BNPA British Neuropsychiatry Association

Programme and Abstract Book BNPA Annual Conference

7th to 8th March 2019
Kings Place, York Way, London

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Journal of NEUROLOGY, NEUROSURGERY & PSYCHIATRY

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dis-sociated
CLEA MARTIN VARGAS PRESENTS 'DIS-SOCIATED'
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DIRECTORS OF PHOTOGRAPHY LEON BREHONY & JOHN LETSINGER
DIRECTED, PRODUCED & EDITED BY CLEA MARTIN VARGAS

Welcome to the 32nd Annual meeting of the British Neuropsychiatry Association

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Day 1 – 7th March 2019
The neuropsychiatry of epilepsy, clinical research updates, global neuropsychiatry

08:15 – Registration		
The neuropsychiatry of Epilepsy - Chair: Marco Mula		
09:00	The neurological, psychiatric and theological significance of mystical seizures	Alasdair Coles
09:35	Epilepsy in people with an intellectual disability- managing the neuropsychiatric challenges	Mike Kerr
10:10	The comorbidity between epilepsy and dissociative seizures	Markus Reuber
10:45 – Refreshments		
Clinical research updates - Chair: Niels Detert		
11:15	Psychological approaches to management in Parkinson's disease	Richard Brown
11:45	Deep Brain Stimulation for OCD: an update	Eileen Joyce
Lunch 12:15 **12:45 – Poster presentation walkaround** ***(poster presenters please stand by your poster)***		
Global neuropsychiatry – Chairs: Chris Butler and Anette Schrag		
13:15	Mental Imagery and Mental Health Science: From Lab to Hospital to a Refugee Camp?	Emily Holmes
13:50	How neuroscience can inform public policy	Facundo Manes
14:25	Culture Ethnic Minorities and Health	Dinesh Bhugra
15:00	Poverty and Brain Development: From Science to Policy	Martha Farah
15:35 - Refreshments		
Guest Speaker – Chair: Thomas Cope		
16:00	Inflamed depression	Ed Bullmore
17:00 – Ed Bullmore Book signing '*The Inflamed Mind: A radical new approach to depression*' **/Close** **19:00-22:00 – BNPA Evening Reception**		

Day 2 – 8th March 2019

Functional Neurological Disorder and it's boundaries, members' platform, BNPA medal lecture, BNPA interactive panel discussion

08:30 – Registration		
FND - Chair: Tim Nicholson		
09:30	The bladder and the brain	Ingrid Hoeritzauer
10:05	Feigned or functional?	Mark Edwards
10:40	Hypermobility and autonomic dysfunction: insights from bench to bedside	Jessica Eccles
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Members' Platform x 3 - Chair: David Okai		
11:45	**Motor functional neurological disorder (mFND) in a large UK mental health service: Clinical characteristics, medication prescription and response to outpatient cognitive behavioural therapy** Nicola O'Connell, Department of Psychological Medicine, King's College, London	
12:00	**Effect of Methylphenidate on risk preference in Attention Deficit Hyperactivity Disorder** Alekhya Mandali, Department of Psychiatry, University of Cambridge	
12:15	**State and Trait Interoception is Disrupted in Functional Seizures.** M Yogarajah, Institute of Molecular & Clinical Sciences, Neurosciences Research Centre, St George's University of London, & Atkinson Morley Regional Neuroscience Centre, St George's Hospital, London	
Lunch	**12:30-13:15 – BNPA AGM** **13:30 – Poster presentation walkaround** ***(poster presenters please stand by your poster)***	
Medal Lecture – Chair: Chris Butler		
14:10	The language disorder in semantic dementia: does it matter which language you speak?	Karalyn Patterson
BNPA interactive panel discussion Chairs: Camilla Nord and Boyd Ghosh		
15:00	Pros and cons of cognitive enhancers	Martha Farah Barbara Sahakian Hazem Zohny
16:00 – Close		

The neurological, psychiatric and theological significance of mystical seizures

Alasdair Coles, Consultant Neurologist, Cambridge University Hospitals

Alasdair Coles is an academic neurologist in Cambridge, mainly working on experimental therapies in multiple sclerosis. He is also an Anglican priest. Funded by the Templeton Foundation, he has led two studies of the effect of neurological diseases on spirituality: Parkinson's disease and temporal lobe epilepsy. He has edited "Neurology and Religion", to be published by CUP in 2019.

Abstract

Since Dostoyevsky's seminal descriptions of mystical seizures in The Idiot, based on his own epilepsy, there has been considerable lay interest in the significance of these seizures. But, they have been little studied. We have conducted a prevalence study of seizures with spiritual content at epilepsy centres in UCL and Cambridge and asked whether their phenomenology is homogeneous and comparable with mystical experiences outside of neurological disease. We have explored their meaning for the individual affected and their families.

Epilepsy in people with an intellectual disability- managing the neuropsychiatric challenges

Mike Kerr, Cardiff University, Department of Psychological Medicine and Clinical Neurosciences

Professor Mike Kerr studied medicine in Bristol (UK), General Practice in York and Psychiatry in Cardiff. His clinical practice is in the epilepsies associated with intellectual disability and in the assessment and treatment of epilepsy and psychiatric disorder. He has been closely associated with initiatives in improving the public health of people with an intellectual disability including developing the Cardiff Health Check, which is used across England and Wales. Academically he has published widely on healthcare, epilepsy and intellectual disability and held research grants from many funding bodies. He is vice chairperson of SUDEP ACTION. He is chair of the International League Against Epilepsy (ILAE) commission on psychiatric aspects of epilepsy. He has been appointed as an Ambassador for Epilepsy by the ILAE and International Bureau for Epilepsy.

Abstract

There is a close relationship between epilepsy and people with intellectual disability, with an estimated 22% prevalence of epilepsy. The epilepsy is also known to be more severe with an increase in likelihood of chronicity and early mortality. From a neurological point of view the group presents challenges in diagnosis, investigation and treatment choice. Especially as so many individuals have childhood severe epilepsies, with seizure freedom a challenging goal. A specific management issue is that the population presents a range of neuropsychiatric challenges in terms of diagnosis, behavioral co-morbidity, and the presence of psychiatric comorbidity. These conditions significantly hinder the ability to manage patients and intensify the impact of epilepsy on the individuals and carers. To some degree this would appear no different to the challenges posed within the epilepsy population without intellectual disability. However, issues of communication need, varied aetiology of the intellectual disability and care provision intensify the complexity of neuropsychiatric presentation.

In the era of personalized and precision medicine people with an intellectual disability are now at the forefront of medical practice. With individual aetiology, in particular, driving assessment and intervention. This paradigm can be applied to neuropsychiatric disorders in people with an intellectual disability, expanding our knowledge of the conditions and improving our clinical formulations and interventions. In this presentation I will discuss the nature of neuropsychiatric presentations in people with an intellectual disability, its aetiology and how clinicians may manage these conditions within the setting of epilepsy care.

The comorbidity between epilepsy and dissociative seizures

Markus Reuber MD PhD FRCP
Professor of Clinical Neurology, University of Sheffield, Honorary Consultant Neurologist, Sheffield Teaching Hospitals NHS Foundation Trust, Editor-in-Chief, Seizure European Journal of Epilepsy

Professor Reuber joined the Univ of Sheffield in 2005. He also works at the Royal Hallamshire Hospital and the Chesterfield Royal Hospital as an Honorary Consultant Neurologist. His clinical work focuses on the treatment of patients with epilepsy and other seizure disorders. He leads the Epilepsy Research Group in the Academic Neurology Unit and is the Epilepsy Research Theme Lead in the Clinical Neuroscience Directorate at Sheffield Teaching Hospitals. He is chair of the Neurosciences Local Priority Group of the Comprehensive Local Research Network and represents South Yorkshire on the National Speciality Group for Nervous System Disorders. He completed his undergraduate medical education in Cologne, Germany, and Nottingham. He pursued neurological training in Leeds, and at the Department of Epileptology of the University of Bonn, Germany.

Abstract

Dissociative seizures are experiential and behavioural responses to internal or external triggers. They superficially resemble epileptic seizures in terms of their subjective and objective manifestations. Although they are theoretically distinct from epileptic seizures in which seizure manifestations are related to hypersynchronous and excessive discharges in the brain, there is a complex relationship between epileptic and dissociative seizures, which extends beyond the (clinically extremely important) problem of misdiagnosis. While epilepsy is more closely associated with clinically detectable structural or biochemical abnormalities than dissociative seizures, the sort of experiential factors which are widely recognized as predisposing individuals to the development of dissociative seizures (such as trauma and neglect in early life) also increase the risk of developing epilepsy. The similar (albeit not identical) developmental background of patients with epilepsy on the one hand and dissociative seizures on the other is reflected by similar (but not identical) psychiatric comorbidity profiles. More specifically, epileptic seizures are a risk factor for the development of dissociative seizures, may precipitate or trigger dissociative seizures, and comorbid epilepsy may contribute to the perpetuation of dissociative seizures disorders. My talk will provide some food for thought about two seizure disorders often associated with high levels of disability which are theoretically different but which - in practice - have much in common.

Key references :

Kutlubaev MA et al. Dual diagnosis of epilepsy and psychogenic nonepileptic seizures: Systematic review and meta-analysis of frequency, correlates, and outcomes. Epilepsy and Behav 2018, 89:70-78.
Brown RJ, Reuber M. Psychological and psychiatric aspects of psychogenic non-epileptic seizures (PNES): A systematic review. Clin Psychol Rev 2016;45:157-182.
Brown RJ, Reuber M. Towards an integrative theory of psychogenic non-epileptic seizures (PNES). Clin Psychol Rev 2016;47:55-70.
Reuber M & Brown RJ. Understanding psychogenic nonepileptic seizures-Phenomenology, semiology and the Integrative Cognitive Model. Seizure 2017;44:199-205.
Baroni G et al. Variables associated with co-existing epileptic and psychogenic nonepileptic seizures: a systematic review. Seizure 2016;37:35-40.

Psychological therapies in Parkinson's disease

Professor Richard Brown, Professor of Neuropsychology and Clinical Neuroscience, Head of Department of Psychology, King's College London, Institute of Psychiatry

Richard Brown is Professor of Neuropsychology and Clinical Neuroscience and Head of Department of Psychology, at the Institute of Psychiatry, Psychology and Neuroscience at King's College London. He is a faculty member of National Institute of Health Research Maudsley Biomedical Research Centre. He has worked extensively in the field of clinical neuroscience and his primary interest is the field of movement disorders and particularly Parkinson's disease. He undertook his PhD with Professor David Marsden and worked for 10 year at the MRC Human Movement and Balance Unit, Queen's Square. He has published over 200 scientific works and his research has involved laboratory-based and clinical research and spanned a range of movement, cognitive, emotional and social aspects of brain disorder. He has been involved in the development and evaluation of novel psychological approaches to management of non-motor symptoms in patients with Parkinson's disease including depression, anxiety and impulse control disorders. He is also an Honorary Consultant Clinical Psychologist with the South London and Maudsley NHS Foundation Trust, Neuropsychiatry Service, where he supports the psychological management of patients with acquired brain injury and neurodegenerative disorders, with a particular focus on Parkinson's disease.

Abstract

Non-motor symptoms (NMS) are ubiquitous in Parkinson's disease and are increasingly seen as core features of the disease. Often predating the onset of motor symptoms, they typically increase in number and severity as the disease progresses, increasing disability and overall disease burden. The high prevalence of psychiatric disturbance in PD and its impact on outcome has been recognised for many years, with increasing recognition of the emotional distress and social impact associated with other disabling motor and non-motor symptoms.

While medical treatments are available to ameliorate many NMS, management remains problematic. Psychological approaches are increasingly common in the field of long-term conditions; for symptom management as well as tackling wider psychological issues such as adjustment and relationship function.

However, they continue to play a relatively minor role (or are inaccessible) in routine PD care. This presentation will summarise some of the main areas where psychological approaches have demonstrated or potential value. Depression, and more recently anxiety, has been the subject of study for many years and supplies most of the existing (if limited) empirical evidence for psychological approaches. The strengths and limitations of that research will be considered to suggest how we might best meet the existing needs of patients using existing treatments, particularly CBT.

Even though evidence is limited or lacking, the talk will consider the potential for psychological approaches to contribute to the management of neurocognitive symptoms including impulse control behaviour and apathy, as well as other disabling and distressing problems such as sleep disturbance, fatigue, pain and motor fluctuations.

The presentation will conclude by considering some of the new or emerging treatment approaches offering alternative to CBT that may have advantages in terms of efficiency, accessibility and acceptability.

Deep Brain Stimulation for OCD: an update

Eileen Joyce, Professor of Neuropsychiatry at The Institute of Neurology and Honorary Consultant Neuropsychiatrist at the National Hospital for Neurology and Neurosurgery

Professor Joyce's 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

Deep brain stimulation (DBS) is an emerging treatment for people with severe OCD who have not responded to SSRI medication, prescribed either at high doses or augmented with additional medication, and several courses of CBT including inpatient treatment. Since it was first described in 1999, there have been 5 randomised controlled trials comparing sham and active DBS at two brain targets: the ventral capsule/ventral striatum site (VC/VS) and the anteromedial subthalamic nucleus (amSTN). A study directly comparing DBS at both sites in the same 6 patients with very severe OCD found that they have equivalent efficacy for reducing OCD symptoms*. Neuroimaging evidence suggested that DBS at each site modulates different brain circuits and has different behavioural actions. VC/VS DBS was much more effective than amSTN DBS at improving mood and affected a circuit connecting the medial orbitofrontal cortex to dorsal thalamus, amygdala and habenula nucleus. amSTN DBS improved cognitive flexibility, whereas VC/VS DBS did not, and affected a circuit connecting the amSTN with lateral orbitofrontal cortex, dorsolateral prefrontal cortex and dorsal anterior cingulate cortex. Both circuits have different functions and are known to be abnormal in OCD. The implications of these findings for the future management of severe OCD will be discussed.

* *A Randomised Trial Directly Comparing Ventral Capsule and Anteromedial Subthalamic Nucleus Stimulation in Obsessive Compulsive Disorder: Clinical and Imaging Evidence for Dissociable Effects. Himanshu Tyagi, Annemieke M. Apergis-Schoute, Harith Akram, Tom Foltynie, Patricia Limousin, Lynne M. Drummond, Naomi A. Fineberg, Keith Matthews, Marjan Jahanshahi, Trevor W. Robbins, Barbara J. Sahakian, Ludvic Zrinzo, Marwan Hariz, and Eileen M. Joyce, Biological Psychiatry, in press.*

Mental Imagery and Mental Health Science: From Lab to Hospital to a Refugee Camp?

Professor Emily A. Holmes, Uppsala University, Sweden

Emily Holmes, PhD, DClinPsych is a Professor in Psychology at the Department of Psychology, Uppsala University, Sweden. She is also affiliated to the Karolinska Institutet's Department of Clinical Neuroscience, and is a Visiting Professor of Clinical Psychology at the Department of Psychiatry, University of Oxford, UK. Holmes received her degree in Experimental Psychology at the University of Oxford, UK. She is also a clinician and completed a clinical psychology training doctorate at Royal Holloway University of London, and a PhD in Cognitive Neuroscience in Cambridge, UK. Holmes' work as a clinical psychologist has fuelled her research questions. She is interested in psychological treatment innovation in mental health – both in creating new techniques and reaching more people. Under the wider umbrella of mental health science, her approach brings together psychology, neuroscience, psychiatry, maths and more. Holmes' research has demonstrated that mental imagery has a more powerful impact on emotion than its verbal counterpart. Her group is particularly interested in understanding and reducing intrusive memories after trauma. This is relevant for people after a traumatic event, whether a severe motor vehicle accident, traumatic childbirth or war. She is an Associate Editor for Behaviour Research and Therapy. She is on the Board of Trustees of the research charity "MQ; transforming mental health".

