

Professor Neil Greenberg is an academic psychiatrist based at King's College London UK and is a consultant occupational and forensic psychiatrist. Neil served in the United Kingdom Armed Forces for more than 23 years and has deployed, as a psychiatrist and researcher, to a number of hostile environments including Afghanistan and Iraq.

Neil studied medicine at Southampton University and graduated in 1993. He then served as a general duties doctor in a variety of Warships, Submarines and with two Royal Marines Commando units. During his time with the Royal Marines he achieved his arctic warfare qualification and completed the all arms commando course, earning the coveted Green Beret.

Neil has specialised in Psychiatry and completed a Masters Degree in Clinical Psychiatry, a Doctorate in Mental Health and is a Fellow of the Royal College of Psychiatrists. He is a specialist in General Adult, Forensic and Liaison Psychiatry and is a member of the faculty of forensic and legal medicine and the faculty of medical leadership and management. Since 1997 Neil has been at the forefront of developing peer led traumatic stress support packages which is now in use by a wide variety of organisations. The use of Trauma Risk Management (TRiM) was initially led by the Royal Marines and has since been taken up by other organisations including the Foreign and Commonwealth Office, media organisations numerous UK police forces and the London Ambulance Service.

Neil provided psychological input for Foreign Office personnel after the events of September 11th 2001 and in Bali after 12th October 2002 bombings. He has also assisted with the aftermath management of number of other significant incidents including assisting the London Ambulance Service in the wake of the London Bombings in 2005. He has also provided mental health input into the psychological repatriation of a number of hostages over the past five years. In 2008 he was awarded the Gilbert Blane Medal by the Royal Navy for his work in supporting the health of Naval personnel through his research work.

Neil has published more than 120 scientific papers, book chapters and has presented to national and international audiences on matters concerning the psychological health of the UK Armed Forces, organisational management of traumatic stress and occupational mental health. He has been the secretary of the European Society for Traumatic Stress Studies, is a current executive board member of the UK Psychological Trauma Society and is an examiner on the Diploma in the Medical Care of Catastrophes.

Neil has extensive experiences of conducting research in military and veteran populations and has successfully published the first two ever randomised controlled trials on the effectiveness of psychological health interventions in the UK Armed Forces. He has established excellent links with veteran health providers and with US and other coalition military mental health providers and researchers. He, along with the team at King's College London, is one of the UK's premier military health researchers and has published very widely on a broad spectrum of military health related topics (www.kcl.ac.uk/kcmhr) and advise both the Armed Forces and UK governments regularly about mental health issues.

Neil has worked with News International and other media organisations including the BBC for about ten years and has provided expert input to emerging crises and clinical assessments.

Abstract
The mental health of the United Kingdom Armed Forces is a 'hot topic'. It is rare that a week goes by without a high profile media article or a senior politician commenting on the topic. However, whilst the research efforts which have investigated the psychological wellbeing of the UK Armed Forces are considerable, the results of these studies are not always well understood. Whilst there can be little doubt that for some individuals deployment to an operational theatre can be highly challenging, the emergence of new mental health problems remains uncommon. The aim of this lecture is to provide an evidence based overview of the health of the UK Armed Forces exploring how personnel's mental health varies depending on the stage of deployment (before, during and after) and the health of reservists and veterans in order to both identify the emerging issues and also to dispel some of the common myths. Much of the presented material originates from the King's Centre for Military Health Research and the Academic Centre for Defence Mental Health both based at King's College London which is the UK's leading military mental health research establishment led by Professor Sir Simon Wessely.

MEMBERS' PLATFORM PRESENTATIONS
Chair: Eileen Joyce

Temporal Lobe Epilepsy & Affective Disorders: The Role of the Subgenual Prefrontal Cortex

Authors *(presenter first):* Cleary RA, Stretton J, Winston G, Symms M, Sidhu M, Thompson PJ, Koepp M, Duncan JS, Foong J., The National Hospital for Neurology & Neurosurgery, Department of Neuropsychiatry, UK
Objective: To explore the neural substrates of affective psychopathology in temporal lobe epilepsy (TLE), using functional MRI (fMRI).
Method: A visuo-spatial "n-back" paradigm was used to compare working-memory network activation in **17** TLE patients (Median age: **40**, **14** female) with a lifetime diagnosis of depression and/or anxiety with **31** TLE patients (Median age: **38**, **17** female) with no formal psychiatric history and 30 healthy controls (Median age: 37, 18 female). There were no significant differences between the TLE groups with respect to age, gender, handedness, epilepsy onset/duration or pre-morbid IQ. All subjects completed the Beck Depression Inventory Fast Screen (BDI-FS) and Beck Anxiety Inventory (BAI) on the day of scanning. Imaging data were analysed using SPM8 software.
Results: Each group activated the fronto-parietal working memory networks, and deactivated the typical default mode network (DMN) in response to the increasing task demands. Group comparison revealed that TLE patients with a lifetime history of affective disorders showed significantly greater deactivation in bilateral subgenual prefrontal cortex than either the TLE without any psychiatric history and the healthy control groups ($p < .001$). Post-hoc analyses indicated that this main group effect persisted after co-varying for current psychotropic medication and severity of current depressive/anxiety symptoms (all p-values < .001). Correlational analysis revealed that this finding was not driven by differences in task performance ($r = .49$, $p = .33$). There were no significant differences in hippocampal volume or amygdala T2 signal between the TLE groups.
Conclusion: Hypometabolismof the subgenual prefrontal cortex bilaterally has been reported in association with primary mood disorders (1), and our findings further implicate this region. The subgenual prefrontal cortex shares extensive and reciprocal anatomical connections with areas implicated in emotional and behavioural regulation, such as the posterior orbitofrontal cortex, amygdala, hippocampus and hypothalamus. We hypothesise that altered modulation of this region with increased deactivation may be associated with a predisposition to develop mood disturbance. Our findings suggest that the same neurobiological substrate involved in the pathogenesis of primary mood disorders may also underpin affective psychopathology in TLE.
Additional information
1. Drevet WC, et al Subgenual prefrontal cortex abnormalities in mood disorders. Nature 1997;386:824-827

Effects of early childhood Posterior Fossa Tumours on IQ

Authors *(presenter first):* Carroll C[1], Clare I[1], Watson P[2], Hawkins MM[3], Spoudeas H[4], Walker D[5], Holland A[1], Ring H A[1],
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[3], Department of Public Health & Epidemiology, University of Birmingham.
[4], Department of Paediatric Endocrinology, University College Hospital, London.
[5], Children's Brain Tumour Research Centre, University of Nottingham.
Department of Applied Psychology, Salomons Campus, Canterbury Christ Church University,
Broomhill Road, Southborough,Tunbridge Wells, TN3 0TG, UK
Objective: IQ deficits have been reported amongst survivors of early childhood brain tumours, with iatrogenic causes commonly being associated with these deficits. In this study we examined IQ scores and their associations in adult survivors of an early childhood posterior fossa brain tumour (PFT).
Method: 113 people who had brain tumours under the age of 5 years had their IQ assessed using the Wechsler Abbreviated Scale of Intelligence (WASI). They were assessed an average of 32 years (range 18 - 53) after their initial tumour diagnosis. 62 of their siblings were also assessed and used as a comparison group.
Results: Overall, 16.1% of survivors had an FIQ below 70, indicating a learning disability, compared to none of the sibling control group. Mean FIQ in survivors was 89, compared to 107 in siblings. Post-hoc analysis of matched controls (n=62) were conducted. Using sibling IQ as a marker for the survivor's premorbid IQ, a significant decline in FIQ of an average of 19 points was seen as a result of PFT and its treatment. For those who received radiotherapy this increased to 24 points. For those who did not receive radiotherapy this decline was 10 points. In survivors, but not in siblings, FIQ was related to height.
In survivors, there was a significant gender difference between men and women on FIQ ($t = 2.47$; $df = 110$; $p = 0.015$), VIQ ($t = 1.99$; $df = 111$; $p = 0.049$) and PIQ ($t = 3.12$; $df = 109$; $p = 0.002$), with females having lower scores. In siblings there was no significant gender difference on any IQ scale. In survivors, radiotherapy was associated with a significant reduction in FIQ, VIQ and PIQ. There was no significant difference on IQ of having received or not received chemotherapy. There was a significant interaction between gender and radiotherapy on FIQ, with females who had radiotherapy having lower IQ than males who had radiotherapy ($F= 4.05$; $p = 0.049$).
Conclusion: Results suggested that early childhood PFT can result in significant decline in IQ, and that this decline is increased by use of radiotherapy, and may be more marked in females who had radiotherapy compared to males who had radiotherapy. Other factors, including growth hormone deficiency may influence the relationship between PFT and IQ, as suggested by the significant relationship between height and IQ.

MEMBERS' PLATFORM PRESENTATIONS
Chair: Eileen Joyce

The neural correlates of Freudian "repression" in Conversion Disorder.