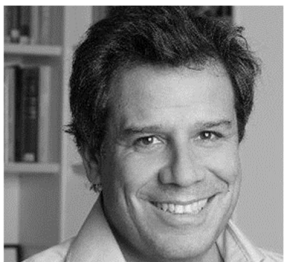
Abstract

Traumatic events can affect anyone from a road traffic accident, to traumatic childbirth or war-related trauma for refugees. A core clinical symptom for many people who experience trauma comprises intrusive memories to the event. Intrusive memories that "flash backwards" to past trauma occur in post-traumatic stress disorder (PTSD). They take the form of mental imagery, that is, an experience like perception in the absence of a percept, such as "seeing in our mind's eye". Indeed, intrusive, affect-laden mental images can cause distress across mental disorders. Mental images allow us to time travel, and can also "flash forwards" to the future such as those can occur related to suicide or in bipolar disorder.

My clinical research group has an interest in understanding and treating maladaptive mental imagery via psychological therapies. To do this, we are curious about what we can learn from cognitive psychology and neuroscience to inform novel treatment development. I will discuss recent work concerning intrusive memory encoding, and a mechanistically informed intervention aiming to disrupt memory re-consolidation via dual task interference, thereby to reduce the frequency of intrusive memories. Recent work on tackling mood instability via focused imagery techniques will be discussed. A broader vision for science-informed psychological treatment innovation will also be explored from working hospitals (Iyadurai et al, 2018, Molecular Psychiatry) to further afield with refugees (Holmes, Ghaderi et al, 2018, Lancet Psychiatry).

How neuroscience can inform public policy

Facundo Manes , Professor of Behavioural Neurology and Cognitive Neuroscience INECO Foundation for Research in Cognitive Neuroscience and Institute of Neuroscience - Favaloro University. Argentina



Facundo Manes is an Argentinian neuroscientist. He was born in 1969, and spent his childhood and adolescence in Salto, Buenos Aires Province. He studied at the Faculty of Medicine, University of Buenos Aires, where he graduated in 1992, and then at the University of Cambridge, England (Master in Sciences). After completing his postgraduate training abroad (USA and England) he returned to the country with the firm commitment to develop local resources to improve clinical standards and research in cognitive neuroscience and neuropsychiatry. He created and currently directs INECO (Institute of Cognitive Neurology) and the Institute of Neurosciences, Favaloro Foundation in Buenos Aires City. Both institutions are world leaders in original scientific publications in cognitive neuroscience. He is also President of the World Federation of Neurology Research Group on Aphasia and Cognitive Disorders (RGACD) and of the Latin American Division of the Society for Social Neuroscience. Facundo Manes has taught at the University of Buenos Aires and the Universidad Católica Argentina. He is currently Professor of Neurology and Cognitive Neuroscience, Faculty of Medicine and Psychology of the Favaloro University and was appointed Professor of Experimental Psychology at the University of South Carolina, USA.He has published over 100 scientific papers in the most prestigious original specialised international journals such as Brain and Nature Neuroscience. He has also given lectures at several international scientific fora as the “Royal Society of Medicine” (London) and the “New York Academy of Sciences”, among others. His current area of research is the neurobiology of mental processes. He believes in the importance of scientific disclosure for Society. He led the program ” The Brain Enigmas ” on Argentina TV and wrote many scientific articles in the national press. Finally, Prof. Facundo Manes is convinced that the wealth of a country is measured by the value of human capital , education, science and technology, and that there is the basis for social development.

Abstract

In the last two decades, mainly in the developed world, we have seen a remarkable shift in how policies are made. Until the late 1990's, the ranks of lead policymakers comprised of economists, lawyers, and financial experts. Behavioral scientists are now increasingly being asked to bring their insights and expertise from the laboratory into the "real" world. As a result, many experts in behavioral sciences are playing a much greater role in policymaking across a range of sectors. We have seen the positive results of applying this scientific knowledge in diverse areas of public policy such as taxation, energy, and education, among others. The insights used to design these interventions were evidence-based cognitive and psycho-social discoveries such as the existence of cognitive biases and the importance of social norms in human behavior.

Behavioral units needed to show significant results for a minimum or zero cost in order to prove their worth and thus, cement the incorporation of behavioral sciences into the public sphere. It is time to move on to a renewed level of complexity by designing more comprehensive interventions with deeper implications and long-term results.
The role of neurosciences has been much less explored but shows a promising potential to improve public policy, especially social policy. For instance, the design of anti-poverty programs based on scientific evidence about brain health would be extremely useful to improve the mental capital of persons living in poverty. Findings in neurosciences along the life cycle can be incorporated into the public policy sphere. Understanding the neuroscientific evidence on how to stimulate cognitive and socio-emotional skills among different types of populations is crucial to improve the design of policies and has relevant implications for the mental capital of nations.

Neurosciences can also facilitate the comprehension of those cognitive skills needed for the jobs of the future such as creativity, emotional intelligence, empathy, resilience, cognitive flexibility, and executive functions, among others. Public policy interventions can focus on promoting these skills, which are not fixed and which can be improved and enhanced.
Other areas in which evidence from neurosciences can be utilized are: infant development, the adolescent brain, addictions (i.e., opioid epidemic, science and policy of marijuana), neuroeducation or the neuroscientific basis in teaching and learning (i.e., dyslexia), neurolaw (i.e. criminal responsibility, deception, juvenile justice, judges' decision making, witness testimony), prejudice and empathy, violence and aggression, loneliness, normal aging, the future of mind-tech cyborgs, and the burden of brain disorders.

Behavioral insights are gaining popularity in developed countries. However, is this enough to create positive structural changes in the economy and in social welfare? While it is fair to say that nudges successfully allowed behavioral science to enter the world of policy we still have much work to do to reach social and health long-term improvements. In this regard, advances in neurosciences over the last years could help reshape the way we think about important policies and could be useful in helping individuals develop their maximum potential.

Culture Ethnic Minorities and Health

Professor Dinesh Bhugra CBE Emeritus Professor of Mental Health and Cultural Diversity at the Institute of Psychiatry, Psychology and Neuroscience at King's College London.

Professor Dinesh Bhugra was President of the World Psychiatric Association from 2014-2017 and in 2018 became President of the British Medical Association. Dinesh Bhugra's research interests are in cultural psychiatry, sexual dysfunction and service development. He is the recipient of over 10 honorary degrees. He has authored/co-authored over 400 scientific papers and 30 books, and is the Editor of three journals (*International Journal of Social Psychiatry, International Review of Psychiatry* and *International Journal of Culture and Mental Health)*. Previously he was the Dean (2003-2008) and then President (2008-2011) of the Royal College of Psychiatrists in the UK, where he led on major policy initiatives on psychiatry's contract with society and the role of the psychiatrist.

Abstract

Culture form us and we form culture. Culture influences the way we think and the way we see the world. Furthermore, recognition and presentation of distress as well as patterns of help seeking are very strongly influenced by cultures and our world views. The accessibility and availability of healthcare system also influences when and where we seek help from. Cultures include religion, spirituality, language, diet, dress etc. In many cultures there are no words to express many psychiatric and neurological disorders even though symptoms of distress are easily recognised. In addition, depending upon the explanatory models of distress patients and their families may seek help from non-professional sources. Studies from the USA have suggested that a vast majority of people seek help from personal, folk or social sectors and only a minority reach the professional health sector. This may reflect the American healthcare system. Gender and other factors such as education, social class, housing, employment and economic factors contribute to help-seeking. Cultural identity and embedded within are micro-identities which help formulate an unique cultural framework which differentiates one group from another and can work at both individual and group levels. Using various models this lecture will explore varying presentations and using examples from around the world will illustrate cultural impact on various neurological and psychiatric disorders. Community support in various forms can enable quick recovery and help support vulnerable groups.

Poverty and Brain Development: From Science to Policy

Martha J. Farah, Director, Center for Neuroscience & Society
Walter H. Annenberg Professor in the Natural Sciences
University of Pennsylvania

Martha Farah was born in New York City and educated at MIT and Harvard. She has taught at Carnegie-Mellon University and the University of Pennsylvania, where she is currently the Walter H. Annenberg Professor of Natural Sciences. In 1999 she founded Penn's Center for Cognitive Neuroscience and ten years later she founded the Center for Neuroscience & Society, which she still directs. Martha's work on the ethical, legal and social impact of neuroscience (aka neuroethics) has focused on cognitive enhancement by normal individuals, including the question of whether drugs and devices believed to enhance normal cognition actually do so, and on nonclinical uses of brain imaging. Her other main interest is in the effects of early socioeconomic deprivation on brain development. She studies the latter using behavioral, neuroendocrine and neuroimaging methods. Martha is a Fellow of the American Academy of Arts and Sciences, a former Guggenheim Fellow and recipient of honors including the National Academy of Science's Troland Research Award and the Association for Psychological Science's lifetime achievement award.

Abstract

Childhood socioeconomic status (SES) predicts many important life outcomes, from physical and mental health to academic achievement and cognitive ability. Why is SES so influential? Part of the answer lies in the relationship between SES and the brain. I will present research from my lab and others aimed at characterizing SES differences in brain structure and function. I will then discuss the causes of these associations – by what mechanisms are they linked? – and their consequences – what roles do they play in the health and achievement disparities mentioned earlier? Finally, I will consider whether and how the neuroscience of SES can help shape policies concerning children of low SES.

Inflamed Depression

Professor Edward Bullmore, Department of Psychiatry, University of Cambridge R&D, GlaxoSmithKline

Edward Bullmore, FRCP, FRCPsych, FMedSci began his medical career as an academic rather than a physician. From 1987 to 1988, he was a lecturer in medicine at the University of Hong Kong. He then returned to England where he began training in his chosen specialisation as a Senior House Officer in psychiatry at St George's Hospital, London. After a year, he moved hospitals, and was appointed a Registrar in psychiatry at Bethlem Royal Hospital and Maudsley Hospital ; both specialist psychiatric hospitals in London. In 1993, Bullmore began his research career. That year, he was appointed a Wellcome Trust Research Training Fellow and served in that role for three years. During that time he studied for a Doctor of Philosophy (PhD) degree at King's College London, which he completed in 1997 with a thesis titled " Analysis of structural and functional magnetic resonance images of the brain ". In 1996, he was promoted to an Advanced Research Training Fellow for a further three years. His research during this time focused on the mathematical analysis of neurophysiological time series. From 1996 to 1999, he was additionally an honorary Consultant Psychiatrist at Maudsley Hospital, London. In 1999, Bullmore joined the University of Cambridge as Professor of Psychiatry. At college level, he was an elected Fellow of Wolfson College, Cambridge between 2002 and 2010. On 9 October 2014, he was appointed Head of the Department of Psychiatry, University of Cambridge. In 2005, he joined GlaxoSmithKline as Vice-President of Experimental Medicine. From 2005 to 2013, he was also Head of its Clinical Unit based in Addenbrooke's Hospital, Cambridge, Cambridgeshire, which focuses on early clinical drug development projects.

Abstract

It is beyond doubt that inflammation and depression are associated with each other. Many patients with arthritis, or other inflammatory disorders of the body, have increased risk of depression; many patients with depression have increased blood levels of inflammatory proteins like cytokines. I will discuss how this association has traditionally been interpreted (or discounted) in the Cartesian framework of medical science; and I will highlight new evidence for a direct mechanistic relationship, whereby inflammation of the body or brain can cause depressive behaviours. This raises a number of further questions. First, can known risk factors for depression – like social stress – cause inflammation? Second, what is the current evidence that anti-inflammatory interventions can have anti-depressant effects and how could new, more personalized approaches to treatment of "inflamed depression" (not all depression) be developed in future?

Bullmore ET (2018) "*The Inflamed Mind*" Short Books, London.

British Neuropsychiatry Association 32nd Annual Meeting 7-8 March 2019

Delegate Feedback Form – DAY 1

Your opinions are important to us and will help us plan future conferences. Please complete the below form and return to the Registration Desk.

How useful did you find this event? Extremely useful [ ] Useful [ ] Fairly useful [ ] Not useful [ ]

What was your overall impression of this event? (1 = Excellent, 2 = Good, 3 = Fairly Good, 4 = Poor, 5 = Very Poor)	1	2	3	4	5
Programme					
Registration process					
Organisation					
Venue					
Catering					

DAY 1: NEUROPSYCHIATRY OF EPILEPSY (1 = Excellent, 2 = Good, 3 = Fairly Good, 4 = Poor, 5 = Very Poor)	1	2	3	4	5
The neurological, psychiatric and theological significance of mystical seizures					
Epilepsy in people with an intellectual disability- managing the Neuropsychiatric challenges					
The comorbidity between Epilepsy and NEAD					
CLINICAL RESEARCH UPDATES	1	2	3	4	5
Psychological Approaches to management of Parkinson's disease					
Deep brain stimulation for OCD: an update					
GLOBAL NEUROPSYCHIATRY	1	2	3	4	5
Mental imagery and mental health science: from lab to hospital to a refugee camp?					
How neuroscience can influence public policy					
Culture ethnic minorities and health					
Poverty and brain development: from science to policy					
GUEST SPEAKER	1	2	3	4	5
Inflamed depression					

What impact will this event have on your future practice?

Comments/Suggestions and ideas for future conferences:

THANK YOU!

Day 2 – 8th March 2019

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16:00 – Close		

The Bladder and The Brain: Exploring Functional Urological Symptoms

Ingrid Hoeritzauer
Clinical Research Fellow, University of Edinburgh

Ingrid Hoeritzauer is a neurologist who also has a degree in psychology. She works with the Functional Research Team led by Jon Stone and Alan Carson in Edinburgh and received an ABN/Patrick Berthoud Clinical Research Training Fellowship. Her research interests are observational studies in functional neurological disorders and cauda equina syndrome and her current research focuses on the interface between Uro-Neurology and functional neurological disorders.

Abstract

This talk will review the current understanding of the bladder-brain axis, and the evidence linking functional disorders, including Functional Neurological Disorder (FND), to bladder symptoms including urinary retention and in the study of patients who present with cauda equina syndrome but have normal scans. Uro-Neurology is the connection between urological symptoms and the neurological system, comprising a complex bladder-brain network involving the brain, spinal cord, sacral nerves and peripheral (pelvic) nerves. Since the time of Charcot there have been hypotheses that urological symptoms, particularly idiopathic urinary retention and overactive bladder symptoms, were part of a functional disorder. However, in the 1980s urethral EMG changes in women with idiopathic urinary retention led to a view that so called 'psychogenic' urinary retention was a primary disorder of the urethral sphincter. Changing views of FND in the last 10 years mean that urological symptoms are once again being investigated as a potential part of functional disorders. Patients presenting with chronic idiopathic urinary retention, diagnosed as Fowler's syndrome, or presenting with acute urinary retention and possible cauda equina syndrome, have been found to have high levels of functional disorder and FND comorbidity[1,2]. Additionally, recent papers have suggested central sensitisation as the mechanism of action in patients with idiopathic overactive bladder symptoms[3]. This newly re-energised field of study requires further exploration, such as investigation of the effect of pathomechanical and medication induced urological symptoms on patients with functional disorders. A practical guide to how to discuss likely functional urological symptoms based on the current level of knowledge will conclude the talk.

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Feigned or functional?