Authors *(presenter first):* Selma Aybek1, Timothy R. Nicholson1, Fernando Zelaya2, Owen G. O'Daly2, Tom J. Craig3, Anthony S. David1, Richard A. Kanaan1
Section of Cognitive Neuropsychiatry, Institute of Psychiatry, King's College London, London SE5 8AF, UK
Objective: Freud proposed that in Conversion disorder (CD), the affect attached to stressful memories is "repressed" and "converted" into physical symptoms. Contemporary neuroscience has shown that the neural correlates for "repression" or memory suppression include dorsolateral prefrontal (DLPFC) cortex activation and hippocampal deactivation. Our objectives were: 1. Test this mechanism in CD and 2. Explore the neural correlates of the associated sensorimotor symptom.
Method: Stressful events were elicited from the Life Events and Difficulties Schedule interview in 12 motor CD patients and 13 healthy controls and rated by a blinded panel for their likelihood to cause CD: severely threatening events were categorised as "escape" if their consequences might plausibly be mitigated by illness. In a block-design functional magnetic resonance imaging (fMRI) task, recall of those events (Escape condition) were compared to the recall of equally threatening non-escape control events (Severe condition).
Results: Relative to controls, patients showed significant increased left DLPFC and decreased left hippocampus activity during the Escape versus Severe condition (compatible with memory suppression) and increased right supplementary motor area (SMA) and temporo-parietal junction (TPJ) activity. Patients failed to activate the right inferior frontal cortex (rIFC) during both conditions. Connectivity between amygdala and motor areas (SMA and cerebellum) was enhanced in patients relative to controls.
Conclusion: These data offer support for the notion that the way adverse events are processed cognitively can lead to physical symptoms. A plausible mechanism for the onset of these symptoms may stem from abnormal emotional control (DLPFC, rIRC) leading to memory suppression (hippocampal) with symptomatic alterations in motor planning and body schema (SMA, TPJ).

NEUROPSYCHIATRY RESEARCH UPDATE
Chair: Hugh Rickards

Deep Brain Stimulation for mental illness

Eileen Joyce is Professor of Neuropsychiatry at The Institute of Neurology and Honorary Consultant Neuropsychiatrist at the The National Hospital for Neurology and Neurosurgery. Her research focuses on neurocognitive dysfunction in the early stages of schizophrenia and how this relates to brain structural changes and clinical manifestations of the disorder. Professor Joyce received a degree in psychology from the University of Cambridge where she also completed her PhD in dopamine psychopharmacology with Susan Iversen. She went on to study medicine at Cambridge and trained in psychiatry at the Maudsley Hospital. She spent her higher clinical and research training in the neuropsychiatry department of Professor Alwyn Lishman which was followed by a period of time as a research associate at The National Institute on Alcohol Abuse and Alcoholism, USA. She returned to the UK in 1991 to take up a senior lectureship at Imperial College and remained there until 2005 when she moved to University College London.

Abstract
The recent literature in deep brain stimulation for depression and OCD will be presented and reviewed.

Autoimmunity and Neuropsychiatry

Tim Nicholson. My undergraduate medical training was at the Royal Free Hospital and School of Medicine during which I did a BSc (UCL) and an MSc (Oxford) in Biological Anthropology to pursue my interest in evolutionary biology, particularly in the factors that have driven the evolution of human biology and behaviour. I was first exposed to neuropsychiatry as a neurology trainee on Hughlings Jackson ward at the National Hospital for Neurology at Queen square which inspired my clinical and academic interest in this area, particularly in the fields of CNS autoimmunity and functional / conversion disorders. I then trained as a psychiatrist at the Maudsley Hospital, including jobs on the Lishman (neuropsychiatry) unit and a research post at both the Institute of Psychiatry and the neuroimmunology department at the Institute of Neurology. I have just completed my PhD in psychological stressors and emotion processing in conversion disorder at the Institute of Psychiatry where I am an Academic Clinical Lecturer in the Section of Cognitive Neuropsychiatry.

Abstract
Autoimmune causes are on differential diagnosis lists for many neurological, some neuropsychiatric but few, if any, purely psychiatric presentations. Why is this? Do autoimmune processes preferentially affect parts of the brain resulting in neurological, rather than psychiatric, symptoms? I propose that this is unlikely and that soon autoimmunity will be found to be the cause of a proportion (albeit probably small) of an increasing number of neuropsychiatric and even some purely psychiatric presentations. In the last few years there has been some accumulating evidence in support of this with specific antibodies associated with neuropsychiatric and pure psychiatric presentations and I will briefly review the key papers.

Speakers Short Biographies and Abstracts Day 2, Friday 8 February

EPILEPSY
Chair: Markus Reuber

Cellular mechanisms of epilepsy

John Jefferys FMedSc, Professor of Neuroscience, Neurotrauma and Neurodegeneration
School of Clinical and Experimental Medicine, College of Medical and Dental Sciences, University of Birmingham, Edgbaston, Birmingham B15 2TT Email
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Abstract
Epilepsy is a chronic disease. While anyone can experience the excessive neuronal activity of a seizure if exposed to convulsant conditions, persons with epilepsy have seizures with no obvious immediate cause. Essentially the functional organisation of the epileptogenic neural networks renders the brain of persons with epilepsy prone to have spontaneous, unpredictable, usually time-limited seizures. I will outline how focal seizures arise from networks of interconnected neurons, essentially as a “chain reaction”, and how the properties of neurons, synapses and networks are changed in chronic epileptic foci. During the prolonged “interictal” periods between seizures, these cellular and network changes provide potential explanations for behavioural and other comorbidities of epilepsy. Finally I will discuss recent work on high frequency oscillations associated with epileptic foci and their potential application in defining epileptogenic zones for surgical resection.

Funded by MRC and Epilepsy Research UK.

Brain networks in human epilepsy

Mark Richardson is a neurologist specialising in epilepsy. He is Paul Getty III Professor of Epilepsy and Head of the Department of Clinical Neuroscience at King’s College London. Professor Richardson studied medicine at Oxford and trained in neurology in London. He began his research career as a PhD student at the Institute of Neurology, joining the faculty there in 2000 as an MRC Clinician Scientist Fellow. He moved to King’s in 2005. Professor Richardson’s clinical practice spans all aspects of epilepsy, from first presentation to complex treatment-resistant epilepsy. His lab studies people with epilepsy using neuroimaging and neurophysiological techniques. Current work aims to understand the brain networks underlying epilepsy, how seizures might arise in these networks, how these network abnormalities affect cognition, and whether interventions targeting specific components of the network can improve epilepsy. He collaborates with mathematicians and engineers in developing computational models to explain phenomena associated with epilepsy.

Abstract
This contribution aims to introduce the concept of epilepsy as a network disorder. I will provide an overview of how neuroimaging and EEG might contribute to describing brain network structure, and describe how dynamic models may be introduced into network structures to reveal how seizures emerge. I will provide examples of how conventional MRI may be used to reveal network abnormalities. Lastly, I will show an example of how data from human subjects may be used to derive network structures, and combined with dynamic modelling, to cast light on how seizure susceptibility varies between individuals.

Speakers Short Biographies and Abstracts Day 2, Friday 8 February

The impact of epilepsy on cognitive function

Prof. Dr. phil. Christoph Helmstaedter, Dept. of Epileptology, University of Bonn, Bonn/ Germany

Current position and activities: Head of the section of clinical neuropsychology of the department of Epileptology, University Clinic Bonn, Germany. The major work focuses on declarative verbal and visual-spatial memory and functional plasticity in healthy subjects and epilepsy patients with temporal lobe epilepsy across the ages with special consideration of developmental neuropsychology in the maturing (developmental hindrance) and ageing (accelerated decline) brain. Other issues of interest are the frontal lobes and executive functions, cerebral functional organisation, and the development of assessment tools. The clinical work focuses on neuropsychological and behavioural monitoring and outcome control of the course of epilepsy and its medical, invasive, semi-invasive and psychological treatment. The work is linked to groups doing structural and functional imaging, surface and intracranial EEG and network analyses, neuropathology, and more recently also genetics.

Abstract
Cognitive problems and behavioral in epilepsy are frequent if they are not the rule. They can have multiple causes, the most important being brain lesions, seizures, epileptic dysfunction, and treatment. Whereas problems originating from lesions are mostly static and irreversible, problems associated with seizures and treatment are more dynamic and generally reversible. However, persisting problems can be the result of active epilepsy or treatment interfering with critical phases of brain development. Under certain individual conditions chronic and more severe epilepsy can cause mental decline. In the majority of patients this is, however, not the case. Now there is increasing evidence that cognitive problems often exist from the beginning of the disease. Mental decline thus results from synergistic effects between initial and later acquired lesions and mental aging rather than from seizures progressively damaging the brain.

This calls for neuropsychological evaluation and countermeasures early in the course of epilepsy. Antiepileptic drug treatment can have positive as well as negative effects on cognition. In chronic epilepsy the patient`s focus shifts from seizure control to the side effects of treatment and comorbidities. Thus, therapy, and polytherapy in particular, need to take the cognitive side effects of antiepileptic drugs into consideration. Repeated application of subjective questionnaires and short screening tests can be helpful. In pharmacoresistant focal epilepsy, surgery can be a successful treatment option. However, surgery and other invasive treatments can cause additional impairments, which on the other hand can be reduced by selective and individual approaches, which aim at the preservation of functional tissues. The longer term cognitive outcomes after surgery appear very promising, particularly when patients become seizure free. Respective findings suggest a reconsideration of the negative impact of interictal epileptic dysfunction on cognition.

Speakers Short Biographies and Abstracts Day 2, Friday 8 February

BNPA Plenary:
Epilepsy, Depression and Anxiety Disorders: A complex relation with significant therapeutic implications for the three conditions

Andres M. Kanner is a senior attending physician at the department of Neurology and Director of the Comprehensive Epilepsy Center at the University of Miami, Miller School of Medicine, which he joined on January 2013. For the previous 21 years, he was Director of the Laboratories of Electroencephalography and Video-EEG-Telemetry, Associate Director of the Section of Epilepsy and of the Rush Epilepsy Center at the Rush Epilepsy Center and Rush University Medical Center, where he also held the position of Professor of Neurological Sciences and Psychiatry at Rush Medical College of Rush University.