Professor Mark Edwards, Eleanor Peel Chair for the Study of Ageing, Professor of Neurology and Honorary Consultant Neurologist, Atkinson Morley Regional Neuroscience Centre

Mark Edwards is Professor of Neurology at St George's University of London and Consultant Neurologist at the Atkinson Morley Regional Neuroscience Centre at St Georges University Hospital. He has a specialist clinical and research interest in Movement Disorders and in neurophysiological and psychophysical methods for exploring their pathophysiology. He did his PhD at the UCL Institute of Neurology with Professor John Rothwell and Professor Kailash Bhatia and was then a Senior Lecturer and Honorary Consultant Neurologist at UCL and the National Hospital for Neurology and Neurosurgery. Here he built up a specific interest in functional neurological symptoms and developed an NIHR funded research program and specialist clinical diagnostic and treatment service for patients with functional movement disorders. At St George's he is part of an integrated diagnostic and treatment service for functional neurological disorder, and continues also with research and clinical work in movement disorders in general.

Abstract

"Poor Hysterics…first they were treated as victims of sexual trouble…then of moral perversity and mediocrity…then of imagination". Over a century since William James wrote these words, the status of people with functional neurological disorder remains uncertain and ambiguous. The language of everyday medical discourse betrays this ambiguity: "Are they real seizures?…Does he have genuine weakness?…The good news is that there's nothing serious wrong…" In this talk I will explore to what extent clinical and experimental work can help address this issue. While we may not be able to resolve the question of "feigned or functional?" – perhaps because it is unanswerable in this form – exploring it may make us more aware of our own biases, hidden or not, and the way they affect our interaction and care for people with functional neurological disorder.

Hypermobility and autonomic dysfunction: insights from bench to bedside

Jessica Eccles, Honorary Consultant in Liaison Psychiatry
Sussex Partnership NHS Foundation Trust and Brighton and Sussex University Hospitals NHS Trust

Dr Jessica Eccles trained in medicine at University of Cambridge and University of Oxford, completing a BA in The History and Philosophy of Science, sparking a keen interest in philosophy of mind and brain-body interactions. Since graduation from medical school has pursued a combined academic clinical path at Brighton and Sussex Medical School. As an MRC Clinical Research Training Fellow she recently completed her PhD in the relationship between joint hypermobility, autonomic dysfunction and psychiatric symptoms and is now an NIHR Clinical Lecturer. She is currently working on an Academy of Medical Sciences grant to explore neural connectivity in hypermobility using leading edge Human Connectome Project techniques and has recently been awarded a MQ Arthritis Research UK Fellows Award to conduct a randomised clinical trial of a new targeted treatment for anxiety in hypermobility. Dr Eccles has also been awarded a grant from Dysautonomia International and will be working with Profs Critchley, Cercignani, Rowe, Murphy and Drs Nagai, Asslanni, Iodice and Giovanni to explore multi-modal correlates of 'brain fog' in Postural Tachycardia Syndrome. Dr Eccles is working with Profs Davies, Harrison, Cercignani, Critchley and Dr Tarzi to explore brain- body interactions in Fibromyalgia and ME/CFS. This involves autonomics, inflammatory and cytokine markers, brain imaging and genomics. This work is funded by Versus Arthritis and Action for ME Alongside clinical academic colleagues at BSMS, Prof Harrison and Dr Colasanti.

Abstract

Joint hypermobility is a common, yet poorly recognised variant of connective tissue affecting up to 20% of the population. Hypermobility is a cardinal feature of Hyermobility Spectrum Disorder (HSD) and hypermobile Ehlers Danlos Syndrome (hEDS), inherited disorders of connective tissue. Individuals with joint hypermobility are over represented in panic, anxiety and neurodevelopmental populations and are prone to dysautonomia, typically postural tachycardia syndrome (PoTS), in which there is a phenomenological overlap with anxiety disorder. Interestingly differences in brain structure and function have been described in hypermobility in regions associated with emotional processing, including amygdala and insula. Individuals with joint hypermobility are more likely to experience severe chronic widespread pain, and many have co-morbid rheumatic conditions. A data-driven theoretical model linking joint hypermobility to psychiatric disorder is proposed, characterised by aberrant autonomic control and central representation, grounded in current theoretical models that seek to frame emotion as interoceptive inference, using leading-edge predictive coding approaches. Ultimately this approach has considerable relevance to personalised psychiatric medicine in this disorder and greater understanding of brain-body mechanisms underpinning neuropsychiatric states.

Motor functional neurological disorder (mFND) in a large UK mental health service: Clinical characteristics, medication prescription and response to outpatient cognitive behavioural therapy

Nicola O'Connell, Timothy Nicholson, Simon Wessely, Anthony S. David
Dr Nicola O'Connell, Research Fellow, Institute of Population Health, Trinity College Dublin

Objective Studies on motor functional neurological disorder (mFND) often originate in neurology settings and are characterised by low sample sizes, and lack control groups. There are few prescription guidelines and no gold standard treatments. This study aims to establish mFND patients' socio-demographic and clinical characteristics, medication prescription patterns and patients' responses to outpatient cognitive behavioural therapy (CBT).
Methods This is a retrospective case-control study of mFND patients in contact with secondary mental health services in South London and Maudsley (SLaM) NHS Foundation Trust between 2006 and 2016. Data were obtained from anonymous electronic health records using the 'Clinical Records Interactive Search' (CRIS) database. Data were extracted on socio-demographic, clinical and medication variables. Control patients were a random sample of contemporaneous psychiatric patients treated within the same Trust and were matched at a ratio of 1:2. In a separate study, we employed these methods to identify mFND patients who attended an outpatient neuropsychiatry CBT clinic in SLaM, comparing therapeutic outcomes in mFND to patients with organic neuropsychiatric disorders (ONP) treated in the same clinic.
Results Our search returned 322 mFND and 644 control patients. Weakness was the most common functional symptom. mFND patients were more likely to be female, British, married, employed pre-morbidly, to have a carer and a physical health condition, but less likely to have had an inpatient psychiatric admission or to receive benefits. There was no difference in rates of childhood sexual and physical abuse between groups. A lower proportion of mFND patients received medication compared to controls (76.6% v. 83.4%, $p < 0.05$), but of medication recipients, mFND patients were prescribed a higher number and variety of agents. We identified 98 mFND and 76 ONP patients attending the outpatient CBT service. Both groups showed significant improvements in psychological functioning post-CBT (measured with the CORE-OM, HoNOS-ABI, and PHQ-9), with physical symptoms improving in 49.4% of mFND patients. A logistic regression found acceptance of psychological formulations prior to CBT ($p < 0.02$) was associated with improvement in physical functioning in mFND patients.
Conclusions mFND patients have a distinct socio-demographic profile and are prescribed a heterogeneous array of psychotropic and somatic medications. mFND patients treated in a specialist CBT clinic show similar improvements in psychological functioning to patients with organic neuropsychiatric disorders. This study establishes the socio-demographic profile of this under-studied patient group and could help guide the development of future therapeutic interventions and inform the design of a pilot RCT.

Effect of Methylphenidate on risk preference in Attention Deficit Hyperactivity Disorder

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Introduction Methylphenidate (MPH) is one of most commonly prescribed drug to patients with Attention Deficit Hyperactivity Disorder (ADHD). While MPH has been known to improve executive functions, its effect on impulsivity, one of the cardinal symptoms in ADHD has mixed findings in part depending on baseline. Data driven computational models such as drift diffusion model utilize behavioural measures to explain subtle changes that are not sensitive to traditional analysis. Here, we aim to analyse risk preference in ADHD and healthy controls and the effects of MPH.
Methods Twenty-four healthy volunteers and 25 ADHD patients were tested on the 2 step sequential learning task in both MPH-ON and MPH-OFF conditions. We calculated the risk associated with each choice (variance of reward probability) and defined the choice with maximum variance as the risky one, for all 134 trials. With behavioural measures (selected choice- risky vs non-risky and response time) as inputs and risk as an independent factor, we extracted threshold (a), drift rate (v) and response bias (z) parameters using a hierarchical drift diffusion model (HDDM) for both groups during ON and OFF drug condition. Statistical analysis on the parameters was analysed using Bayesian factors.
Results Bayesian repeated measures ANOVA showed evidence for changes in response bias (z) but not in threshold and drift rate. A strong evidence for main effect of drug(BF_{10} =6.03x10^{11}), group(BF_{10}=86344) and group by drug interaction(BF_{10}=3.65x10^{6}) was observed. Post-hoc Bayesian independent sample t-tests showed strong evidence that the patient group had a higher preference towards the risky choice during both the ON (BF_{10}=8.94x10^{14}) and OFF (BF_{10}=20.9) conditions. Post-hoc Bayesian paired sample t-tests showed strong evidence for the drug to induce a preference towards the risky choice in both the HV(BF_{10}=397.1) and ADHD(BF_{10}=1.16x10^{10}) population. Behavioural results show a drug by group interaction (F(1,0.01)=11.80, p=0.001) on number of risky choices. Post-hoc analysis using paired sample t-test showed a significant increase in risky behaviour due to drug in the ADHD(t(24)= -3.5, p<0.005) but not healthy subjects. No differences were found in the traditional reinforcement learning parameters between the groups.
Conclusions Using a novel analysis, we showed that ADHD subjects had a greater bias towards risk preference and further that MPH increases risk preference in both ADHD and HV with a comparatively greater effect on the patient population. Critically we observe an effect on response bias highlighting the role of *apriori* information in influencing risky decision making.

State and Trait Interoception is Disrupted in Functional Seizures.

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Objectives The continuity and integrity of a conscious sense of self, is proposed to be dependent upon the control of internal physiological state and its predictive representation through interoception, that is, the sensing of internal bodily changes. We investigated dissociation, interoception and their relationship, in patients with functional seizures (FS), before and after a stressor intervention.

Methods 41 participants with functional seizures (FS) and 30 age/gender matched healthy controls (HC) were assessed with the somatoform dissociation questionnaire (SDQ20), multi-scale dissociation inventory (MDI), and the state and trait anxiety inventory (STAI). Standardized measures of interoceptive sensibility, accuracy, and awareness were acquired with the Porges Body Perception Questionnaire (PBPQ), and heartbeat discrimination (HDT), tracking (HTT) and time-tracking tasks (TTT), before and after a cold pressor test. Continuous non-invasive blood pressure monitoring was carried out before, during and after the cold pressor test. Interoceptive trait (ITPE) and state (ISPE) prediction errors, that is, the discrepancy between interoceptive accuracy and the PBPQ (trait), and trial-by-trial confidence estimates (state), were calculated before and after the cold pressor test respectively, for HTT and HDT. An autonomic prediction error (APE), or the discrepancy between the reported increase in pain and the change in blood pressure after the cold pressor, was also calculated.

Results Patients with FS differ significantly from HC for HTT, ITPE and ISPE suggesting that they are overall less interoceptively accurate and aware than HC. This is confirmed by a correlation between APE and the ISPE derived from the HDT task ($r=0.359$, $p=0.033$) in FS subjects only, after correcting for state anxiety and duration of cold pressor. Furthermore, in FS patients only, ITPE scores, adjusted for trait anxiety, correlated with SDQ-20 and MDI-depersonalization scores for both HTT ($r=0.378$, $p=0.008$; $r=0.408$,$p=0.005$) and HDT ($r=0.364$, $p=0.011$; $r=0.281$, $p=0.044$). All results survived FDR correction at a 0.05 threshold.

Conclusions These findings demonstrate that state and trait interoception are disrupted in patients with FS. The severity of the disruption in trait interoception correlates with measures of dissociation, such that the bigger the ITPE, the more severe are the dissociative traits. Similarly, the greater the ISPE, the larger the discrepancy between subjective symptoms and objective physiological changes, after a stressor intervention. Our findings suggest that the selective disruption of interoceptive processing is both a potential predisposing and precipitating factor in FS.

The language disorder in semantic dementia: does it matter which language you speak?

Karalyn Patterson, University of Cambridge

From its inception, Karalyn Patterson has shaped the field of cognitive neuropsychology - the study of how the brain's structure and function relates to mental processes concerning the generation and use of knowledge. Specifically, Karalyn has used the effects of brain disease and injury to improve our understanding of language and memory. Her approaches are varied and rigorous, combining computer modelling and structural and functional brain imaging with observations of behaviour in normal and brain-damaged adults, as well as those affected by certain brain diseases. This has allowed Karalyn to directly link particular structures in the brain with specific cognitive issues. Karalyn's research has important consequences for our understanding of brain conditions that affect memory; for example, Alzheimer's disease and other forms of dementia. She has also revealed the impact of the same brain disorder on the speakers of two diverse languages, English and Japanese.

Abstract

Semantic dementia (SD) is a neurodegenerative condition in the spectrum of frontotemporal dementia, and considered to be one of the main varieties of primary progressive aphasia. The question in the title of this talk will be addressed from two different perspectives. The first asks whether the *pattern* of language features observed in SD varies in any principled and significant way across different languages. English and Japanese, for example, differ in almost every component of language – phonology, syntax, written form, etc; yet the profiles of language deficit in SD patients from these two language communities are virtually identical. From this perspective, therefore, the answer is no, it does not matter which language you speak. The second question asks whether the *severity* of the language disorder in SD varies in a principled and significant way across the two languages spoken by bilingual cases of SD. A high proportion of people living in India speak two or more languages. When bilingual Indian SD patients are given the same tests in their L1 and L2 languages, of course they are impaired in both, but they show a striking advantage for L1. Furthermore, and of substantial theoretical interest, the patients' correct responses to test items in L2 are a virtually perfect subset of correct responses to the same test items in L1. From this perspective, therefore, the answer is yes, it does matter which language you are speaking. These contrasting answers to the two forms of the question follow from the following pair of hypotheses: (a) the language disorder in SD is a fairly pure reflection of a disintegrating semantic system, and (b) the semantic system is fundamentally language-independent.

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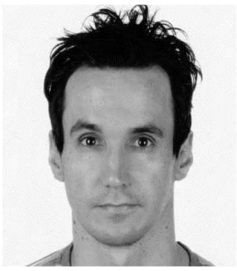
Martha J. Farah, PhD is a cognitive neuroscientist who works on problems at the interface of neuroscience and society. These include: the effects of childhood poverty on brain development; the expanding use of neuropsychiatric medications by healthy people for brain enhancement; novel uses of brain imaging, in e.g. legal, diagnostic and educational contexts; the many ways in which neuroscience is changing the way we think of ourselves as physical, mental, moral and spiritual beings.

Barbara J Sahakian is Professor of Clinical Neuropsychology at the University of Cambridge Department of Psychiatry and MRC/Wellcome Trust Behavioural and Clinical Neuroscience Institute. She is also an Honorary Clinical Psychologist at Addenbrooke's Hospital, Cambridge. She holds a PhD and a DSc from the University of Cambridge. She is President of the International Neuroethics Society, Past-President of the British Association for Psychopharmacology and a Fellow of the Academy of Medical Sciences. She is co-author of 'Bad Moves: How decision making goes wrong and the ethics of smart drugs' (OUP, 2013) and co-editor of The Oxford Handbook of Neuroethics (OUP, 2011). She is a member of ACNP, CINP Council and ECNP Review Board and a member of the Human Brain Project. Sahakian has an international reputation in the fields of psychopharmacology, neuropsychology, neuropsychiatry, neuroimaging and neuroethics.



Dr Hazem Zohny is a Research Fellow in Bioethics and Bioprediction at the Oxford Uehiro Centre for Practical Ethics. His current work focuses on the bioprediction of behaviour, particularly the use of neurointerventions for crime prevention. He has a PhD in Bioethics from the University of Otago, where he worked on ethical and conceptual issues related to human enhancement. His research interests also include moral responsibility, well-being, and global justice.