Dr. Kanner was born in Mexico City, where he grew-up and attended Medical School at the National Autonomous University of Mexico. After graduating from Mexico he moved to the USA where he completed a residency in Psychiatry at the Long-Island Jewish Hillside Medical Center in New Hyde Park, New York. In addition, Dr. Kanner completed a research fellowship in Child Psychiatry, sponsored by the National Institute of Mental Health, at the College of Physicians and Surgeons of Columbia University in New York City, a residency in Neurology at the Department of Neurology of Mount Sinai Medical Center in New York City and completed his training with a fellowship in Epilepsy and Clinical Neurophysiology at the Cleveland Clinic Foundation in Cleveland, Ohio. Dr. Kanner is triple boarded in Neurology, Psychiatry and Clinical Neurophysiology.

Dr Kanner has long-standing research interests in the areas of pharmacology of epilepsy, psychiatric aspects of epilepsy and surgical treatment of temporal lobe epilepsy. He has authored or coauthored over 80 research publications, over 75 invited review articles and over 73 book chapters and has edited two textbooks and co-edited four. In 2011, he was just appointed as Associate Editor of Epilepsy Currents, the official journal of the American Epilepsy Society. Dr. Kanner was awarded the J. Kiffin Penry Award for Excellence in Clinical Care in Epilepsy by the American Epilepsy Society in December 2010, the Epilepsy Ambassador Award from the International League Against Epilepsy in August 2011 and the Award for Outstanding Medical Service from the Epilepsy Foundation of Chicago in November 2011.

Dr Kanner serves as Co-Chair of the Neuropsychobiology Commission of the International League Against Epilepsy and as Co-Chair of the Work Group on Psychiatric Aspects of Epilepsy of the American Epilepsy Society and is as the Past Chair of Epilepsy Section of the American Academy of Neurology.

Abstract
Depression and anxiety disorders are the most frequent comorbidities in patients with epilepsy (PWE) with lifetime prevalence rates of 30 to 35%. These disorders have been typically considered to result from the seizure disorder. Yet, their relation is more complex, as the presence of primary depressive and anxiety disorders is associated with a three to seven fold higher risk to develop epilepsy. Furthermore, A lifetime history of depression and anxiety disorders preceding the onset of epilepsy have been associated with a lower probability of achieving a seizure-free state with pharmacotherapy. In patients with treatment-resistant temporal lobe epilepsy, a lifetime history of depression has been associated with a lower probability of reaching a complete seizure-free state following an antero-temporal lobectomy.

As in primary depressive and anxiety disorders, PWE are more likely to suffer from the co-occurrence of these two psychiatric conditions. The bidirectional relation with epilepsy does not necessarily imply causality (e.g., epilepsy does not cause depressive and/or anxiety disorders and vice-versa). Rather, the high comorbidity of these three conditions is suggestive of the existence of common pathogenic mechanisms. These include: 1) neurotransmitter disturbances in the transmission of serotonin, norepinephrine, glutamate, gamma-amino-butyric acid. 2) neuro-endocrine disturbances such as a hyperactive hypothalamic pituitary adrenal axis and 3) inflammatory processes involving the central nervous system.

The complex relation between these three conditions is also illustrated in the impact that epilepsy surgery has on the course of presurgical depressive and anxiety disorders in patients with treatment-resistant temporal lobe epilepsy. Indeed, about 30% to 50% of patients with presurgical disorders experience a remission after surgery, while another 20% have an exacerbation in severity or recurrence post-surgically and yet in another 10 to 15% of patients report the development of de-novo depressive and anxiety disorders.

This presentation will review the complex relation between depression, anxiety and epilepsy and its impact on their therapeutic response to treatment.

Speakers Short Biographies and Abstracts Day 2, Friday 8 February

CONSCIOUSNESS
Chair: Peter Halligan

Decoding consciousness

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.

Abstract
Consciousness is central to the human condition, furnishing us with phenomenal awareness of the external world and the ability to reflect upon our own thoughts and experiences. Almost half our communication concerns the contents of our thoughts and experiences. The shared language we use to do this obscures the recent realization that there is substantial variability in how different people experience the same physical environment. Moreover, key aspects of this variability in conscious experience are heritable, suggesting a conscious phenotype with adaptive significance. In this talk I will explore the nature of individual differences in conscious perception and their neural basis, focusing on both structure and function of the human brain.

Psychedelic drugs, magical thinking and psychosis

Robin Carhart-Harris, Imperial College, London

After completing an undergraduate degree in Psychology in 2003, Robin studied psychoanalysis at Masters level, receiving his MA in 2004. In 2005, Robin began a four year PhD in Psychopharmacology at the University of Bristol. Working for Professor David Nutt and Dr Sue Wilson, Robin's thesis focused on sleep and serotonin function in ecstasy users. Robin conducted a clinical study involving sleep electroencephalography (EEG) and tryptophan depletion.

In 2009, working closely with the Beckley Foundation, he successfully coordinated the first clinical study of psilocybin in the UK and the first clinical study of a classic psychedelic drug in the UK for over 40 years. Also in 2009, Robin moved to Imperial College London to continue his work under the supervision of Professor David Nutt. With the collaboration of Professor Richard Wise at Cardiff University, Robin has since coordinated the first resting state fMRI investigation of a classic psychedelic drug and the first fMRI and PET investigations of psilocybin and MDMA. Robin is first author on a number of publications in peer-reviewed scientific journals including review articles with eminent neuroscientists Professor's Helen Mayberg and Karl Friston. He has presented his data at several international conferences and has appeared on BBC News.

Abstract

Psychedelic ('psyche' = soul/mind and 'delos' = to make visible or clear) drugs have been used for centuries in mystical ceremonies. In the 1950/60s they were used widely in psychotherapy, under the premise that they lower psychological defences and facilitate psychological insight. Since the 1950s, the psychedelic state has been considered a model of psychosis. Today, all of these properties of psychedelics are researched: a single high-dose of psilocybin (magic mushrooms) has been found to produce profound, personality-changing spiritual-type experiences in healthy participants1, 2, psilocybin has been found to be effective in psychotherapy for end-of-life anxiety3 and the pharmacological pathways through which psychedelic effects are elicited (i.e. stimulation of the serotonin 2A receptor) continue to be linked with psychosis4. These variegated properties do not seem entirely consistent with one another – e.g. how can the same drug be both psychotomimetic and therapeutically useful? This presentation will attempt to resolve this apparent paradox. Evidence will be cited to support the hypothesis that the prodromal phase of first-episode psychosis, spontaneously occurring spiritual experiences and the psychedelic drug state rest on the same neurobiological state – hereafter referred to as the primitive state. This state is described psychologically as evolutionarily regressive, i.e. it is a state the mind and brain falls back to under certain conditions. This evolutionarily primitive state is characterised by magical thinking: i.e. fallacious thinking in which reality-testing is disavowed and thoughts are easily biased by wishes and anxieties. In the spiritual experience it is wishful fantasies that predominate (although not entirely) and in psychosis, it is paranoid thinking. In the psychedelic state, both wishful and paranoid thinking are common and the valence of the experience is highly sensitive to the environment in which it unfolds. That this state is so sensitive to environmental perturbation has important implications for both psychotherapy with psychedelics and treatment approaches for patients exhibiting signs of psychosis-risk – as it emphasises the importance of shepherding the experience in a positive direction. Underneath its sensitivity to suggestion however, is a more fundamental property of the primitive state: that the perception of difference or separateness breaks down. Evidence from functional brain imaging of a decrease in the orthogonality of different brain states in psychosis, the psychedelic-state and the meditative state is presented – and used to support the hypothesis that there is a breakdown of 'multiplicity' in the primitive state which lies at the base of descriptions of 'union' or 'oneness'5.

1. Griffiths RR, Richards WA, McCann U, Jesse R. Psilocybin can occasion mystical-type experiences having substantial and sustained personal meaning and spiritual significance. Psychopharmacology. 2006; 187(3): 268-83; discussion 84-92.
2. MacLean KA, Johnson MW, Griffiths RR. Mystical experiences occasioned by the hallucinogen psilocybin lead to increases in the personality domain of openness. J Psychopharmacol. 2011; 25(11): 1453-61.
3. Grob CS, Danforth AL, Chopra GS, Hagerty M, McKay CR, Halberstadt AL, et al. Pilot study of psilocybin treatment for anxiety in patients with advanced-stage cancer. Archives of general psychiatry. 2011; 68(1): 71-8.
4. Wischhof L, Koch M. Pre-treatment with the mGlu2/3 receptor agonist LY379268 attenuates DOI-induced impulsive responding and regional c-Fos protein expression. Psychopharmacology. 2012; 219(2): 387-400.
5. Carhart-Harris RL, Leech R, Erritzoe D, Williams TM, Stone JM, Evans J, et al. Functional Connectivity Measures After Psilocybin Inform a Novel Hypothesis of Early Psychosis. Schizophrenia bulletin. 2012.