The BNPA Interactive Panel Discussion - Pros and Cons and Cognitive enhancers
Panel: Martha Farah, Hazem Zohny, Barbara Sahakian
Chairs: Camilla Nord and Boyd Ghosh

Have you ever wondered what it would be like to supercharge your brain? Three experts, Barbara Sahakian (Cambridge) and Martha Farah (Pennsylvania) and Hazem Zohny (Oxford), will discuss cognitive enhancers, how they work, who they work for and the ethics behind their use.

You the delegates, will have the opportunity answer the following questions before and after using a mobile phone app and we'll see if the audience feels differently about issues surrounding these drugs after the talks.

Before the panel discussion ONLY:
1) Do you feel that you are currently functioning at your optimum level?
2) Do you have healthy friends or colleagues who take cognitive enhancing drugs, or have you taken them yourself?

Before *and* after the panel discussion:
3) Should medical practitioners be able to prescribe cognitive enhancers to people without neurological or psychiatric disorders (or brain injury) who want to boost their abilities (e.g. at work or in a hobby)?
4) If these cognitive enhancing drugs or 'smart drugs' were legal for healthy people to use, would you use them?
5) Do you think there is a clear and useful line that distinguishes "treatments" from "enhancements" for cognition?
6) To what degree to you think the social or physical benefits of cognitive enhancers outweigh the risk?

British Neuropsychiatry Association 32nd Annual Meeting 7-8 March 2019

Delegate Feedback Form – DAY 2

Your opinions are important to us and will help us plan future conferences. Please complete the below form and return to the Registration Desk.

How useful did you find this event? Extremely useful [ ] Useful [ ] Fairly useful [ ] Not useful [ ]

What was your overall impression of this event? (1 = Excellent, 2 = Good, 3 = Fairly Good, 4 = Poor, 5 = Very Poor)	1	2	3	4	5
Programme					
Registration process					
Organisation					
Venue					
Catering					

DAY 2: FND (1 = Excellent, 2 = Good, 3 = Fairly Good, 4 = Poor, 5 = Very Poor)	1	2	3	4	5
The bladder and the brain					
Feigned or functional?					
Hypermobility and autonomic dysfunction: insights from bench to bedside					
MEMBERS PLATFORMS	1	2	3	4	5
Characteristics of patients with motor functional neurological disorder in a large UK mental health service: a case control study					
Effects of Methylphenidate on risk preference in Attention Deficit Hyperactivity Disorder					
State and Trait Interoception is Disrupted in Functional Seizures					
MEDAL LECTURE	1	2	3	4	5
The Language disorder in semantic dementia: does it matter which language you speak?					
BNPA INTERACTIVE PANEL DISCUSSION	1	2	3	4	5
Pros and cons of cognitive enhancers					

What impact will this event have on your future practice?

Comments/Suggestions and ideas for future conferences:

THANK YOU!

1. High levels of anxiety and depression in patients attending with headaches to a UK general neurology clinic

Thomas Cronin, Ronald Pearce

Objective: The published literature on headache epidemiology comes from specialist headache clinics, compared to the general neurology clinic. This study set out to investigate the characteristics and diagnoses of patients with headaches attending a general neurology clinic in the UK.
Methods: Data were collected retrospectively from a two-year period on 217 patients with headaches referred to a general neurology clinic at a UK district-general hospital seen by a single consultant. Clinic letters were reviewed, and information was inputted using a pre-formed Microsoft Excel spreadsheet. All data were anonymised, with no identifiable patient characteristics being recorded.
Results: A total of 217 were seen in this period. The mean age was 42 and 72% were female. In 56% of cases, more than one diagnosis was made. The most frequent diagnosis was migraine (72%), followed by tension-type headache (56%). Dizziness and/or vertigo was a commonly reported co-existing symptom, with 24% reporting this. For 13% (n=29) of cases, it was documented that time was taken out of work or studies due to headache symptoms. In 205 patients it was reported whether sleep was affected, with 70% (n=144) of cases indicating it was. For 195 cases, 80% (n=155) reported a normal appetite, 16% (n=32) a decrease, and 4% (n=8) an increase in appetite. In 195 patients, 65% (n=126) reported reduced energy levels. For 21% (n=46) there was documentation of anxiety. Regarding mood, in 176 cases where this was recorded, 33% gave a negative mood description.
Conclusion: To the authors' knowledge, this study is the first to report on headache characteristics in patients presenting to a UK general neurology clinic. The diagnostic frequency of different headaches presented in this study are comparable to those described in specialist headache clinics. Our population demonstrate significant psychiatric morbidity associated with headaches, with 33% reporting negative mood value and in addition higher proportions reporting energy and sleep disturbance. To conclude, this study has shown the common headache diagnoses encountered in a general neurology clinic, and indeed is comparable to headaches managed in primary care. Improved integration between these services is key to ensuring effective care for such patients.

2. "Nobody prepared me for this!" Parents' experiences of seeking help and support with post-brain injury symptoms and changes in children and adolescents with Acquired Brain Injury.

Dr Robyn MCCarron

Objectives/aims This research aims to:

-Understand the journeys and processes that parents follow in seeking help and support for post-brain injury symptoms and changes in their children.
-Identify the barriers and challenges that parents face in obtaining help and support for their children and family following paediatric Acquired Brain Injury (ABI).
-Propose ways in which services can be more responsive to the evolving needs of children and adolescents with ABI and their families.
Method: Five mothers were interviewed using semi-structured interviews about their experiences of seeking help and support for post-brain injury symptoms and changes in their children. The participants' children were 60% male, 10-16 years old and were 2-6 years post injury. Four had suffered strokes and one had a Traumatic Brain Injury. The age at injury was 4-14 years old. The interviews were audio-recorded and transcribed into 2698 units of data. The data was analysed using a Glaserian grounded theory constant comparative approach.
Results: After a child suffers an ABI parents feel that it is down to them to ensure that their child gets the help and support that they need. Identifying and accessing support requires a high level of research and persistence that parents experience as a "battle". Obtaining support for mental-health problems is especially challenging. Common barriers include communication issues, time and funding delays, a lack of consistent professionals, not meeting the criteria for services, and a lack of local or age-appropriate services. The challenges of trying to seek help and support impact upon the whole family in terms of time, finances, socially and emotionally. However, through this process parents do become expert parents/carers. They gain new knowledge and language and they look to share this to support other parents going through similar experiences.
Conclusions: Providing parents with accessible psychoeducation around post-brain injury symptoms and changes in the acute period following a paediatric ABI could be a helpful intervention to make parents feel more prepared and supported in the medium to long term. More specialist paediatric ABI services that can provide integrated, holistic and developmental care are needed. Current services may need a more flexible and longer-term approach to be responsive to the needs of young people with ABI.

3. Clinical Characteristics of Pathological Confirmed Early Onset Dementia with Lewy Bodies

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Background Early-onset dementia (EOD) is characterized by distinct clinical profiles and prognosis when compared to late-onset dementia (LOD). As the second most common neurodegenerative form of dementia, little is known about the clinical profile of early-onset Dementia with Lewy Bodies (DLB). A current challenge for clinicians when managing patients with DLB is the suboptimal diagnosis rate which will affect treatment efficacy and outcome. To address this knowledge gap, by hypothesizing early-onset DLB will have a distinct profile when compared to Alzheimer's disease (AD), we accessed and reviewed data of patients with pathological confirmed DLB from National Alzheimer's Coordinating Center (NACC) database.

Methods Patients with first visit that fulfill criteria for dementia of AD or DLB were analyzed. Early onset age was defined as less than 65 years old. Variables included in the analyses include baseline demographics, cognitive, behavioral, motor symptoms, neuropsychological battery scores and clinician diagnosis. Comparisons were made between early-onset AD (EOAD) versus early-onset DLB (EODLB), and early versus late-onset DLB.

Results This study included 363 patients with EOAD, 32 EODLB and 147 late-onset DLB. Patients with EODLB were more likely to present with psychosis, apathy, REM sleep behavioral disorder, and motor symptoms. While EOAD patients were more likely to present with cognitive symptoms as first recognized and predominant presentation and perform worse in memory assessment. Motor as first recognized presentation, slowness, visual hallucination, caregiver reporting of agitation and apathy were the significant predictors to differentiate the two. Late-onset DLB patients were less depressed and more impaired in memory and executive function related scores than EODLB. Significant number of EODLB patients were misdiagnosed as EOAD (46.9%, $p<0.0001$).

Conclusions EODLB is characterized by motor and neuropsychiatric symptoms while neuropsychological tests appear less reliable to differentiate EODLB from EOAD. Given that misdiagnosis of DLB remain significantly high, we propose a more careful and comprehensive clinical approach may improve the diagnosis rate.

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4. Understanding Foreign Accent Syndrome

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Objectives/aims Foreign accent syndrome (FAS) is a disorder of speech in which listeners perceive the affected individual as speaking with a foreign or different regional accent that is not their habitual accent. FAS is widely understood as an unusual consequence of stroke or other lesions within speech-motor networks. However, case reports of FAS occurring in the absence of structural damage and difficulty identifying neural correlates has led to increasing recognition that that FAS sometimes represents a functional neurological disorder. We aimed to characterise symptoms, comorbidities, and features of recorded speech in individuals with self-reported FAS.
Methods Participants self-reporting FAS recruited from informal unmoderated online support forums and recruited via professional networks completed an online survey. Recorded samples of spontaneous speech and reading of a standardised text were analysed in a subgroup of 13 cases.
Results Forty-nine respondents (24 UK, 23 North America, 2 Australia) reported FAS of mean duration three years (range two months – 18 years). Common triggers were: migraine / severe headache (15), stroke (12), surgery or injury to mouth or face (six), and seizure (five, including three non-epileptic). High levels of comorbidity included migraine (33), irritable bowel syndrome (17), functional neurological disorder (12), and chronic pain (12). Five reported structural lesions on imaging. Author consensus on aetiology divided into, 'probably functional' (n=35, 71%), 'possibly structural' (n=4, 8%), and 'probably structural'(n=10, 20%), but positive features of functional FAS were present in all groups. Blinded analysis of speech recordings supplied by 13 respondents correctly categorised 11 (85%) based on probable aetiology (functional vs structural) in agreement with case history assignment. Analysis of speech recordings identified a range of features demonstrating internal inconsistency with potential utility in the diagnosis of functional FAS.
Conclusions This study, of the largest FAS case series to date, details the experience and characteristics of individuals with self-reported FAS, and describes an approach to

auditory-perceptual analysis of speech with potential diagnostic utility. Although conclusions are limited by the recruitment methods, high levels of functional comorbidity, symptom variability and additional linguistic and behavioural features suggest that chronic FAS may in many cases represent a functional neurological disorder, even when a structural lesion is present

5. Missed diagnosis of ADHD in children referred to a Tic Disorder Clinic

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Aim To examine whether Attention Deficit Hyperactive Disorder (ADHD), a common comorbid disorder in Tourette's Syndrome (TS) patients, is often missed by physicians referring to a Tic Disorder Clinic.

Method Referral letters and first clinic attendance reports for 119 new patients aged between 4-17 that attended a national tic disorder clinic between 2015-2017 were analysed to see how many new diagnoses of ADHD were made at first consultation that were not included in the referral letters. Other variables that were noted for each patient included age, sex, if referrer had a suspicion of ADHD (rather than established or firm diagnosis), medication for ADHD and the main treatment target decided at the tic disorder clinic.

Results Out of 119 patients 13 (11%) already had a diagnosis of ADHD, which is in line with the prevalence of comorbid ADHD in the general population but not with the known increased prevalence in patients with TS (up to 80% in some studies). The assessment at the Tic Disorder Clinic found 46 cases of ADHD (38%). Referrals were from pediatricians (51%), general practitioners (35%) and from mental health services (10%).

Conclusions As the prevalence of comorbid ADHD is high in Tourette's patients and this can sometimes be obscured by the presentation of the tic disorder, referrers should have a low threshold for suspecting and managing ADHD in cases where specialist input for tics is awaited. It is likely that CAMHS referrals were under-represented in the sample and it may be expected that prior ADHD diagnoses would be more likely from that source.

6. Epidemiology of Catatonia in a Large Dataset

Jonathan Rogers, Thomas Pollak, Nazifa Begum, Anna Griffin, Rashmi Patel, Megan Stewart, Graham Blackman, Timothy Nicholson, Anthony David

Objectives / Aims Catatonia is an important neuropsychiatric disorder with a high morbidity and mortality. However, due to a perception that it is very infrequent and because of the acuity of the patients, it has remained poorly studied and research has often been confined to small groups. We aimed to establish the demographic, disease-related variables and blood-based biomarkers for catatonia in a large dataset.
Methods We used the Clinical Records Interactive Search (CRIS) system hosted at the NIHR Maudsley Biomedical Research Centre to search the clinical records for patients with catatonia. An initial free-text search was refined by use of a natural language processing app. The results of the app were validated by three of the authors, who included patients in the analysis only if a clinician had made a diagnosis of catatonia and two or more items of the Bush-Francis Catatonia Screening Instrument were in evidence. Demographics, disease-related variables and blood-based biomarkers could then be extracted for these patients and compared, where relevant, to non-catatonic psychiatric patients.
Results The natural language processing app extracted the records of 2766 patients with at least one mention of catatonia in their records. The majority of cases identified by the app could be validated by the researchers. A high proportion of patients had more than one episode of catatonia.
Full results will be available in time for the presentation.
Conclusions This study demonstrates that catatonia is not very rare, even relying on clinician identification. The frequency of recurrence is interesting, as it suggests that catatonia might indicate an underlying trait, rather than merely a transient state.

7. Relationship between interoception and stress in patients with Functional Neurological Symptom Disorder

Dr. Isobel Anne Williams, Prof. Markus Reuber, and Dr. Liat Levita

Objectives /aims Self-report studies of alexithymic traits in individuals with Functional Neurological Symptom Disorder (FND), suggest that emotion dysregulation in this population is characterised by an impaired ability to detect and identify their own emotions (identification impairments) (1). This regulatory deficit might be particularly problematic for a patient group with an increased incidence of stressful life events relative to healthy controls (2), for whom the ability to regulate emotions might therefore be more crucial. Examining sensitivity to changes in physiological cues associated with emotional experience (interoception) is a way of assessing one aspect of participants' capacity to identify their own emotions. However, no studies have yet experimentally investigated how stress might interact with interoception in this population. Therefore, the aim of this study was to investigate patients' interoceptive sensitivity both at baseline and under stress.
Methods Twenty-six patients with FND and twenty-seven healthy controls performed the Heartbeat Detection Task (HBDT) pre- and post- stress-induction with the Cold Pressor Test. The HBDT is a behavioural paradigm, measuring participants' sensitivity to a physiological cue associated with emotional experience - the heartbeat. Participants also completed a self-report measure of emotion dysregulation (The Emotional

Processing Scale-25) which includes a subscale capturing 'a detached experience of one's emotions due to poor emotional insight', and a measure of Major Depressive symptomology (The PHQ-9).
Results Relative to healthy controls, patients with FND performed more poorly on the HBDT both at baseline and following stress-induction (p = .032*).* Patients also reported greater impairments across all domains of the EPS-25 and higher scores on the PHQ-9 than healthy controls (both p <.001). Group differences on HBDT performance were not explained by group differences in age or depressive symptomology.
Conclusions Impaired HBDT performance suggests that patients with FND lack sensitivity to their heartbeat, both under 'normal' conditions and following stress-induction. Physiological cues (like the heartbeat) are an important source of interoceptive information for emotional experience, for example during stress. Our findings therefore represent a form of identification impairment that may contribute to stress-vulnerability in this population. Raised levels of self- reported 'impoverished emotional experience' corroborate the suggestion that patients with FND have difficulty identifying and understanding their emotions. These findings have direct implications for understanding and treating emotion dysregulation in FND.
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8. On being autoimmune in psychiatric places: 10 characteristic mental state features in patients with definite NMDAR-antibody encephalitis

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Objectives/aims NMDAR-antibody encephalitis frequently presents with psychiatric symptoms. However, new-onset mental illness does not usually receive detailed biomedical investigations. Yet, early diagnosis and treatment correlates with improved outcomes. Here we used detailed psychiatric phenotyping to explore the nature of mental state abnormalities in this immunologically-defined illness.
Methods Prospective and retrospective semi-structured interviews with patients, carers, and clinicians in five consecutive cases of definite NMDAR-antibody encephalitis (all female, median age=20 years, range=16-30, ovarian teratoma in 4). Weekly multi-disciplinary assessment using the Neuropsychiatric Inventory Nursing Home version

(NPI-NH) in 2/5. Network analysis was used to evaluate connectedness of psychopathologic features and a qualitative synthesis distilled recurrent psychopathologic features. Finally, each time point was compared with operationalised diagnoses using an automated classifier and plotted with corresponding symptom complexes over time.