Members' Posters

1. Title: Metabolic Profiles of Young Patients with Tourette Syndrome treated with Aripiprazole and Pimozide.
Authors: Andrea E. Cavanna, Clare M. Eddy, Paola CalÃ-, Mariangela Gulisano, Renata Rizzo
Department of Neuropsychiatry, The Barberry National Centre for Mental Health, BSMHFT and University of Birmingham, UK

2. Title: The entrainment test in tremor assessment: Influence of historical factors and clinical methodology
Authors: Louise S Roper, Hugh Rickards

3. Title: Central expression of abnormal and unexplained skin sensations
Authors: Jessica A Eccles (1), Sarah N Garfinkel (1,2), Ruth E Taylor (3), Anthony P Bewley (4), HugoD Critchley(1,2)
Clinical Imaging Sciences Centre, Brighton and Sussex Medical School, University of Sussex, Falmer BN1 9RR, United Kingdom
1. Department of Psychiatry, Brighton and Sussex Medical School, UK
2. Sackler Centre for Consciousness Science, University of Sussex, UK
3. Department of Psychiatry, Barts and the London NHS Trust, London, UK
4. Department of Dermatology, Barts and the London NHS Trust, London UK

4. Title: Impaired decision-making and diffusion orientational complexity in people with multiple sclerosis
Authors: Muhlert N, Sethi V, Ron M, Cipolotti L, Parker G, Haroon HA, Yousry T,
Wheeler-Kingshott CA, Miller DH, Chard DT, NMR Research Unit, Department of Neuroinflammation, UCL Institute of Neurology, London, WC1N 3BG, United Kingdom

5. Title: A review of stress and endogenous opioid interaction in alcohol addiction
Authors: Emsley E,1 Lees R,2 Lingford-Hughes A,2 Nutt D.2
1. University of Southampton, 19 Whitehill, Bradford on Avon, Wiltshire, BA15 1SG, United Kingdom, 2. Imperial College London

6. Title: Psychopathology in adults with intellectual disabilities with or without epilepsy: A case control study.
Authors: Okafor GE, Deb S, Cavanna AE, Unwin G

7. Title: Are psychiatric symptoms a core phenotype of Myoclonus Dystonia Syndrome caused by SGCE mutations?
Authors: Peall KJ, Smith DJ, Kurian MA, Wardle M, Waite AJ, Hedderly T, Lin JP, Smith M, Whone A, Pall H, White C, Lux A, Jardine P, Bajaj N, Lynch B, Kirov G, O'Riordan S, Samuel M, Lynch T, King MD, Chinnery PF, Warner TT, Blake DJ, Owen MJ, Morris HR. Cowbridge, South Wales, UK.

8. Title: Depressive symptoms in Tourette syndrome and affective disorders: A controlled study
Authors: John Carlo P., Piedad, Katherine Gordon-Smith, Lisa A. Jones, Andrea E. Cavanna
Department of Neuropsychiatry, BSMHFT and University of Birmingham, UK

9. Title: Behavioural profile of zonisamide in adult patients with epilepsy and neuropsychiatric comorbidity
Authors: Andrea E. Cavanna, Stefano Seri,Department of Neuropsychiatry, BSMHFT and University of Birmingham, United Kingdom

10. Title: A case of organic amnesic disorder syndrome diagnosed with fMRI
Authors: Michihiko Koeda, Yuichi Takizawa, Kaoru Minagawa, Masahiro Yamamoto, Tetsuya Ichimiya, Amane Tateno, Pascal Belin, and Yoshiro Okubo, 1.Voice Neurocognition Lab, Dept of Psychology, Centre for Cognitive Neuroimaging, University of Glasgow, 58 Hillhead Street, Glasgow, G12 8QB
2. Department of Neuropsychiatry, Nippon Medical School, 1-1-5, Sendagi, Bunkyo-Ku, 113-8603, Tokyo, Japan

11. Title: Attention deficit and hyperactivity symptoms in adult patients with Tourette syndrome
Authors: Ashley Liew, Andrea E. Cavanna, CAMHS LD, 40 Rupert Street, Nechells, Birmingham, B7 4PS, United Kingdom

12. Title: Inpatient treatment of severe motor conversion disorder: a case-control study.
Authors: McCormack R (1), David AS (2).84 Gowan Avenue, London SW6 6RG. United Kingdom
1. ST3 Academic Clinical Fellow, NIHR Biomedical Research Centre, South London & Maudsley NHS Foundation Trust and Institute of Psychiatry, King's College London.
2. Section of Cognitive Neuropsychiatry, Institute of Psychiatry, King's College London and NIHR Biomedical Research Centre, South London and Maudsley NHS Foundation Trust and Institute of Psychiatry, King's College London.

13. Title: Serial drug usage for tics in Tourette syndrome - When to give up?
Authors: Mena Farag, Jeremy S. Stern, David Williams, Kathryn Grabecki, Helen Simmons, Mary M. Robertson
Department of Neurology, Atkinson Morley's Wing, St. George's Hospital, Blackshaw Road
London, SW17 0QQ, United Kingdom

Members' Posters

14. Title: Epilepsy in Tourette syndrome
Authors: David Williams, Jeremy S. Stern, Kathryn Grabecki, Helen Simmons, Mary M. Robertson
Department of Neurology, Atkinson Morley's Wing, St. George's Hospital, Blackshaw Road
London, SW17 0QQ, United Kingdom

15. Title: A Questionnaire Study Investigating Relationships Between Physical and Mental Health, Repressive Coping and Ageing
Authors: S Khodatars, J Erskine, London, United Kingdom

16. Title: The development of a new apathy measurement scale: Dimensional Apathy Scale
Authors: Mr Ratko Radakovic and Dr Sharon Abrahams
Department of Psychology, School of Philosophy, Psychology and Language Sciences, The University of Edinburgh, 7 George Square, EH8 9JZ, United Kingdom

17. Title: Neural Correlates of De novo Depression Following Left Temporal Lobe Epilepsy Surgery: A Voxel Based Morphometry Study of Pre-surgical Structural MRI
Authors: Cleary RA, Centano M, Flugel D, Symms M, Thompson PJ, Koepp M, Foong J.
The National Hospital for Neurology & Neurosurgery, Department of Neuropsychiatry, UK

18. Title: Cognitive impairments in ALS relate to white matter integrity in specific regions of interest
Authors: Pettit L. D., Bastin, M., & Abrahams, S.
Psychology Department PPLS, 7 George Sq, University of Edinburgh, EH8 9JZ, United Kingdom

19. Title: Association of apathy with frontal lobe dysfunction in amnestic mild cognitive impairment and Alzheimer's disease
Authors: Dr Manoj George, Tim Whitfield, Zuzanna Walker

20. Title: Association between apathy and the caregiver burden in amnestic mild cognitive impairment and Alzheimer's disease
Authors: Dr Manoj George

Members' Posters

1. Title: Metabolic Profiles of Young Patients with Tourette Syndrome treated with Aripiprazole and Pimozide.
Authors: Andrea E. Cavanna, Clare M. Eddy, Paola CalÃ-, Mariangela Gulisano, Renata Rizzo
Department of Neuropsychiatry, The Barberry National Centre for Mental Health, BSMHFT and University of Birmingham, UK
Objective: This study assessed the metabolic effects of aripiprazole and pimozide in children with Tourette syndrome, a neurodevelopmental condition characterised by multiple motor and phonic tics.
Method: Young patients treated with aripiprazole (n=25) and pimozide (n=25) were compared to patients who were medication-free (n=25) for metabolic parameters. Body mass index, glycemia, triglyceridemia and cholesterolemia were monitored at baseline, 12 and 24 months after treatment commencement.
Results: The aripiprazole group showed significant increases in cholesterolemia, while the pimozide group showed significant increases in glycemia. Both groups showed elevations in triglyceridemia which were not significantly different to those seen in un-medicated controls.
Conclusion: While both aripiprazole and pimozide appear relatively safe for use in children with Tourette syndrome, these findings will help guide medication selection in cases with specific medical vulnerabilities.

2. Title: The entrainment test in tremor assessment: Influence of historical factors and clinical methodology
Authors: Louise S Roper, Hugh Rickards. Birmingham, UK
Objective: Diagnosing functional tremors is challenging, usually relying solely on history and examination. However, it has been suggested that clinicians may disregard the outcome of observational tests; basing diagnoses on history alone. The entrainment test is described as a useful clinical test for distinguishing between functional and organic tremors but the literature suggests variability in the way that it is performed and interpreted. This study aimed to investigate the extent of the variability in the way that clinicians perform and interpret the entrainment test when assessing tremors, as well as the influence of history on clinicians' assessment of the test.
Method: 31 clinicians, recruited from specialist movement disorder centres and conferences, answered a novel questionnaire assessing performance and interpretation of the entrainment test. Clinicians watched videos of patients with organic and functional tremors performing the test. After each video clinicians decided whether the test was positive or negative. They were then read a fictional history and given the opportunity to change their assessment.
Results: 4 out of 62 initial assessments changed, independent of the history that the clinician heard (ï £2=1.974, p=0.542). The mean questionnaire score was 6.7 out of 12 (standard deviation 2.3). Those reporting confidence in their knowledge of the entrainment test scored significantly higher, indicating greater knowledge, than those reporting limited confidence (mean=7.8, standard deviation 1.9, 95% confidence interval, 7.0-8.7 vs. mean=5.2, standard deviation 2.1, 95% confidence interval, 4.1-6.4. t=3.658, p=0.001). 5 clinicians did not include a definition of "pure entrainment" when asked for signs signifying a positive test.
Conclusion: History does not exert undue influence over assessment of the entrainment test, except in a minority of cases. Training and published guidelines are needed to standardise entrainment test methodology.