Results All had psychiatric features at onset and were seen first by general practitioners or emergency departments. All received an incorrect initial diagnosis (1 neurological, 4 primary psychiatric). Two patients were referred to mental health services and detained while three were admitted to a general hospital. Psychiatric diagnoses spanned psychotic, mood, and stress categories. None had a personal or family history of serious mental illness or substance misuse. Despite the atypicality all were ascribed to non-specific psycho-social aetiologies. Autoimmune encephalitis was then first suspected between 4-28 days from onset (median=21 days) because of the psychopathology (n=2) or development of clear-cut seizures or movement disorder (n=3). 10 consistently reported features were identified: sleep disturbance, nightmares, mixed unstable mood, perplexity, incoherent repetitive speech, musical ± visual hallucinosis, catatonic facies, possession-like/drugged, dissociative-disorganised, and regressed behaviour. The symptom complex peaked rapidly (within 3 weeks). The peak burden was large and crossed multiple psychopathologic domains. Overall the syndrome is poorly-described by any single primary disorder; mixtures of mixed mood-psychotic-catatonic disorders performed best. Furthermore, it showed clear qualitative and hence diagnostic shifts between onset, peak, and resolution.

Conclusions The psychopathology of NMDAR-antibody encephalitis is complex and dynamic, likely contributing to diagnostic difficulties. However, it appears stereotyped between individuals, hence sensitive features can be derived. Inconsistency with psychosis and/or mood disorder constructs and better approximation with 'mixtures of mixtures' suggests specificity is possible but similar studies with primary disorder comparators are needed. As the disease can only be ruled out with cerebrospinal fluid antibody testing the practical implication is that the mental health system needs to embrace lumbar puncture as a routine part of practice in high risk groups.

9.Predictors of Carer Burden in Impulse Control Disorders in Parkinson's disease.

Dr Leigh Townsend, Dr Daniel Johnson, Prof Anthony David, Dr Sally Askey-Jones, Prof Richard Brown, Dr David Okai

Aims/objectives Impulse control behaviors (ICBs) affect 15-35% of Parkinson's Disease patients. There is evidence of increased carer strain due to these behaviours; however, little is known about clinical variables mediating this effect. This study aims to investigate the factors predictive of carer burden within a cohort of Parkinson's Disease patients with ICBs. Identification of such factors may allow for targeted therapeutic intervention.

Method Data was collected from 45 patients with clinically significant ICBs and their carers including levodopa equivalent daily dosage, assessments of motor and neuropsychiatric symptoms, cognitive function and ICBs. Carer burden was assessed

using the Zarit Burden Interview (ZBI). Univariate analyses were performed using Spearman's Rank Correlation Coefficient. A backward model was used to remove variables to create a final multivariate model using ZBI score as the dependent variable.
Results Univariate analysis identified significant correlations between ZBI and total NPI ($rs=0.50$, $p<0.0005$); four NPI sub-scores: agitation-aggression ($rs=0.41$, $p=0.005$), depression-dysphoria ($rs=0.47$, $p=0.001$), apathy-indifference ($rs=0.49$, $p=0.001$) and irritability-lability ($rs=0.38$, $p=0.03$); and carer GHQ ($rs=0.52$, $p<0.0005$). Multivariate linear regression retained total NPI and GHQ scores, collectively predictive of 36.6% of variance in ZBI.
Conclusions This is the largest study to date, looking at associations between carer burden and ICBs. Our findings indicate low mood, poor motivation, social disinhibition and neuropsychiatric symptom burden to be significant factors in carer burden. We also observe that carers reporting poorer health had increased carer strain. Further work should explore methods of physical and psychosocial support and coping strategies for carers.

10. Medical student education in Sleep and its disorders: Has it improved over 20 years?

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OBJECTIVES/AIMS: There is growing recognition that sound sleep is a pillar of health, alongside adequate nutrition and exercise. Sleep problems are common and often treatable, improving lives. Twenty years ago, Stores[1] revealed the paucity of UK medical school-education on sleep disorders, with a median teaching time of 20 minutes: we investigate here whether this situation has changed.
METHODS: A cross-sectional survey of 34 medical degree courses in the UK, adapted from Stores' 1998 questionnaire, including time spent on teaching sleep medicine, sub-topics covered, and forms of assessment. Responses were coded and analysed numerically where possible; free text was analysed thematically. We excluded responses not concerned with general undergraduate education.
RESULTS Twenty-five (74%) UK medical schools responded to our survey. The time devoted to teaching sleep medicine during undergraduate training was median 1.5-hours, mode-<1 hour, and mean 3.2-hours (standard deviation =-2.6).
Only two schools reported a sleep medicine syllabus or dedicated compulsory module (8%), whilst two had optional student-selected sleep medicine modules (8%). Sleep medicine was generally described as being subsumed into other areas, primarily respiratory medicine, sometimes ENT, Psychiatry and Neurology; coverage of subtopics mirrored this pattern. Asked if enough time is allotted for teaching on sleep medicine, 50% said Yes, 38% No, 13% were unsure.
Free-text comments made by our respondents had recurring themes: sleep medicine is typically **subsumed** into teaching by other specialties, consequently course directors are **uncertain** about the details of provision, **obstructive sleep-apnoea** is often identified as the key or only relevant sleep disorder, knowledge of sleep disorders is regarded as

optional, and there is inertia about the prospect of change. However, a substantial minority of respondents are enthusiastic about making improvements to the sleep education they currently provide, and keen to use additional resources. Examples of good practice exist already, with one school offering an optional 30-hour sleep medicine module annually to 12 students.
CONCLUSIONS Little has changed since Stores' previous survey 20 years ago: sleep medicine remains a neglected topic despite agreement on the importance of sleep for general health. Sleep research is the exception rather than the rule. Obstacles to change are akin to those noted by Stores, including the views that "sleep is not a core topic", or the "curriculum is too crowded". However, there is some enthusiasm for improving sleep education. Given its broad importance to health, and the existence of effective therapies, we recommend that medical schools should implement a sleep medicine curriculum.
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11. Characteristics of the neurocognitive examination in young adults feigning dementia.

Brendan Sargent, Alan Carson, Jon Stone, Laura McWhirter.

Objectives/aims Symptoms and signs of functional (psychogenic) motor and sensory disorder often depend on the individual's ideas about symptoms rather than on the rules of anatomy and physiology. The possibility that functional cognitive symptoms might similarly reflect beliefs about cognitive disorders such as dementia has not been widely explored. We aimed to assess the ideas and beliefs of healthy non-medical adults about symptoms of mild dementia.
Methods Healthy adult volunteers aged 18-35, without medical training, underwent a 25 minute assessment during which they were asked to imagine that they in fact had symptoms of 'mild or early stage dementia'. The assessment included the Montreal Cognitive Examination (MOCA), examination of gait, digit span, the Luria 3-step test, interlocking finger test of parietal function, and the 'coin in hand' forced-choice test. Close family history of dementia and specific perceptions about dementia were recorded.
Results In 27 participants, mean age 21, mean MOCA score was 16 (SD1.4, range 5 – 26). Summed response time ranged from 5.5 to 13 minutes (mean 8.19 minutes SD 1.43). Delayed recall was the most frequently failed item (96%) followed by forward 5-digit span (92%) and letter vigilance (92%). Cube drawing was the least frequently failed item (41%). 33% failed forward 3-digit span and 33% failed reverse 2-digit span. On the 'coin in hand' test, 30% scored at (11%) or below (19%) the level of chance. 22% successfully completed the Luria 3-step test. 85% successfully copied 3 or 4 interlocking finger positions. 37% had abnormal gait. Inconsistent response patterns and difficulty following and remembering instructions was common. There were no significant

differences between those with and without a family history of dementia.
Conclusions Non-medical subjects feigning mild dementia perform worse than we would expect individuals with mild dementia to, suggesting that they perceive dementia as causing severe impairments even in early stages. Most notably, 33% could not repeat a forward span of 3 or backward span of 2 digits. Subjects accurately identified impaired recall as an important symptom of dementia, but failed less often than normative data would lead us to expect on cube drawing and interlocking finger tests. Contrary to expectations, family history did not influence performance. Further investigation will help to establish whether similar patterns of results are produced in individuals with functional cognitive symptoms.

12. Assessing point-prevalence and documentation of head injury in the North London Forensic Service

Berry AJ, Kelsey D, Mitchell R, Taylor R

Objectives/Aims Traumatic brain injury (TBI) is highly prevalent in prison populations, but little is known about TBI prevalence amongst mentally disordered offenders in secure settings. The North London Forensic Service (NLFS) is a tertiary forensic secure psychiatric service, serving a catchment population of over 1.4 million. We aimed to estimate the point-prevalence of head injury amongst inpatients at NLFS, and audit the quality of documentation of head injury.
Method We performed a retrospective case note review of the NLFS electronic records system to identify all documented head injuries in all NLFS inpatients on a selected day. To increase detection, we reviewed neuroradiology reports, and medical records were electronically screened for the following text-strings: "brain injury", "TBI", "head injury", "road traffic accident", "loss of consciousness", "LOC", "concussion", "MRI", "GCS" and "boxing". For each injury identified, we assessed whether amnesia and altered consciousness at the time of the injury were documented, in line with established criteria for assessing severity of TBI (Department of Veterans Affairs/Department of Defence criteria). Injury mechanism and date, patient age, and admission length were recorded. Authors AJB and RM co-rated the findings, and disagreements were resolved by consulting a third party.
Results 100% of inpatient records were screened (n=194, 6% female, 94% male, mean age 40.2 years). 58 documented head injuries were identified, involving 50 patients (25.8%). 43 head injuries occurred prior to admission to NLFS. 16 patients (8.2%) had head injuries associated with clearly documented impairment of consciousness and/or amnesia, suggestive of TBI. 13.7% of all recorded head injuries included explicit documentation of both consciousness level and amnesia (33% when limited to head injuries occurring during admission to NLFS). The most common identified mechanism was assault (n=30). Mean admission length was 1306 days, and was not significantly different in patients with a history of head injury (p=0.825, *Mann-Whitney U*). 34% of inpatients had undergone neuroimaging. Abnormalities were identified in 32% of

reports, with leukoaraiosis and generalised cerebral involutional changes being most frequently reported.
Conclusion: These findings highlight a local need to improve quality of documentation in assessment of patients with head injuries, and provide an estimate of point-prevalence of head injury and TBI at NLFS. The high frequency of assault observed in our sample is comparable to previously published findings in prison populations.

13. Best foot forward? Successful multi-disciplinary novel treatment of fixed equinovarus dystonia in three patients with functional neurological disorder.

Isabel Cary, Martine Nadler, Mike Dilley, Chris Symeon

Objective/aims
Functional Neurological Disorder (FND) affects 10-30% of neurology outpatients. Symptoms commonly include sensory, motor and cognitive changes without structural nervous system damage. Fixed equinovarus dystonia (FEVD) of the foot and ankle is a common feature of FND characterised by plantar flexion and inversion of the foot which cannot be corrected passively. This prevents weight-bearing often causing permanent wheelchair dependence. FEVD correction is necessary for patients to walk again. Consensus opinion is that invasive treatments are ill-advised and potentially detrimental in patients with FND. However, we have developed a novel approach that may challenge this opinion for a specific patient group, combining invasive treatments and neuropsychiatry interventions.
Methods
A patient-led, goal oriented, multidisciplinary approach guided treatment. Treatments included functional electrical stimulation, botulinum toxin, tibial nerve block, serial casting and surgical intervention as an adjunct to specialist physiotherapy, occupational therapy, psychology. Standardised outcome measures of gait and mobility, balance, anxiety and depression were performed on admission and discharge. Patient consent was obtained for photo and video recording.
Results
For three, wheelchair-dependent patients with FND and FEVD admitted to The Wolfson Neuro-rehabilitation Centre for 12-24 week inpatient treatment, our approach resulted in two walking independently and one with supervision. Care needs were reduced and wheelchair dependence was eliminated. Patients reported improvement in independence and quality of life with one patient returning to part-time employment as a PA.

Case	19 years female dancer 5 year history symptoms 2 year FND diagnosis 19 week admission	33 years mother 12 year history symptoms 18 month FND diagnosis 12 week admission	30 years female PA 4 year history symptoms 3 year FND diagnosis 24 week admission
Wheelchair dependence	2 years	1.5 years	2.5 years
Discharge mobility	Independent walking	Supervised walking with crutches	Independent walking with frame
Pre- admission care needs	Carer QDS Hoist transfer Hospital bed Commode Downstairs living	Carer TDS Slide board transfer Downstairs living	Boyfriend full time carer Turning aid transfer
Discharge care needs	Nil	Carer BD	Nil
Six-month follow up	Dancing No care needs No recurrence of FEVD	Furniture walking Family support only No recurrence of FEVD	Returned to work Independent walking with crutches No care needs No recurrence of FEVD
Intervention 1	FES	FES	FES
Intervention 2	BoNT	TNB BoNT	TNB BoNT
Intervention 3	Not required	Ankle cast/splint	Ankle cast/splint
Intervention 4	Not required	Not required	Ankle manipulation under anaesthetic
Intervention 5	Not required	Not required	Surgical release of Achilles Tendon, Tibialis Posterior, long toe flexors

Table

Functional Electrical Stimulation to common peroneal nerve (FES)
Botulinum Toxin to posterior tibial muscles (BoNT)
Tibial Nerve Block (TNB)

Conclusions With selective psychological and medical screening, invasive treatment for FEVD in FND patients delivered through a careful, stepwise treatment pathway produced excellent results for this subgroup of patients. Though such interventions are usually avoided for patients with FND, there may be a subgroup of patients for whom they remain useful as a treatment adjunct in order to maximise rehabilitation and functional outcomes.