3. Title: Central expression of abnormal and unexplained skin sensations
Authors: Jessica A Eccles (1), Sarah N Garfinkel (1,2), Ruth E Taylor (3), Anthony P Bewley (4), HugoD Critchley(1,2)
Clinical Imaging Sciences Centre, Brighton and Sussex Medical School, University of Sussex, Falmer BN1 9RR, UK
1. Department of Psychiatry, Brighton and Sussex Medical School, UK
2. Sackler Centre for Consciousness Science, University of Sussex, UK
3. Department of Psychiatry, Barts and the London NHS Trust, London, UK
4. Department of Dermatology, Barts and the London NHS Trust, London UK
Objective: A sub-group of patients present to dermatological services with unexplained skin sensations that characteristically evoked a subjective sense of infestation. The consequent psychological and behavioural impacts of these experiences are considerable and difficult to manage therapeutically. Underlying neurobiological mechanisms are unclear. We therefore undertook the first functional MRI study to test the hypothesis that such patients will differ from controls in central neural processing of affective and infestation-related stimuli
Method: Participants: Five patients presenting with medically unexplained skin sensations were recruited from the specialist psychodermatology service at The Royal London Hospital, mean age 52.8 years, 4 female, 1 male. Five healthy controls were matched for age and gender. Image acquisition: Whole brain MRI data was acquired on a 1.5 T scanner. Task: In a randomized event- related design participants were shown 6 classes of images - insects on skin; insects on leaf; other objects on skin; other objects on leaf; neutral images; disgusting and fearful images. Analysis: Functional images were analyzed using SPM8. A full factorial model was used to analyze the results with two factors - group and stimulus type.
Results: Across all conditions patients showed greater activity in the right parahippocampus. Insect versus non insect images evoked greater activation within occipital regions. The main effect of presentation of skin rather than leaf stimuli was to activate inferior parietal lobule and the patients showed enhanced activity within this area. Formal testing of differential responses of patients v. controls to images of insects on skin (three way interaction) revealed differences in the engagement of dorsal anterior cingulate and right lateral prefrontal cortices. Patients also showed greater activity in bilateral temporal lobes when viewing disgusting/fearful images compared to neutral images.
Conclusion: We confirm that regional neural activity differs between patients with abnormal skin sensations and controls to condition-relevant and affective visual stimuli. These data provide insight into central mechanisms that potentially represent novel treatment targets.

Members' Posters

4. Title: Impaired decision-making and diffusion orientational complexity in people with multiple sclerosis
Authors: Muhlert N, Sethi V, Ron M, Cipolotti L, Parker G, Haroon HA, Yousry T,
Wheeler-Kingshott CA, Miller DH, Chard DT, NMR Research Unit, Department of Neuroinflammation, UCL Institute of Neurology, London, WC1N 3BG, UK
Objective: Difficulties with decision-making have been reported in people with multiple sclerosis (MS). It is however unclear what aspect of decision making is impaired, for example whether they are more impulsive, and how any impairments relate to grey matter pathology. In this study we assess grey matter microstructure using a novel measure of the number of diffusion orientations on diffusion MRI " diffusion orientation complexity (DOC)". We studied DOC in cortical areas known to be associated with decision making and looked at its associations with performance on the Cambridge Gambling Task (CGT).
Method: One hundred and five patients with MS (61 RR, 26 SP, 18 PP; mean age: 45.9 years) and 36 healthy controls (mean age: 39.6 years) were studied. Decision making performance was assessed using the CGT. T1-weighted (T1w) scans (1x1x1mm) and cardiac-gated diffusion scans (2x2x2mm, 61 directions at b=1200s/mm2, 7 at b=0) were acquired on a 3T system. T1w images were registered to diffusion scans and segmented to extract GM. GM regions with a priori evidence of an association with decision-making (caudate, hippocampus, middle frontal gyrus, anterior cingulate, medial prefrontal cortex; all bilateral) were masked using the Oxford-Harvard template and mean DOC was measured in these GM regions.
Results: On the CGT, patients showed less adjustment of bets to account for the level of risk (t-test, p <0.01) and were significantly slower at making decisions (p=0.01). Patients also showed significantly lower DOC in the caudate (p><0.01), the middle frontal gyrus (p><0.001), the anterior cingulate (p><0.01), and the medial prefrontal cortex (p=0.001), and significantly higher DOC in the hippocampus (p><0.05), relative to controls. In patients, risk adjustment correlated with hippocampal DOC (r=-.23, p><0.05), the length of deliberation correlated with medial prefrontal DOC (r=-0.22, p><0.05) and the quality of decision-making correlated with DOC in the anterior cingulate (r=0.34, p=0.001). >
Conclusion: Decision-making deficits in people with MS relate to difficulties in adjusting to levels of risk rather than increased impulsivity. Changes in decision-making correlate with DOC in grey matter regions associated with those functions, suggesting a relationship with abnormal grey matter microstructure, such as loss of neurites, which is known to occur in MS.

5. Title: A review of stress and endogenous opioid interaction in alcohol addiction
Authors: Emsley E,1 Lees R,2 Lingford-Hughes A,2 Nutt D.2
1. University of Southampton, 19 Whitehill, Bradford on Avon, Wiltshire, BA15 1SG, United Kingdom, 2. Imperial College London
Objective: The endogenous opioid system (EOS) is involved in hedonic processing of reward, positive reinforcement, impulsivity, and potentially craving in alcohol dependence. Stress manifests in multiple stages of addiction, including early susceptibility to alcohol abuse, progression to dependence and risk of stress-related relapse. This literature review aims to collate evidence for the role of stress and opioid systems in alcohol addiction and identify novel interactions, with major importance in alcoholism treatment. Further, the actions of nalmefene and naltrexone, which may inform stress/opioid interaction in alcohol dependence, will be discussed.
Method: Published articles were identified with a MEDLINE search (January 1980 to March 2012). Key terms included: opioid, hypothalamic-pituitary-adrenal (HPA) axis, alcoholism, naltrexone, nalmefene. An initial review of titles preceded a second review of abstracts to identify articles meeting inclusion criteria.
Results: Alcohol addiction involves dysregulation of the EOS and stress systems, though precise abnormalities are uncertain. Normalisation of these systems in abstinence may occur, with inconsistent timings and degrees. The EOS has considerable interactions with stress at multiple levels of the HPA axis. Naltrexone, a relatively 1¼- specific opioid antagonist, paradoxically improves blunted 1²-endorphin activity in alcoholism. Naltrexone may also increase HPA responsiveness, with potential HPA normalisation, indicating an EOS-stress link. Nalmefene acts at all opioid receptors, especially K- which binds dynorphin. Dynorphin and stress interact in withdrawal, thus nalmefene may be effective in targeting relapse by negative reinforcement.
Conclusion: Stress and EOS abnormalities in alcoholism, and extent of normalisation of these systems in abstinence, require further investigation. There is evidence for multifaceted interactions between opioid and stress systems in alcoholism. Since naltrexone and nalmefene have differential activity at EOS receptors, comprehensive characterisation of naltrexone and nalmefene action on stress systems is critical. Delineation of the effect of these drugs on HPA normalisation in abstinence, potentially via the EOS, is timely. This could ensure targeted and evidence-based care in those with alcohol dependency.

Members' Posters

6. Title: Psychopathology in adults with intellectual disabilities with or without epilepsy: A case control study.
Authors: Okafor GE, Deb S, Cavanna AE, Unwin G
Aims: Epilepsy-related factors may increase the risk of developing psychopathology in adults with intellectual disabilities (ID). We examined the patterns of problem behaviours and psychiatric disorders among adults with ID and epilepsy.
Methods: We recruited 23 adults with ID and epilepsy (11 males) and a control group of 23 adults with ID alone (13 males) matched for age and ID level. The mean age of the overall sample was 41 years (SD=17). Their carers were interviewed using the Modified Overt Aggression Scale (MOAS) (which provided a total score for aggression but also separate scores for verbal aggression, and physical aggression against people, objects and self respectively), and mini PAS-ADD Interview to assess co-morbid psychiatric disorders.
Results: A significantly higher proportion of adults with ID and epilepsy, compared with adults with ID alone manifested aggression against people and objects. Although there was no significant difference between the two groups in the proportion of adults meeting mini PAS-ADD Interview threshold scores for the presence of at least one psychiatric disorder, a higher proportion of patients in the non-epilepsy group (26%) compared with the epilepsy group had depression (4%). The results also showed that higher seizure frequency was associated with aggression against people and total MOAS aggression scores.
Conclusions: This study supports previous research (Deb and Hunter 1991a, b) that the overall rates of psychopathology and patterns of aggressive behaviours or psychiatric disorders are similar among adults with ID with or without epilepsy, although frequent seizures increases the risk for aggression against people.
Reference :
Deb S. & Hunter D. (1991a) Psychopathology of people with mental handicap and epilepsy. I: Maladaptive behaviour. *British Journal of Psychiatry*, **159**, 822-826.
Deb S. & Hunter D. (1991b) Psychopathology of people with mental handicap and epilepsy. II. Psychiatric illness. *British Journal of Psychiatry*, **159**, 826-830.

7. Title: Are psychiatric symptoms a core phenotype of Myoclonus Dystonia Syndrome caused by SGCE mutations?
Authors: Peall KJ, Smith DJ, Kurian MA, Wardle M, Waite AJ, Hedderly T, Lin JP, Smith M, Whone A, Pall H, White C, Lux A, Jardine P, Bajaj N, Lynch B, Kirov G, O'Riordan S, Samuel M, Lynch T, King MD, Chinnery PF, Warner TT, Blake DJ, Owen MJ, Morris HR. South Wales, United Kingdom
Objective: Myoclonus Dystonia Syndrome (MDS) is a childhood onset, alcohol responsive movement disorder caused by mutations in the SGCE gene in a proportion of cases. Single family and case series have suggested co-morbid psychiatric disease but have not compared cases to a control group.
Aims: To establish a cohort of MDS patients with SGCE mutations and a control group of alcohol-responsive tremor patients, and to systematically assess for psychiatric symptoms using standardised questionnaires.
Method: We collected 27 patients with SGCE mutations and 45 tremor control cases. The MINI International Neuropsychiatric Interview, PHQ-9, MADRS, YBOCS and AUDIT were used to assess psychiatric disease according to DSM-IV criteria.
Results: There was a higher rate of psychiatric disease in MDS patients compared to controls (p <0.05), specifically social phobia (p><0.05) and Obsessive-Compulsive disease (OCD) (p><0.001). Excess alcohol use was higher amongst the MDS group once cases ><18yrs were excluded. >
Conclusion: Overall psychiatric disease is elevated amongst the MDS cohort compared to a control group with a chronic, socially stigmatizing disorder. OCD appears to be the greatest contributor to this effect and may reflect a pleiotropic function for the SGCE gene.

8. Title: Depressive symptoms in Tourette syndrome and affective disorders: A controlled study
Authors: John Carlo P., Piedad, Katherine Gordon-Smith, Lisa A. Jones, Andrea E. Cavanna
Department of Neuropsychiatry, BSMHFT and University of Birmingham, UK
Objective: Tourette syndrome (TS) is a neuropsychiatric condition characterised by multiple motor and vocal tics, as well as a spectrum of behavioural problems. Previous research found that 76% of patients with TS experience depressive symptoms, with 13% fulfilling diagnostic criteria for depression. We set out to assess the severity of affective symptoms in patients with TS, in comparison to patients with primary affective disorders (recurrent major depressive disorder, rMDD; bipolar affective disorder types I/II, BPD-I/II), and healthy controls.
Method: Both patients with affective disorders and controls completed the Beck Depression Inventory (BDI)-IA, whilst patients with TS completed the BDI-II. Total BDI-II scores were transformed using an equipercentile equating method for converting raw total BDI-II to BDI-IA scores. Data from 14/21 items had corresponding anchor points between the two versions and were therefore suitable for analysis.
Results: This cross-sectional study included N=3,066 participants: TS (N=65), rMDD (N=696), BPD-I (N=1515), BPD-II (N=497), and controls (N=293). Depressive symptoms did not show any association with ethnicity or age. Patients with TS scored significantly higher than healthy controls (P=<.001) for all relevant items. When comparing depression ratings between patients with TS and patients with primary affective disorders, total BDI scores in TS were not significantly different from BPD-I or BPD-II, but significantly lower than rMDD (P=.030). Specifically, patients with rMDD scored higher on the self-criticalness, libido, suicidality and anergia items (P=.003 - ><.001). The TS group also showed statistically significant differences in BDI scores across gender, with female patients reporting higher scores (P=.013), particularly in the guilt, suicidality, crying, irritability and libido items (P=.047 - .002). >
Conclusion: Depression appears to be a prominent feature in TS and seems to have a different phenotype to that in rMDD. Female patients with TS present a particularly high risk to develop severe depressive symptoms. This has relevant clinical implications in terms of screening, management and prognosis of this patient population.

Members' Posters

9. Title: Behavioural profile of zonisamide in adult patients with epilepsy and neuropsychiatric comorbidity
Authors: Andrea E. Cavanna, Stefano Seri,Department of Neuropsychiatry, BSMHFT and University of Birmingham
Objective: Zonisamide is a newer antiepileptic drug indicated as adjunctive therapy in the treatment of adult patients with partial-onset seizures, with or without secondary generalization. Following isolated reports of zonisamide-induced mania, other behavioural adverse effects, including psychosis and suicidal ideation have been associated with its use, and it was suggested that past psychiatric history is among the factors associated with discontinuation of zonisamide therapy in patients with epilepsy. We therefore set out to assess the tolerability profile of zonisamide in this particular group of patients with epilepsy, who are at risk of developing adverse reactions to zonisamide.
Method: This study investigated the prevalence and characteristics of adverse effects resulting from the use of zonisamide in a retrospective chart review of patients with epilepsy and co-morbid cognitive and/or behavioural problems, recruited from the specialist neuropsychiatry clinic at the National Centre for Mental Health, BSMHFT and University of Birmingham.
Results: We identified 12 eligible patients (3 males, mean age 36 years, range 16-59 years). All patients had a clinical diagnosis of treatment-refractory epilepsy (9 = temporal lobe epilepsy), supported by neurophysiological and neuroimaging findings, and had concomitant and/or previous antiepileptic medications (11 = levetiracetam, 8 = carbamazepine, 6 = lamotrigine, 6 = valproate). In our neuropsychiatric sample, 6 patients had a previous diagnosis of depression, 2 anxiety disorders, 2 learning disability, 2 neurodevelopmental disorders (Tourette syndrome and autism) and 1 psychosis. Co-morbid non-epileptic attack disorder was documented in 4 patients. In the majority of cases, zonisamide (mean maintenance dose = 212.5mg daily, range 50-500 mg daily) was well tolerated and behavioural adverse effects were not severe. Three patients (25.0%) discontinued zonisamide over the observation period (mean duration 15 months, range 1-48 months). The main reasons for discontinuation were lack of efficacy on seizure control (two cases) and emerging depression as an adverse effect (one case).
Conclusion: This preliminary observation of relatively low discontinuation rate of zonisamide in a selected population of patients with epilepsy and neuropsychiatric comorbidity prompts further research to establish whether this medication is a safe treatment option for vulnerable patients with treatment-refractory epilepsy.

10. Title: A case of organic amnesic disorder syndrome diagnosed with fMRI
Authors: Michihiko Koeda, Yuichi Takizawa, Kaoru Minagawa, Masahiro Yamamoto, Tetsuya Ichimiya, Amane Tateno, Pascal Belin, and Yoshiro Okubo, 1.Voice Neurocognition Lab, Dept of Psychology, Centre for Cognitive Neuroimaging, University of Glasgow, 58 Hillhead Street, Glasgow, G12 8QB
2. Department of Neuropsychiatry, Nippon Medical School, 1-1-5, Sendagi, Bunkyo-Ku, 113-8603, Tokyo, Japan
Objective: Post-concussion syndrome after traffic accident is often clinically associated with prolonged cognitive impairment due to brain lesions, but sometimes a symptom like amnesia may occur without structural abnormality in neuroimaging. The question is whether cognitive impairment may be caused by subtle lesions that cannot be detected with conventional structural MRI or CT. We report a case in which measurement of cognitive function with functional Magnetic Resonance Imaging (fMRI) was useful to assess the organic origin of an amnesic disorder after concussion.
Method: A 48 year old female suffered from prolonged memory disturbance after concussion. Her brain structural CT and MRI (T1 and T2) appeared normal. Her consciousness level was alert. Insomnia, physical anxiety, and depressive symptom were not observed. In order to investigate cerebral function with regards to memory processing, we carried out an fMRI study to test working memory (WM) for emotional voice stimuli from the Montreal Affective Voices. Furthermore, the neurobehavioral cognitive status was tested with a battery of neuropsychological tests (Cognistat).
Results: Brain structural CT and MRI were normal, however, the patient's cerebral activation was significantly reduced in the bilateral hippocampi and left inferior frontal gyrus compared to 14 healthy control subjects in the WM vs Rest contrast. Furthermore, amnesic deficits were observed in Cognistat.
Conclusion: These results demonstrate that fMRI along with neuropsychological assessment is a useful tool to unravel organic origin of an amnesic disorder even if structural brain imaging appears normal.

11. Title: Attention deficit and hyperactivity symptoms in adult patients with Tourette syndrome
Authors: Ashley Liew, Andrea E. Cavanna, CAMHS LD, 40 Rupert Street, Nechells Birmingham, B7 4PS
Objective: Although attention deficit and hyperactivity disorder (ADHD) is a common co-morbidity in children and young people with Tourette syndrome (TS), little is known about the clinical correlates and impact of ADHD symptoms in adults with TS. We set out to compare tic severity and health-related quality of life (HR-QOL) ratings between adult patients with TS only versus patients with TS plus ADHD.
Method: Patients were recruited from the specialist Tourette syndrome clinic at the Department of Neuropsychiatry, Birmingham & Solihull Mental Health Foundation Trust and University of Birmingham. We compared 40 patients with TS only with 32 patients with TS plus ADHD using standardised self-report measures of Adult ADHD (Adult ADHD Severity Scale), tic severity (MOVES) and disease-specific HR-QOL (Gilles de la Tourette Syndrome â€“ Quality of Life Scale, GTS-QOL).
Results: We found a highly significant difference in tic severity as measured by the MOVES scale, with the TS plus ADHD group reporting higher scores. Likewise, there were highly significant differences in GTS-QOL scores, with the TS plus ADHD group demonstrating worse HR-QOL perception. Finally, the severity of ADHD symptoms showed a significant association with poorer HR-QOL.
Conclusion: Adult patients with TS and co-morbid ADHD tend to have higher tic severity and poorer HR-QOL compared to patients with TS only. Clinicians treating adults with TS should screen their patients for residual ADHD symptoms, in order to ensure that appropriate management is provided to prevent potential behavioural difficulties and other functional impairments.