14. Prediction of emerging amyloid and tau pathology in cognitively healthy individuals: A longitudinal CSF study

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Objectives/aims The importance of identifying emerging rather than widespread amyloid pathology is highlighted by evidence showing that rapid amyloid accumulation associates with cortical atrophy, cognitive deficits and tau deposition even where individuals are in the 'amyloid negative' range. The study aimed to test the hypothesis that among cognitively healthy individuals distinct groups exist in terms of amyloid and phosphorylated tau accumulation rates. We also hypothesised that membership to the faster accumulator group can be predicted (using age, genetics, cognition and hippocampal volume). Finally we aimed to identify time points of significant increase in the rate of AD protein accumulation and the associated predictors.
Methods The analysis reports data from 263 individuals from the BIOCARD NIH-funded study who had a mean of 2.23 cerebrospinal fluid (CSF) measurements since the study began in 1995. We used latent class mixed-effect models to identify distinct classes in terms of amyloid and p-tau accumulation rates. To investigate non-linear changes of AD-related CSF biomarkers we used generalised additive modelling. For both analyses demographic, genetic (APOE4 genotype) as well as repeated cognitive and structural MRI data were explored as predictors.
Results For both amyloid and p-tau latent class models we confirmed the existence of two separate classes: accumulators and non-accumulators. The accumulator and non-accumulator groups differed significantly in terms of baseline AD protein levels (mean age 60 at study inclusion) and slope. APOE4 homozygosity and cognitive reserve predicted amyloid class membership while APOE4 genotype only predicted p-tau class. The non-linear models revealed significant increase in the rate of amyloid and p-tau accumulation at ages 54 and age 55 respectively. APOE4 carrier status associated with earlier age of rapid accumulation start (ages 53 and 50 for amyloid p-tau respectively). Higher hippocampal volume but not cognitive ability was protective for amyloid accumulation. Neither hippocampal volume nor cognition were significant in the p-tau non-linear models.
Conclusions The current analysis demonstrates the existence of distinct classes of amyloid and p-Tau accumulators. Predictors for class membership were identified but the overall accuracy of the models was modest highlighting the need for biomarkers that are sensitive to early disease phenotypes. That amyloid and p-tau concentrations remain largely static until the mid-50s with a simultaneous sharp increase in

accumulation thereafter also suggests that prediction of these points of inflection can be valuable in targeting specific time periods in disease modification trials.

15. Emotional lability in hippocampal atrophy due to autoimmune limbic encephalitis

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Objective/Aims Autoimmune limbic encephalitis (LE) is commonly associated with cognitive and psychiatric disturbances at the acute stage of the disease, and with residual episodic memory impairment. While behavioural and psychiatric symptoms generally dissipate post-acutely, very little is known about the profile of persistent neuropsychiatric symptoms. In particular, emotional lability represents an elusive entity that may be misdiagnosed as a manifestation of comorbid mood or personality change and can have disabling consequences, due to the stigma attached to the loss of emotional control. We aimed to assess the post-acute profile of emotional lability and its neuroanatomical correlates in LE.
Methods We analysed acute neuroradiological reports, clinical notes, scores on post-acute neuropsychological tests and self-administered questionnaires on mood, emotion, and affect (including the Centre for Neurologic Study-Lability Scale; CNS-LS), along with structural MRI and resting-state fMRI datasets in relation to emotional lability in a large cohort of patients (n = 36) that had received a neurological diagnosis of LE, presented with focal hippocampal structural abnormalities in the acute phase, and post-acute hippocampal atrophy and thalamic volume reduction.
Results Emotional lability was present in 50% of the patients. It was associated with increased tearfulness compared with non-labile patients and healthy controls, whereas no patient presented with labile laughter (CNS-LS). Patients with emotional lability (n = 18) did not differ from those without (n = 18) in any demographic or clinical details in their acute or post-acute presentation (autoantibodies, immunosuppressive therapy, seizures, antidepressant medication, age at or delay from symptom onset), or in residual depression, anxiety, impulsiveness, memory impairment, or executive dysfunction, or in hippocampal and thalamic volumes. Instead, the presence and extent of emotional lability across patients was associated with reduced resting-state hemodynamic activity in and hippocampal functional connectivity with regions in the inferior and superior parietal lobules.
Conclusions We present the first investigation of persistent affective dysregulation in LE. Emotional lability is common following LE, but is not a manifestation of depression, anxiety, impulsiveness, or executive dysfunction. The type of emotional lability seen in LE is semiologically distinct from pseudobulbar affect observed in other neurological diseases. While LE is characterised by focal hippocampal atrophy, functional abnormalities in regions interacting with the hippocampus may provide a more parsimonious explanation of emotional lability than the volume of medial temporal lobe

structures. Functional abnormalities in parietal regions supporting perspective taking and social-affective processing may compromise patients' emotion regulation.

16. First episode psychosis in a patient with extensive leukoencephalopathy due to 3-methylglutaconic aciduria Type 4.

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Objectives/aims This aim of case report is to discuss the clinical conundrum and diagnostic challenges in a young patient presenting with First Episode Psychosis. Investigations revealed Extensive Leukoencephalopathy due to a rare metabolic disorder- 3-methyloglutaconic aciduria (3-MGA) type IV. Several studies have shown that 3-MGA type IV can present with psychosis, epilepsy or depression as part of the spectrum of symptoms. The role of Organic Brain condition in the onset of first episode psychosis in this patient is discussed in this report.
Methods A 23-year-old female presented with insidious onset of paranoid delusions and auditory hallucinations over an 18-month period on a background of a diagnosis of 3-methylgutaconic aciduria type IV confirmed on urine testing. On admission under Section 2 of the Mental Health Act, she expressed little spontaneous speech and echolalia. She was flat in affect. She appeared vacant in expression, stared inappropriately and was very self-isolating. She was responding to external stimuli. She lacked insight into her condition. Physical examination was unremarkable. An MRI brain scan was performed, and comparison made to scan done previously to demonstrate any interval change and to correlate changes if present to deterioration of clinical symptoms.
Results MRI scan showed extensive diffuse leukoencephalopathy. Comparison to MRI scan done 6 years previously did not show any change or progression to the white matter lesions. An EEG showed a mild degree of general cerebral dysfunction with no interictal epileptiform activity. There was no correlation found between her clinical symptoms of acute onset psychosis and her diagnosis of 3-MGA Type IV. She was commenced on Aripiprazole and her presentation improved significantly. Both Auditory hallucinations and Paranoid delusions improved considerably, with moderate improvement in mood, affect and apathy. Some Catatonic symptoms persisted but were less intense. She was given a diagnosis of Undifferentiated Schizophrenia under ICD 10, as she displayed features of Paranoid, Hebephrenic and Catatonic without clear predominance of particular subtype of Schizophrenia. She was discharged home with follow-up from Neuropsychiatry and community Mental health teams. She continues to be investigated for the genetic cause of 3-methylglucatonic aciduria Type 4.
Conclusions To conclude, although often metabolic disorders, including 3-methylglucaconic aciduria, can present with psychosis, it is prudent to establish a causative link in order to manage appropriately and effectively.

17. The future role of fMRI neurofeedback in depression treatment and research

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Objectives/Aims FMRI-neurofeedback for major depressive disorder (MDD) is of great interest to clinicians and neuroscientists. Here, the aim was to review the current clinical trials evidence.
Methods We undertook a systematic literature search of fMRI-neurofeedback trials in MDD, including our unpublished results.
Results fMRI-neurofeedback was effective in current MDD when reinforcing brain responses to positive pictures(*1*), but was not superior to a control neurofeedback intervention in a recent randomised controlled trial(*2*) (RCT). Another RCT showed that reinforcing amygdala responses to positive autobiographical memories(*3*) was superior versus a control neurofeedback intervention(*4*). We have developed neurofeedback of self-blame-selective functional connectivity between right superior anterior temporal (AT) and subgenual frontal regions. In remitted MDD, we demonstrated that self-esteem can be increased using this approach in a double-blind RCT. In a recently completed RCT in early treatment-resistant MDD, the majority of patients responded to guilt-related AT-subgenual connectivity neurofeedback. Surprisingly, a self-guided matched psychological intervention tackling self-blame without neurofeedback showed comparable levels of response. Secondary analyses, however, showed that neurofeedback was superior for those patients without anxious distress features.
Conclusions This calls for longer-term studies to reproduce previous results and stratified trials to combine psychological and neurofeedback interventions. As a research tool, neurofeedback uncovers causal relationships between functions and anatomical subdivisions.

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18. The epidemiology and symptomology of functional stroke mimics: a systematic review and meta- analysis.

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Aims Reconfiguration of stroke services in England has emphasised fast diagnosis and treatment and subsequently, the proportion of stroke mimic patients entering stroke pathways has been highlighted. Stroke mimic patients may be 'medical mimics', with medical explanations for symptoms, e.g. syncope, seizure, but a proportion of presenting patients have a functional aetiology. Functional stroke mimics accounted

for 8% of admissions to an acute stroke service in London (Gargalas et al., 2015) and the prevalence and burden of functional stroke is well recognised by clinicians. We present a systematic review and meta-analysis aiming to: 1) estimate the prevalence of stroke mimics and functional stroke mimics across medical settings; and 2) describe the demographic and symptom profiles of functional stroke patients.
Methods Three literature searches took place between 2015-2018 utilising OvidSP, PubMed, CINAHL and Google Scholar. A total of 13,974 abstracts were reviewed and 114 papers met inclusion criteria. Age and sex proportions were compared between stroke, stroke mimic and functional mimic groups. Prevalence rates across settings and moderators of functional mimic rates were calculated using random-effects models.
Results Stroke, stroke mimic and functional mimic definitions varied between studies. Across settings, 25% of suspected stroke patients were stroke mimics and 15% of stroke mimics had a functional aetiology. Stroke mimics were younger than stroke patients and more likely to be female. Similarly, functional patients were younger and more often female than medical mimics. 10 papers gave symptom information for functional patients; compared to medical mimics, functional patients were more likely to display weakness/numbness and less likely to present with reduced consciousness, visual symptoms or speech/language symptoms. Meta-analyses show a higher rate of stroke mimics in primary care (38%) vs more acute settings (12%) but the inverse for functional mimics (24% in stroke units vs only 12% in primary care). Functional rates were highest in studies that were descriptive, retrospective, from high income countries and in studies where all patients received thrombolysis.
Conclusions Functional diagnoses are an important differential of suspected stroke. Definitions of functional stroke mimics vary widely in stroke literature. Our findings suggest functional stroke patients are most commonly seen in tertiary settings. There are no guidelines on the management of these patients within acute stroke settings. In the context of these findings, a feasibility study is underway investigating the presentation of functional stroke patients and their views on possible interventions and this research may help improve current care pathways.

19. Medication prescriptions in 322 functional motor disorder patients in a large UK mental health service: a case control study

Nicola O'Connell, Timothy Nicholson, Graham Blackman, Jennifer Tavener, Anthony S. David

Objectives: This study aims i) to describe the patterns of prescription medication in functional motor disorder (mFND) treated in a Mental Health Trust, using a control group comprised of a random sample of contemporaneous patients treated in the same trust; and
ii) explore the sociodemographic variables and symptoms linked to higher medication usage.
Methods: This is a retrospective case-control study using an electronic psychiatric case register in South London and Maudsley NHS Foundation Trust (SLaM). SLaM provides psychiatric inpatient and community services and receives national referrals for

functional neurological disorder (FND) patients. Data were obtained from 322 mFND patients and 644 controls between 1st January 2006 and 31st December 2016 using the 'Clinical Records Interactive Search' (CRIS) database.
Results: 247 (76.7%) mFND patients were prescribed medication, lower than 83.4% in the control group (OR: 0.59, 95% CI: 0.39 – 0.89, $p < 0.02$). The mean number of prescribed medications in the mFND group was 4.77 (SD: 2.4), higher than 2.98 (SD: 2.7) in the psychiatric control group ($t(782) = 7.9$, $p = 0.001$). Amongst mFND patients receiving medication, the most common prescriptions were antidepressants (68% received one or more), anti-epileptics (33.5% on one or more), non-opioid painkillers (32.4%), and opioid analgesics (31.2%). Compared to psychiatric controls, mFND patients had a higher likelihood of receiving anti-depressants, medications for cardiovascular disease, statins, antihistamines, anti-asthmatics, corticosteroids, anti-epileptics, hormone replacement therapy, proton pump inhibitors, bowel and urinary dysfunction medication, NSAIDs, and muscle relaxants. mFND patients were significantly less likely to receive antipsychotic medication or treatments for substance misuse. An adjusted analysis of mFND patients found co-morbid physical conditions and previous psychiatric admissions were associated with higher numbers of medication prescriptions.
Conclusions: mFND patients are prescribed an extensive range of medications for psychiatric and somatic symptoms, most commonly anti-depressants, anti-epileptics and analgesics. The diversity in medications may be partially explained by higher rates of physical co- morbidities but may also reflect 'somatisation' or excessive symptom reporting combined with a lack of therapeutic options for clinicians managing patients with complex functional and 'organic' conditions and chronic pain. Non-essential medication prescribing may reinforce somatic illness beliefs and cause iatrogenic harm, particularly high rates of opioid pain medication, with important implications for clinical management in primary and secondary care.

20. Outpatient cognitive behavioural therapy for 'functional' and 'organic' neuropsychiatric disorders: a retrospective case control comparison

Nicola O'Connell, Gillian Watson, Clare Grey, Rosa Pastena, Kenneth McKeown & Anthony

S. David

Objectives There is no gold standard treatment for functional neurological disorder, motor type (mFND). Cognitive behavioural therapy (CBT) is effective in the treatment of certain somatoform disorders. This study aims to evaluate the characteristics and outcomes of mFND patients receiving CBT in a neuropsychiatry outpatient clinic.
Method We utilise a large psychiatric register to assess all mFND patients receiving outpatient CBT in a neuropsychiatric clinic between 2006 and 2011. We assess socio-demographic characteristics, changes in physical and psychological outcomes using standardised rating scales, and rates of CBT uptake and dropout. We compare mFND

patients to patients with psychiatric and behavioural manifestations of organic neuropsychiatric diseases treated in the same clinic (ONP patients).
Results The most common reasons for patients not accepting a CBT referral were referral to the Trust's inpatient neuropsychiatry ward, or non-attendance at assessment or treatment sessions. We identified a total of 98 patients with mFND and 76 ONP patients treated with CBT. 56.1% of mFND and 56.6% of ONP patients attended all sessions offered. There were no socio-demographic characteristics associated with mFND patients who completed all therapy sessions versus those who dropped out early. Compared to controls, mFND patients were more likely to be female (72.4% v. 44.7%, χ^2: 13.6, 95% CI: 12.2 – 41.9, $p = 0.001$), unemployed (52.6% versus 35.5%, χ2: 5, 95% CI: 2.2 –
30.8, $p = 0.03$), to have a carer (27.6% versus 14.3%, χ2: 4.4, 95% CI: 0.9 – 24.7, $p = 0.04$) and to have experienced child sexual abuse (23.8% v 8.2%, χ^2: 7.3, $p = 0.01$). The most common mFND symptom was weakness (26.9%), most frequently in the leg or entire body. Both mFND and ONP patients showed significant improvements in measures of psychological functioning (measured with the CORE-OM, HoNOS-ABI, and PHQ-9), with physical symptoms improving in 49.4% of mFND patients. A logistic regression showed acceptance of psychological formulations prior to CBT ($p < 0.02$) was associated with the improvement of physical symptoms in mFND patients.
Conclusion mFND patients treated in a specialist CBT clinic show similar improvements in physical and psychological functioning to ONP patients with positive effects on motor symptoms, distress, depression, general health and social functioning. With little evidence on the effectiveness of CBT in treating motor variants of FND, this study could help inform the design of a pilot RCT.

21. Are there any factors that predict the diagnosis of epilepsy or psychogenic non-epileptic seizures (PNES) in patients admitted to a specialist epilepsy unit?

Dr Stuart Gibson, Dr Samuel Leighton, Dr Maria Oto

Introduction Differentiating between epilepsy and psychogenic non-epileptic seizures (PNES) can be difficult. Although clearly not a substitute for taking a careful history, certain patient characteristics may assist the clinicians towards diagnosis. The population of patients referred to an epilepsy specialist centre represent a complex and distinct group of patients and it is not clear which factors, if any, could point towards a diagnosis of epilepsy or PNES.
Aims/Objectives To investigate if the diagnosis of epilepsy or PNES is predicted by baseline demographic and clinical variables, including Hospital Anxiety and Depression (HADS) scores and medication prescription, in patients admitted to a specialist adult epilepsy centre.
Methods We conducted an observational retrospective cohort of consecutive patients admitted to the William Quarrier Scottish Epilepsy Centre (WQSEC) over a period of one

year (01/09/16-01/09/17). Chosen predictor variables at baseline included: sex, age, employment education or training (EET), Scottish Index of Multiple Deprivation 2016 (SIMD 2016) rank status, attack frequency, length of index admission, number of anti-epileptic agents prescribed, prescription of benzodiazepines, of analgesia, or of psychotropic medications, and HADS scores. Outcome measures were diagnosis of epilepsy or PNES, from diagnosis made by expert clinicians on discharge from index admission. Because of the presence of dual diagnosis, two multivariable binary logistic regression models were built – one for the epilepsy and one for the PNES diagnosis outcomes.

Results 50/73 (69%) of patients admitted were diagnosed with epilepsy and 39/73 (53%) with PNES. These respective groups include 16/73 (22%) who had a dual diagnosis of both epilepsy and PNES. The model to predict epilepsy showed that significant individual predictor variables included number of antiepileptic agents prescribed (Odds Ratio (OR) = 3.59 (95%CI: 1.37, 9.42), p = 0.010), prescription of psychotropic medications (OR = 0.19 (95%CI: 0.04, 0.91), p = 0.038), and length of index admission (OR = 0.89 (95%CI: 0.81, 0.98), p = 0.018). The model to predict presence of PNES revealed only one significant individual predictor variable, which was EET status (OR 0.13 (95%CI: 0.02, 0.86), p = 0.035).

Conclusions Baseline clinical and demographic factors may be of some utility to the clinician in anticipating a diagnosis of epilepsy or of PNES.

22. The Immunomodulatory Effect of Clozapine in Patients with Treatment Resistant Schizophrenia: A Retrospective Cohort Study

Lisshammar, J., Blackman, G., Carter, B., Zafar, R., Stewart, R., Pritchard, M., Pollak, T., Rogers, J., Cullen, A., McGuire, P., David, A. & MacCabe, J.

Objectives The pathoetiology of Schizophrenia remains elusive, however, a growing body of literature suggests immune dysfunction may contribute. Clozapine, an atypical antipsychotic, has superior efficacy in treatment-resistant Schizophrenia compared to other antipsychotics – however underlying mechanisms remain unknown. Clozapine has recognised immunomodulatory effects, responsible for potentially fatal haematological side-effects - such as agranulocytosis. Whether Clozapine's immunomodulatory properties contribute toward its unique efficacy in treatment-resistant schizophrenia has not been systematically explored.

Methods A retrospective cohort study design was employed to examine the relationship between white cell, neutrophil, and platelet temporal trajectories and Clozapine response in treatment-resistant schizophrenia. Eligible patients were initiated on Clozapine for the first time and continued treatment for at least twelve weeks between 2007 and 2014 within the South London and Maudsley NHS Foundation Trust, and underwent weekly haematological monitoring. Retrospective clinical ratings were performed at baseline and three months following initiation, based upon patients' electronic clinical notes accessed through the Maudsley BRC Clinical Records Interactive Search system. Treatment response was defined as 'much' or 'very much' improved on the Clinical Global Impression

– Improvement subscale. Serial cell counts were extracted from a Clozapine haematological monitoring database.

Results Of 188 included patients, 114 (61%) responded to treatment. Response did not significantly vary by ethnicity or age. Mean interval between haematological assessments was 6.9 (*SD* 2.0) days. General linear models revealed a significant increase from baseline for all cell lines in the second treatment week ($p = 0.001$) which persisted for three weeks ($p = 0.001$). Group based trajectory modelling indicated that 15% of patients showed a temporary increase in white cell and neutrophil trajectories. Logistic regression revealed that treatment response was not associated with a 'spike' in cell count. However, females were 2.08 times more likely to respond to treatment (95% CI [1.09, 4.08], $p = 0.017$) and 2.67 times more likely to exhibit a 'spike' in cell counts following Clozapine initiation (95% CI [1.14, 6.24], $p = 0.023$).

Conclusions This is the first study to examine whether Clozapine's immunomodulatory properties contribute towards its unique efficacy. We found that clozapine was associated with an early 'spike' in all cell lines. Further analysis revealed a relatively small portion of patients were responsible for this transient increase. Increased cell counts did not predict Clozapine response at three months, but gender was implicated as a potential moderator variable.

23. A multi-speciality conundrum: neuropsychiatric sequelae of thyrotoxicosis.

Lori Black

A 51 year old man with bipolar affective disorder and an extensive forensic history was admitted informally to the forensic low secure inpatient unit for alcohol detoxification and to establish more effective long-term community management. 3 weeks into the admission his presentation abruptly changed. He would have prolonged periods of unresponsiveness, lasting hours to days, where he would lie in bed or stand rigidly with no vocal response to commands or questions. These were interspersed by periods where he would become agitated, pacing the wards, urinating and defecating in public spaces. This could last several hours and required rapid tranquilisation regularly.

On examination in his stupor, he was seen to be lying in bed with his eyes closed, opening them a fraction to voice. He was sometimes able to follow simple commands but this was slow and inconsistent. He was unable to communicate through head movements or blinking and stared with a fixed expression. There was waxy flexibility of his limbs and arching on the back at regular intervals. Power was normal with flexor plantars. There were no ictal signs (i.e. no nystagmus/gaze deviation/twitching/dystonia). When agitated he was seen to have echopraxia. He consistently had hyperhydrosis and tachycardia.

Bloods showed an elevated T4 (22.7) with a suppressed TSH (<0.01) and a normal T3. TSH receptor and thyroid peroxidise antibodies were positive. A CT head was normal. Channel and receptor antibodies were normal.

It was felt that the patient had developed catatonia secondary to thyrotoxicosis due to his underlying neuropsychiatric susceptibility. He was treated effectively with high dose lorazepam as per the Maudsley Guidelines along with olanzapine (20mg) and sodium valproate, which was used as an alternative to lithium. The thyrotoxicosis was effectively treated with carbimazole and the patient made a good recovery.

Hyperthyroidism is a rare but recognised cause of psychosis and multiple case reports have demonstrated an association between thyrotoxicosis and catatonic states. In this case report, it is probable that the patient's underlying bipolar affective disorder made him more susceptible to developing neuropsychiatric features as a consequence of his thyrotoxicosis. However, this is particularly pertinent given that lithium, the most evidence-based treatment for bipolar affective disorder, has potential to disrupt thyroid function.

24. Korsakoff's syndrome: Neurocognitive Domains impairments and potential Therapeutic interventions.

Jennifer Hoblyn MD MRCPsych, Tayler Sulse, Emma Mae Huston, Patrick Byrne MRCPSI and Kieran O' Driscoll MRCPSych

Objectives/aims To explore the burden of neurocognitive impairments in a cohort of Individuals with Korsakoff's psychosis requiring current long stay psychiatric care.
To consider additional therapeutic interventions to target the health care burden potentially created by these comorbidities.
Methods As part of a comprehensive systematic review of Korsakoff's Psychosis, an audit was performed of 114 Individuals currently requiring long-term care in an approved psychiatric facility during the year of 2018. Medical and psychiatric diagnoses as well as pharmacological histories were examined. Alcoholic and non-alcoholic aetiologies were considered, the latter may be underdiagnosed (Nikolakaros et al, 2018).
Results Thirteen individuals were identified with a formal diagnosis of Korsakoff's syndrome (KS) and all continue to require structured Inpatient care due to their levels of neurocognitive impairment and psychiatric presentations. Episodic memory is severely affected, as is the learning of new semantic memories. Patients with Korsakoff's psychosis are capable of new learning in a calm, structured environment with cued new information (Kopelman et al, 2009).

Table 1: DSM 5 Neurocognitive Domains

Patient Number	Complex Attention	Executive Function	Learning and Memory	Confabulation	Language	Perceptual Motor	Social Cognition
1	X	X	X	X		X	X
2*	X	X	X			X	X
3	X	X	X	X			
4	X	X	X			X	X
5*	X	X	X	X		X	X
6		X	X	X			
7	X	X	X				X
8*	X	X	X	X	X	X	X
9	X	X	X	X	X		
10			X		X		
11	X	X	X				
12		X	X	X			X
13**	X	X	X		X	X	

Key - *TBI Co Morbidity
** Co Morbid Intellectual Disability

Conclusions Individuals with Korsakoff's psychosis may have comorbid psychiatric symptoms including mood, anxiety, aggression or psychotic disorders that command therapeutic interventions. Specific memory targeting intervention may not prioritized. Potential therapeutic interventions include Errorless learning (EL) which target levels of competence and independence (Rensen et al, 2017). EL is reported to improve symptoms of psychosis, aggression, apathy or mood disorders. Behavioural Interventions include environmental adaptations and cognitive remediation, which may be combined with pharmacological approaches such as donepezil or memantine to target cognition (Johnson and Fox, 2018). However, these approaches are not identical to those required by Alzheimer's disease or other dementing disorders. Epidemiological

and genomic studies could be preformed to identify those particularly at risk of developing this potentially life-altering condition.

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25. Multi-agent allergies as predictor of Functional Neurological Disorder

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Objectives/Aims Functional neurological disorders (FND) account for 20% of patients in neurology clinics and can lead to functional impairment, multiple re-attendances and significant cost. However, diagnosing FND remains challenging; identifying associated factors could aid earlier diagnosis. We aimed to determine the value of self-reported multi-allergies as predictor for FND.

Methods We retrospectively reviewed records of consecutive patients from two clinics (General Neurology & FND), St George's Hospital, January 2015-June 2018. A logistic regression model was used in conditional fashion; statistically significant variables in univariate analysis were included.

Results Of 720 patients with definitive diagnosis, 243 (33.8%) had FND and 477 (66.3%) another neurological disorder. Mean age was 43 years (range 16-93), 63.9% (453) were female. 81 patients with FND (33%) had Non-epileptic attack disorder (NEAD).

In multivariate analysis, factors associated with FND were female sex (Odds Ratio [95% Confidence Intervals], OR = 0.49[0.33, 0.73], p<0.001), psychiatric comorbidity (OR=4.28[2.89, 6.35], p<0.001), younger age (OR=0.97[0.96, 0.98], p<0.001), coexisting fibromyalgia/chronic fatigue syndrome (OR=4.85[1.26, 18.73], p=0.02) and allergies (OR= 2.54[1.79, 3.62], p<0.001). Polypharmacy, medical comorbidities, atopy and hypermobility were not significant.

Increased number of allergies increased the probability of FND: one allergy OR=4.53[3.08, 6.65, p<0.001], two OR=9.09[3.92, 21.09, p<0.001], three OR=16.74[3.82, 73.43, p<0.001] and >=4 allergies OR=42.94[2.51, 736.02, p=0.009].

Conclusions Previous studies highlighted the increased prevalence of allergies in NEAD compared to epilepsy. Our study expand this to all FND, as only 1 in 3 FND patients had

NEAD. Presence of allergies, particularly to multiple agents, should raise the suspicion of FND.

26. Effect of Dopaminergic medication on risk preference in Parkinson's disease

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Introduction Dopaminergic medication being the standard therapeutic treatment improves motor symptoms in Parkinson's disease (PD) but also implicated in the occurrence of impulse control disorders. Data driven computational models such as drift diffusion model utilize behavioural measures to explain subtle changes that are not sensitive to traditional analysis. Here, we aim to analyse risk preference in PD subjects in OFF and ON medication and the effect of dopamine on risk.
Methods Sixteen patients PD patients during OFF medication and 14 during ON were tested on the 2 step sequential learning task. We calculated the risk associated with each choice (variance of reward probability) and defined the choice with maximum variance as the risky one, for all 134 trials. With behavioural measures (selected choice-risky vs non-risky and response time) as inputs and risk as an independent factor, we extracted threshold (a), drift rate (v) and response bias (z) parameters using a hierarchical drift diffusion model (HDDM) for both groups during ON and OFF drug condition. Statistical analysis on the parameters was analysed using Bayesian factors.
Results Bayesian Independent sample t-test between the 2 groups (ON vs OFF) showed a strong evidence for differences in drift rate (BF_{10} = 34.28) and response bias (BF_{10} = 1.5 x 10^{13}). We did not observe any evidence for correlation between RL parameters and z for both ON and OFF condition. Behaviourally, with respect to response time, independent sample t-test showed no significance difference between time taken to make risky (t (28) = -1.28, p = ns) and non-risky choices (t (28) = -1.06, p = ns). Similarly, no difference was found for change in risky choice selection in presence of the drug (t (28) = -1.41, p = ns). No differences were found in the traditional reinforcement learning parameters between the groups.
Conclusions Using a novel computational analysis, we showed that dopaminergic medication increased the preference to select a risky choice by modulating drift rate and response bias which was not captured by the behavioural measures. Critically we observe an effect on response bias highlighting the role of *apriori* information in influencing risky decision making.

27. Lifespan of Negative Experiences in Functional Neurological Disorder Patients

Aneesa Mehmood, Dr Liat Levita, Professor Markus Reuber, Dr Emily Mayberry

Aims Exploration of the relationship between negative life experiences and patients with Functional Neurological Disorder (FND), by analysing patient and non-clinical group responses to a new questionnaire called the Lifespan of Negative Experiences Scale

(LiNES). LiNES was designed to examine predisposing vulnerabilities and perpetuating factors in individuals with FND by retrospectively assessing experiences of interpersonal trauma, affect and relationship insecurity at three developmental stages – childhood, adolescence and adulthood.
Methods LiNES, CATS (measure of childhood abuse and trauma), RSQ (measure of relationship insecurity) and PANAS (measure of affect) questionnaires were administrated to 71 individuals with FND. Analyses were conducted to assess the reliability of the LiNES, explore correlations between different psychological domains within the FND group and to test whether LiNES scores predicted FND group membership. In addition, FND patients' responses where compared to 270 matched healthy controls.
Results The LiNES subscales had high internal consistency and correlated with CATS, RSQ and PANAS. Levels of interpersonal trauma were higher in FND patients than controls during childhood, adolescence and adulthood. High levels of negative affect were found in FND patients in adulthood compared to controls but no significant differences were found between FND patients and controls in relationship insecurity at any developmental stage. On the RSQ, FND patients had higher anxious and avoidant relationship styles. LiNES trauma scores at each developmental stage predicted FND status with over 80% accuracy. Additionally, FND patients self-reported more symptoms (SDQ-20) and a higher prevalence of comorbid conditions compared to controls.
Conclusions The LiNES is a new brief retrospective measure of negative life experiences. Although psychological factors may not be necessary to the diagnosis of FND, they are substantially more common in FND patients compared to controls. In particular, a history of interpersonal trauma seems to play an important role in those with FND. These factors therefore are likely to play a pathophysiological role in many patients and their recognition is important for treatment. This study provides new insights into the association between the timing of negative experiences and the subsequent effect on an individual. Furthermore, the results support the use of LiNES as a valid screening tool in the clinical setting in patients presenting with functional symptoms with diagnostic and therapeutic implications.