Members' Posters

12. Title: Inpatient treatment of severe motor conversion disorder: a case-control study.

Authors: McCormack R (1), David AS (2).84 Gowan Avenue, London SW6 6RG. United Kingdom
1. ST3 Academic Clinical Fellow, NIHR Biomedical Research Centre, South London & Maudsley NHS Foundation Trust and Institute of Psychiatry, King's College London.
2. Section of Cognitive Neuropsychiatry, Institute of Psychiatry, King's College London and NIHR Biomedical Research Centre, South London and Maudsley NHS Foundation Trust and Institute of Psychiatry, King's College London.

Objective: AIMS: To evaluate the characteristics and outcomes of patients with motor conversion disorder admitted to a specialist neuropsychiatry unit.
Method: The study included all (n=33) patients discharged from the Lishman Neuropsychiatry Unit (Maudsley Hospital) between 2007 and 2011 with an ICD-10 diagnosis of dissociative motor disorder following multidisciplinary treatment. Brain injury inpatients (n=33, age-and sex-matched) acted as controls. Data extracted included demographic details, duration of illness prior to admission, length of stay, medical/psychiatric co-morbidity and history, history of abuse, history as a carer or health/social-care professional, and status with regards to employment, mobility, and activities of daily living (ADLs) on admission and discharge.
Results: The mean age of cases was 40.8yrs (s.d. 12.1, range 20-59; not significantly different from controls p=0.299). Both groups were 78.8%(n=26) female. Cases had marked levels of functional impairment on admission with 60.6% (n=20) wheelchair or bedbound, 42.4%(n=14) dependent for ADLs, and an 87.9%(n=29) unemployment rate. The mean Modified Rankin Scale (MRS) score for cases was 3.64 (s.d. 0.86, range disability 2 [slight]-5 [severe]), with a median length of illness pre-admission of 48months (IQR 19-72), and median length of stay 101days (IQR 84-130). All three values were higher than controls (p=0.003, p<0.001, p=0.03 respectively). >
Cases were significantly more likely than controls to have a history of all forms of abuse, particularly childhood sexual abuse (p<0.001; n=12/36.4% of cases), a pre-morbid non-dissociative psychiatric history (p><0.001; n=27/81.8% of cases), and a history of prior employment as a health/social-care worker (p=0.002; n=15/45.5% of cases). >
Regarding outcomes, cases showed a significant improvement in MRS scores (p<0.001), mobility (p><0.001), and ADLs (p=0.049). Degree of dependence for ADLs reduced by 50% in cases, while the number wheelchair/bed-bound reduced by 70%. Regression analysis could not identify statistically significant predictors of response to inpatient treatment in conversion patients. Having co-morbid non-epileptic dissociative features predicted an increased length of stay in hospital (p=0.04). >
Conclusion: Even patients with severe, long-standing motor conversion disorder can benefit from an inpatient admission to a specialist neuropsychiatry unit. Our study suggests links between motor conversion disorder, physical/sexual abuse, and prior employment as a health/social-care professional. Previous studies have had similar problems identifying predictors of outcomes in conversion disorder. This study was limited by its retrospective design and observational nature. It is therefore not possible to state what elements of the treatment package brought about improvement. Generalisability is also limited by the inclusion of a particularly morbid subset of motor conversion cases.

13. Title: Serial drug usage for tics in Tourette syndrome - When to give up?

Authors: Mena Farag, Jeremy S. Stern, David Williams, Kathryn Grabecki, Helen Simmons, Mary M. Robertson
Department of Neurology, Atkinson Morley's Wing, St. George's Hospital, Blackshaw Road London, SW17 0QQ

Objective: Clinicians recognise that pharmacological treatment with any given drug for tics is variable in efficacy between patients. The level of evidence-based medicine for agents in use is sometimes low and generally does not demonstrate long-term effectiveness. It is common to serially try reasonable options as seems appropriate. The success of this strategy has not been previously examined.
Method: 272 sets of notes of children and adults with Tourette Syndrome seen in a specialist clinic were retrospectively reviewed in terms of their drug histories and outcome at last outpatient review. Continuing prescription of the last tried drug was used as proxy evidence of ongoing beneficial effect, as opposed to those patients no longer taking medication.
Results: 172 patients had been prescribed drugs for tics either previously by other clinicians or under our supervision. The most commonly used drugs tried over the whole history of the patients were aripiprazole, clonidine, sulpiride, risperidone and the "older" option haloperidol (some patients had first been treated over 20 years ago). Numbers of different drugs tried ranged from only 1 so far (77 patients) to a series of 8 (1 patient). The proportion of patients still being followed up on their latest treatment or having been discharged still on the treatment varied from 69% for 1 drug only to 80-90% for 2nd to 4th treatment choice and was 100% for the single patient who had reached an 8th option. This patient had a particularly high Yale Global Tic Severity Score (YGTSS) at first assessment at our clinic, but there was no significant correlation between YGTSS and number of different drugs tried for the other patients.
Conclusion: Using only this proxy assessment of the success of using serial drugs for tics in TS ie. Without any objective or prospective measure, it appears that where high numbers of successive agents are used it is still possible for the final option selected to be successful, at least in the short or medium term. The more relevant observation for many patients is that usage of several drugs over the course of medical supervision is not uncommon, illustrating the long-term unreliability of drugs including those showing success in clinical trials.

Members' Posters

14. Title: Epilepsy in Tourette syndrome

Authors: David Williams, Jeremy S. Stern, Kathryn Grabecki, Helen Simmons, Mary M. Robertson
Department of Neurology, Atkinson Morley's Wing, St. George's Hospital, Blackshaw Road
London, SW17 0QQ, United Kingdom

Objective: Tourette Syndrome (TS) is a neurodevelopmental disorder frequently associated with comorbidities such as OCD, ADHD and autistic spectrum disorders (ASD). Tics are more common in Learning Difficulty (LD) populations. The mechanism of these associations is felt to vary for instance appearing to be more genetically based for OCD than for ADHD. The comorbid conditions seen with TS are known to be associated with increased or high rates of epilepsy. In turn, epilepsy cohorts also have high rates of neurodevelopmental and behavioural disorders. There has been little literature on epilepsy in TS.

Method: Clinical records of 347 patients with TS seen at a specialist clinic were reviewed. Associated conditions were diagnosed clinically but it was not possible to stratify LD by IQ. Epilepsy diagnoses were rated as definite or probable by a neurologist taking into account previous investigations including EEG where available, clinical descriptions and treatment. Cases where epilepsy had been inappropriately suspected or misdiagnosed were excluded.

Results: The cohort was 23% female and 50% under the age of 17 with the following comorbidities: OCD (24%), ADHD (54%), LD (10%) and ASD (10%). Epilepsy was seen in 21 cases (6%) and was felt to be definite in half of these cases. Mean age of seizure onset was 7 years and was within a year of onset of tics in 33% of the epilepsy cases. In 4-6 cases the seizures were felt to be symptomatic, in 6 were focal and in 9 had remitted. Cases with epilepsy were not more severe on Yale Global Tic Severity Scores but had more comorbidity. There was an earlier age of onset of tics and significantly higher rates of ADHD, OCD and LD with a non-significant trend for an increased rate of ASD. Looking at the figures from the other direction, patients in the TS cohort with LD and OCD had significantly increased rates of epilepsy (18.2% v. 4.8% for LD) and there were non-significant trends for ADHD and ASD.

Conclusion: Patients with Tourette syndrome have a higher than expected rate of epilepsy, and are also sometimes misdiagnosed with seizures. Rates are higher still in patients with various comorbidities, especially LD, and uncommon in "pure" TS. Seizures could be a marker for a more severe neurodevelopment syndrome, or could reflect a shared substrate. Thalamocortical dopaminergic dysfunction has been linked to seizures and there could be contributions from epileptogenesis effects of neurodevelopmental genes or drug treatment for tics.

15. Title: A Questionnaire Study Investigating Relationships Between Physical and Mental Health, Repressive Coping and Ageing

Authors: S Khodatars, J Erskine, 59 Graveney Road, Tooting, London, SW17 0EG, United Kingdom

Objective: This study aims to examine the differences in physical and mental health as a result of normal ageing. Repressive coping is a mechanism of the human mind where an individual automatically and unknowingly will avoid negative information generated both internally and received externally. Previous research indicates that repressive coping may increase with age whilst having a detrimental effect on physical health. Therefore, the prevalence of repressive coping by age, the relationship of physical and mental health with age, and the relationship between physical health and repression are key aspects that will be explored.

Method: Several different existing psychological questionnaires measuring anxiety, depression and defensiveness will be used. Each questionnaire examines physical or mental health and repressive coping status.
The study is cross sectional, and participants will be separated into three categories according to their age.

Results:

1. There was a higher proportion of repressive copers amongst older participants
2. As an individual ages, their physical health declines as the number of physical health problems increases.
3. Anxiety decreases with age
4. Repressors are more likely to have two or more physical health problems compared to non-repressors.
5. Depression neither increased or decreased with age.

Conclusion: Results have shown that the prevalence of repressive copers did increase with age, whilst physical health declined and mental health seemed to improve. Repressive copers also seem to have more physical health problems compared to non-repressive copers. This may be due to a cohort effect or developmental progression but would require longitudinal investigation.

Members' Posters

16. Title: The development of a new apathy measurement scale: Dimensional Apathy Scale

Authors: Mr Ratko Radakovic and Dr Sharon Abrahams
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The University of Edinburgh, 7 George Square, EH8 9JZ, United Kingdom

Objective: To develop a new questionnaire, the Dimensional Apathy Scale (DAS) which aims to be sensitive to Levy and Dubois' 3 subtypes of apathy, Auto-Activation, Emotional-affective and Cognitive.
To develop a scale suitable for patients with motor disorders in which apathy may be prevalent.
To explore the relationship of the DAS with depression.

Method: Study design and item development: Twelve published single dimension Apathy scales were reviewed. Item selection was based on the three subtypes of apathy. 45 suitable items were designed. A balanced 4 point Likert scale was used with item rating based on the frequency of occurrence in the last month associated with how they felt, behaved or thought.
Procedure: Study A (N = 261) was an online 45 item questionnaire using Limesurvey, a free and open source survey software tool.
Study B (N = 50) was a paper and pencil version of the 45 item questionnaire accompanied by completion of Becks Depression Inventory 2.

Results:
Stage 1 Analysis:
Horn's parallel analysis of principal factors of all 311 participants highly factorable data indicated 4 factors to be extracted. Exploratory factor analysis produced 4 factors accounting for 28.9% of the total variance. The factors were labelled Executive (Ex), Emotional (Em), Cognitive Initiation (CI) and Behavioural Initiation (BI) with the final two factors combined Behavioural Cognitive Initiation (BCI) apathetic factor. The most meaningful and relevant 8 items were extracted from each factor to make 3 subscales (Ex, Em and BCI) to construct the new 24 item DAS.
Stage 2 Analysis:
Internal consistency reliability of the extracted DAS items was high, Cronbach's standardized 1± = .798. A 50 participant subsample showed moderate correlations between depression and the three different apathy subscale total scores. Both the Ex and Em subscales correlated significantly with depression scores (Ex r = .553, p<.001, Em r = .365, p><.01) and BCI being least significant (r = .354, p><.05).

Conclusion: The question analyses revealed four factors, which have been combined to make 3 dimensions of Executive, Emotional and Behaviour/Cognitive Initiation of the DAS. The subscale items show good internal consistency reliability. The BCI subscale was most associated with depression levels. Future studies will apply the DAS to different disease profile with the aim of distinguishing between the different apathy subtypes associated with each.

17. Title: Neural Correlates of De novo Depression Following Left Temporal Lobe Epilepsy Surgery: A Voxel Based Morphometry Study of Pre-surgical Structural MRI

Authors: Cleary RA, Centano M, Flugel D, Symms M, Thompson PJ, Koepp M, Foong J.
The National Hospital for Neurology & Neurosurgery, Department of Neuropsychiatry

Objective: To investigate cerebral grey matter (GM) abnormalities in temporal lobe epilepsy (TLE) patients who develop de novo depression following TLE surgery using voxel-based morphometry (VBM).

Method: We retrospectively examined the pre-surgical grey matter (GM) abnormalities in 45 patients with TLE due to unilateral left-sided hippocampal sclerosis using a 1.5 T MRI scanner, which were segmented with optimised VBM parameters using SPM8 software. Grey matter maps were normalised to a sample template using DARTEL. Voxel-wise GM differences between patients that developed de novo post-surgical depression (n=6) were compared with patients with no pre- or postoperative psychiatric diagnoses (n=25), using independent samples t-tests. Analysis of covariance with age and gender as covariates was adopted for the VBM statistics; the level of statistical significance was set at p<.001, uncorrected. >

Results: Reduced preoperative GM in both the ipsilateral thalamic and orbitofrontal cortices (OFC) were significantly associated with the development of de novo depression within 4 years postoperatively. Further analyses revealed that GM atrophy of these structures was unrelated to a history or frequency of secondary generalised tonic-clonic seizures (SGTCS). We observed no differences in seizure freedom (ILAE 1 vs 2-6) or seizure recurrence (ILAE 2 vs 3-6) between the groups.

Conclusion: Although the development of postoperative de novo depression following TLE surgery is likely to be multifactorial, our results suggest that ipsilateral thalamic and OFC atrophy in LTLE patients may play a modulatory role. Structural and functional abnormalities in these areas have also been implicated in primary mood disorders (1). Prospective studies with larger cohorts utilising in vivo imaging techniques are warranted to replicate these results, and further elucidate the neural correlates of de novo postoperative mood disorders.

Additional Information:
1.Drevets WC. Neuroimaging and neuropathological studies of depression: implications for the cognitive-emotional features of mood disorders. Curr Opin Neurobiol. 2001:240-249

Members' Posters

18. Title: Cognitive impairments in ALS relate to white matter integrity in specific regions of interest
Authors: Pettit L. D., Bastin, M., & Abrahams, S. **,** Psychology Department PPLS, 7 George Sq, University of Edinburgh
Objective: Cognitive impairment in amyotrophic lateral sclerosis is characterized primarily by deficits in executive functioning, and structural and functional imaging studies have revealed changes in extra-motor areas which are consistent with a continuum of multi-system involvement. More recently investigations into white matter in ALS have demonstrated reduced volume and integrity in multiple structures, with a preponderance for frontotemporal involvement. However, the contribution of white matter pathology to cognitive impairment in ALS remains largely unknown. The current study sought to investigate the relationship between white matter integrity and cognitive impairments in tasks assessing executive functioning, memory, and processing speed.
Method: The current investigation employed a cross-sectional design comparing 30 ALS patients to 30 age and education matched healthy controls. Several experimental tests were specifically developed to be sensitive to executive dysfunction and processing speed deficits in populations with motor impairments. In addition, diffusion tensor magnetic resonance imaging was employed to investigate the neural correlates of any observed cognitve impairments
Results: ALS patients showed impairments in verbal fluency and dual task measures which correlated with reduced white matter integrity in specific and discrete regions of the prefrontal lobes. Verbal fluency performance was associated with integrity reductions in the white matter adjacent to Broaca's area and Brodmann's Area 10. Dual task performance was associated with reduced integrity of the frontal white matter and white matter adjacent to dorsolateral prefrontal cortex. In addition, ALS patients showed impairments in a working memory task which correlated with reduced integrity in the posterior cingulum, and in verbal memory which correlated with reduced integrity in the inferior longitudinal fasciculus.
Conclusion: The current study demonstrates that specific white matter pathology may underpin impairments in specific cognitive functions and further highlights the heterogeneity that exists in this disorder.

19. Title: Association of apathy with frontal lobe dysfunction in amnestic mild cognitive impairment and Alzheimer's disease
Authors: Dr Manoj George, Tim Whitfield, Zuzanna Walker
Objective: The primary aim of this study is to examine an association between apathy and frontal lobe dysfunction in patients with memory problems. We also aimed to look into the association between apathy and praxis.
Method: This was a retrospective cross sectional study. We selected 160 consecutive patients diagnosed with Alzheimer's dementia and Amnestic Mild Cognitive Impairment who had a comprehensive battery of neuropsychological tests and a behaviour rating scale of interest for this study recorded in the database. Correlation between apathy with and without depression were tested against frontal lobe test including Trail making A, Trial making B, Letter Fluency, Ideational Fluency, Category fluency, Abstract Thinking and Executive functioning subtest of CAMGOG-R.
Results: Statistically significant relationship were found between apathy and executive function scores, ideational fluency scores, abstract thinking and category fluency scores.
Conclusion: Apathy is negatively related to executive function and ideational fluency. This finding has important clinical significance because poor scores in executive function influence memory abilities by preventing people to employ compensatory strategies that can help them remember information and maintain functional abilities. It is also associated with greater neuropsychiatric disturbances especially a greater degree of agitated and disinhibited behaviour.

20. Title: Association between apathy and the caregiver burden in amnestic mild cognitive impairment and Alzheimer's disease
Authors: Dr Manoj George
Three quarter of care for Alzheimer's patient is provided by family and friends and they often report adverse experience which might be physical, psychological, or financial in nature. These adverse consequences as a result of the care provided are known as caregiver burden (Kaufer et al., 2005)
Objective: The main objective of this study is to look into the association between apathy severity and the caregiver burden and also identify significant predictors of apathy.
Method: This was a retrospective cross sectional study. We selected 160 consecutive patients diagnosed with Alzheimer's dementia and Amnestic Mild Cognitive Impairment who had a comprehensive battery of neuropsychological tests and a behaviour rating scale of interest for this study recorded in the database.
Nonparametric Spearman correlational analyses examined relationships between apathy and caregiver burden scores. Further correlational analyses was carried out after removing patients with depression (HADS depression scores > or = to 8) .Forward stepwise linear multiple regressions identified significant predictors of apathy.
Results: In our study, series of exploratory Spearman rho correlation confirmed the significant relationship between apathy and caregiver burden with and without depression. We did further sub analysis and found apathy to be significantly related to caregiver burden in both AD and aMCI patients
Conclusion: This study has shown high association of apathy with caregiver burden both in AD & aMCI. It is clear from the current literature that apathy is a common symptom in both AD & aMCI and has significantly adverse consequence to both the patients and the caregivers. Actively screening, monitoring and managing apathy in clinical practise would improve the prognosis and quality of life of these patients.