28. Functional neurological disorder in geriatric rehabilitation: Incidence, clinical presentations, and impact on discharge

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Background/ aims Functional neurological disorder (FND) may be present amongst elderly people in hospital. FND could hinder patients' rehabilitation progress and impact negatively on discharge outcomes. Little data exist for FND in the elderly.
We aimed to report the incidence of FND, clinical presentations, co-morbidities, and impact of FND on discharge in elderly patients receiving inpatient rehabilitation.
Methods In our retrospective case series, a consultant geriatrician reviewed electronic case notes of consecutive discharges from a 28-bed geriatric rehabilitation unit at St

John's Hospital, which serves all patients requiring inpatient rehabilitation in West Lothian—a mixed rural and urban area with a population of 180,000 and high levels of deprivation. Data collected: demographics, suspected/definite diagnosis of FND and its presentation, significant co-morbidities and impact on discharge.
Results We reviewed case notes of 100 patients discharged consecutively from 30/3/2018 to 30/10/2018 (age range 41-101, mean 79, SD 11; 55% men). 20% received a diagnosis of suspected or definite FND. FND diagnosis was made by a geriatrician (17%) or a neurologist (3%). Clinical description of FND cases and their co-morbidities will be presented in a summary table. Of the 20 FND cases (mean age 77, SD 14), 9/20 (45%) were men. FND impacted on discharges in 13/20 (8/20 had delayed discharge, 5/20 had increased care needs, 7/20 had no impact on discharge).
Conclusion
Key finding
FND was common amongst elderly patients receiving inpatient rehabilitation. FND presentations were varied. Patients with FND also had chronic conditions common in the elderly e.g. Parkinson's disease, stroke, dementia, anxiety or depression.
Weakness & strength of our study
Assessor bias might be introduced as diagnosis was made by a geriatrician with an interest in neuropsychiatry. Our data are likely generalisable to the geriatric rehabilitation population as sample was obtained from the only unit that served the entire population of West Lothian.
Implications for future research and practice
FND presents a unique challenge in the geriatric population. Geriatricians are not accustomed to assessing and managing FND, sometimes dismissing symptoms as "behavioural". This can lead to symptoms remaining unexplained and untreated. Specialist neurology or neuropsychiatry services are not always available.
The identification of FND and its effective treatment during rehabilitation could have potential impact on hospital length of stay and associated cost. Further research in FND in the elderly is needed. Better education would raise awareness of FND amongst geriatricians and thus its identification in clinical practice.

29. predictors of delirium in patients admitted in a general hospital
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Background and aims: Delirium is associated with numerous complications such as physical problems, cognitive impairment, prolonged hospital stay and increased mortality rates. Therefore, the diagnosis and prevention of delirium is an essential issue in admitted patients care. The purpose of this study was to investigate the prevalence and risk factors of delirium in a general hospital.

Materials and methods: This study has a descriptive method done on 220 samples between 2017 to 2018. Subjects were recruited among all adult patients admitted to the general hospital who had a Richmond Agitation Sedation Scale (RASS scale) of more than 3. Patients with decrease in the level of consciousness who were unable to answer questions and patients who were admitted to the Intensive care unit were excluded. After primary assessment, demographic data were obtained and secondly the rate of delirium was evaluated by using CAM (Confusion Assessment Method)
Results: The prevalence of delirium was 10% in hospital admissions. The rate of delirium was in emergency ward (31.3%), hematology (22.2%), internal medicine (11.6%) and surgery (2.4%). The prevalence of delirium increased with age, visual deficits, sleep disorders, dementia, and neurological diseases, diabetes and malignancies. The use of antibiotics, analgesics and sedative agents was associated with an increase in delirium, but only there was a significant relation with the use of anticoagulants.
Conclusions: Delirium almost has been detected in all general hospital wards and its prevalence was related to age, visual problems, sleep disturbances, existence of dementia and neurological diseases and the usage of anticoagulant agents.
Keywords: delirium, predictors, delirium

30. Undergraduate teaching and perceptions of Functional Neurological Disorders

Alex Storrar, Dane Rayment and Elizabeth Mallam

Objectives: To explore the level of teaching given to medical students on functional neurological disorders (FND), and whether this was considered sufficient. In addition, to assess the understanding and perceptions of FND within the medical student population.
Methods: Data were collected over 2 weeks from University of Bristol medical students through use of an online survey. This comprised of 2 open questions exploring personal opinions on the extent of teaching and aetiology of FND, and a further 21 closed questions on student demographics, clinical experience and teaching.
Results: The level of teaching was viewed by the majority (27/44) to be insufficient and most students would value more training. When provided, FND teaching was delivered during psychiatry rotations. 34% (15/44) of students had not encountered FND before. Of those that had, 58% (17/29) gained awareness of FND through medical school. Students estimated 63% of cases were associated with a recent stressful life event and additionally 75% (33/44) believed FND symptoms were not 'faked'.
Conclusions: The level of teaching was viewed as being inadequate and in general the students would value more. Despite this, the majority held positives attitudes towards FND. This study recommends integration of evidence-based teaching on FND into the medical school curriculum.

31. Understanding the current challenges in neuropsychiatry: modelling care and treatment.

Julie Moonga

Background Neuropsychiatry is an emerging field of medicine which links neurology and psychiatry. The convergence of neuropsychiatry comes in the wake of the advancements in neuroscience, genomics and neuroimaging including analytic technology. Data Analytics and Artificial intelligence (AI)brought a paradigm shift in the treatment of many diseases. However, innovation in health care technology often outpaces innovation in disease management. Healthcare decision makers are considering economic modelling in an effort to optimise patient services, care and treatment. Recently, NHS England proposed a Five Year Forward View to set out a future vision for a new model of care for patients. We seek to employ data intelligence to explore some of the issues associated with neuropsychiatry for a better understanding of disease management and decision making.
Objective/aim We aim to evaluate the whole neuropsychiatry pathways of care and treatment to optimise clinical outcomes across the NHS Trusts. We will assess the current status of primary care, secondary care and acute care to provide an economic model of neuropsychiatry. We will evaluate current methods of care to provide a better tool for neuropsychiatric disease management.
Methods We analyse econometrics data related to specific neuropsychiatric diseases. The data is gathered from NHS Digital, Office of National Statistics, and Public Health England. Pattern recognition, real time responsive solutions, optimisation, predictive models, whole system approaches and explanative models is used. Data is processed by cleaning, pulling, filtering, mining, and extraction to address the relevant questions.
Result Economic models in neuropsychiatry are important for providing optimum patient care and treatment. Limitations in quality management and pathway methodologies create gaps in the delivery of care. The implementation of new frameworks of care and service is an essential tool for disease management and clinical outcomes.

32. Loss of insight for cognitive symptoms in posterior cortical atrophy

S Ahmed, N Drummond, C Butler

Objective Lack of awareness or underestimation of cognitive deficits is a common feature in several neurodegenerative disorders, including Alzheimer's Disease (AD). Little is known about the profile of insight loss in atypical clinical variants of AD, such as posterior cortical atrophy (PCA). Insight loss is typically associated with negative outcomes for patients and their carers. Characterising the profile in PCA may therefore have important implications for management.
Methods Seventeen PCA patients and 21 healthy controls estimated their ability in five cognitive domains: visuospatial skills, visual imagery, attention, verbal memory and language, before undertaking objective neuropsychological testing in these domains. An

index of insight was obtained by calculating a discrepancy score between estimated and objective memory scores.
Results A repeated measures ANOVA using group (healthy controls and PCA) and insight domain (visuospatial, visual imagery, attention, verbal memory and language) revealed a significant interaction of group by domain (p<.001). PCA patients showed significantly less insight in all 5 domains compared to healthy controls (ps<.001). Comparing cognitive domains, PCA patients showed significantly less insight in visuospatial and visual imagery ability compared to all other domains (ps<.000).
Conclusions Loss of insight affects the core visuospatial deficits which define PCA more than other cognitive domains. The findings of this study are the first to investigate loss of insight in PCA.

33. Predicting psychiatric and cognitive outcomes after surgery for frontal lobe epilepsy

D Bindman, J Foong, P Thompson

Aims Previous research suggests that the side and site of epilepsy surgery may have important implications for post-surgical psychiatric and behavioural outcomes. Most of the published studies are in temporal lobe epilepsy surgery providing some evidence of frontal lobe dysfunction associated with postoperative mood disorder. Our study aims to identify potential clinical and cognitive predictors of postoperative psychiatric outcome following frontal lobe epilepsy surgery.
Methods A retrospective review of 78 consecutive patients who had undergone frontal lobe epilepsy (FLE) surgery at the National Hospital for Neurology and Neurosurgery between 1997 and 2016. Notes were scrutinised for pre- and postoperative psychiatric diagnoses, neuropsychological test findings, seizure data, histopathological data, and MRI and video-EEG data. Chi-square analysis and paired-sample t- tests will be used to examine the relationship between variables and to investigate differences between pre- and postoperative cognitive test scores.
Results Data analysis is in progress and final results will be presented in the conference poster. Preliminary data shows an incidence of post-operative psychopathology in the region of 29% with 14% of patients developing a de novo psychiatric condition within 48 months follow up. Preoperative non-executive function deficits were significantly associated with de novo psychopathology in our initial analysis ($\chi 2$ (1) = 4.99, p = .03).
Conclusions Initial analysis has identified aspects of pre-operative cognitive function that could act as predictors of post-operative psychiatric comorbidity. The results of the completed data analysis will be presented including data on the site of surgery and its relationship to post-operative psychopathology. Identification of patients at high risk of poor outcome will allow greater precision in patient selection and more pro-active treatment approaches in the post-operative period.

34. Korsakoff's psychosis: Is Polypharmacy a concern?

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Objectives/aims Case studies of all current Inpatients with Korsakoff's Syndrome in a 114 bed Psychiatric Hospital

Methods Audit and review of 114 individuals residing in a long stay Psychiatric Hospital during 2018. Medical records and Pharmacy Data base were reviewed to identify co-morbidities and medication exposure.

Results Thirteen individuals were identified with the formal diagnosis of Korsakoff's syndrome. Five were female with and age range from 51 years to 84 years. Eight were male with an age range from 55 to 84 years.

Patient Number	Gender	Age	Medications on Admission	Current Medications
1	M	65	Thiamine; Perindopril; Olanzapine; Ensure; Lorazepam; Ipratropium spray; Sodium Valproate; Trazadone	Perindopril; Bisoprolol; Lactulose; Ipratropium bromide; Sinemet; Macrogol; Sodium Valproate ; Folic Acid; Mirtazepine; Ferrous fumerate
2	M	58	Pyridoxine; Sertraline; Lorazepam; Lactulose; Movicol; Paracetamol; Cetrine; Zopiclone;Trazodone; Cyclizine	Fortisip: Sertraline; Artelac drops, Olanzapine
3	M	68	Haloperidol; Lorazepam; Fresubin; Cyclizine; Symbicort Turbohaler; Lansoprazole; Spiriva.	Sodium Valproate; Seretide; Tiotropium; Sertraline; Olanzapine; Pregabalin; Zopiclone; Fresubin; Calcichew D3.
4	F	51	Zopiclone; Trazadone; Lansoprazole; Lorazepam; Haldoperidol.	Tiotropium; Vit D3; Sertraline; Sodium Valproate; Mirtazapine; Topiramate; Aripiprazole; Atorvastatin; Haloperidol; Citalopram.
5	M	75	Quetiapine; Zolpidem; Lipitor; Glucophage; Asprin; Ramipril; Tamsulosin; Valproate; Diamicron	Atorvastatin; Aspirin; Quetiapine; Sertraline; Bupropion; Procol Nutricreme; Sodium Valproate
6	M	58	Multiitamin; Forticreme; Cubitan, Calogen; Olanzapine; Calcichew; Omeprazole; Alendronic Acid	Citalopram; Sodium valproate; Omeprazole; Prolia; Timolol/latanoprost drops; Calcichew D3; Olanzapine; Iron.
7	F	59	Mebeverine; Clonazepam;	Multivitamin; Vitamin C;

			Olanzapine; Haloperidol; Atrovastatin; Diazepam; Tegretol; Thiamine; Clonazepam; Lipitor.	Clonazepam; Atorvastatin; Carbamazepine; Sertraline; Olanzapine; Neurontin; Calcichew D3; Alendronic Acid; Haloperidol.
8	M	84	Quetiapine; Donepezil; Levetiracetam; Frumil; Asprin; Flurazepam;	Levetiracetam; Ketoconazole; Artelac drops; Vit D Macrogol; Tamsulocin/Duasteride; Sertraline.
9	M	55	Thiamine; Haloperidol; Trazadone; Fexofenadine; Omeprazole:	Folic Acid; Sertraline; Clonazepam; Mirtazapine; Pregabalin; Bupropion; Sinemet; Aripiprazole.
10	F	73	Thiamine; Zopiclone; Sodium Valproate; Clonazepam; Risperidone; Procyclidine; Mirtazepine.	Aspirin; Calcichew D3 Sertraline; Sodium Valproate; Furosemide;
11	F	84	Venlafaxine; Spironolactone-aldactone; Aspirin EC; Pantoprazole; Furosemide; Bisoprolol; Temazepam; Aldactone	Lansoprazole;Nutricreme; Olanzapine; Citalopram.
12	F	61	Quetiapine; Lorazepam; Sodium Valproate; Procyclidine; Carbamazepine; Mirtazapine; Risperidone; Phenergan; Thiamine; Citalopram.	Mirtazepine; Quetiapine; Sodium Valproate; Lorazepam.
13	M	58	Glicazide; Doxazosin; Asprin; Amlodipine; Aripiprazole; Simvastatin; Omeprazole; Metformin; Nebivolol; Lisinopril; Olanzapine.	Amlodipine; Nebivolol; Simvastatin; Aspirin; Sodium Valproate; Aripiprazole; Lansoprazole; Doxazosin; Sertraline; Lisinopril/Hydrochlorathiazide

Conclusions Careful consideration must continue to minimize polypharmacy. KS patients have complex comorbid health conditions. Careful consideration of potential interactions, disabling side effects, potential adverse reactions and collaboration with a Pharmacist is invaluable.

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35. The Arc de Siècle- An examination of attitudes to hysteria in twentieth-century medicine, through the eyes of Norman Dott and his pupils

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Objectives Hysteria was once a topic of research for leading neurologists; however this interest faded over the course of the twentieth century. Little has been written about the presentation and management of functional disorders in the post Charcot period, and some have gone so far as to suggest that the patients 'disappeared'. This project aims to shed light on how Edinburgh neurologists interacted with and managed this cohort during the period from 1930-1970.

Methods The Lothian Health Services Archive holds 28,000 case files written by Norman Dott CBE, the first chair of Neurosurgery in Scotland, and the department he built around him, with cases spanning the years between 1930-1970. Cases pertaining to hysteria or hypochondriasis were analysed, recording demographics, symptoms, diagnostic and management processes, and evidence for any attitude or opinion exhibited by the physicians. Retired neurologists from Edinburgh and elsewhere also provided oral histories on how they interacted with this group.

Results 209 cases were analysed, of which 178 were relevant, and 100 of which included a diagnosis of hysteria. Of these 100, it is of note that 42 were referred to psychiatry. The majority of the remaining patients were given advice or reassurance (48).

Conclusions Hysteria in Dott's department was both a diagnosis based on positive findings of inconsistency, and a personality trait. Although there is evidence of a negative sentiment towards functional patients, there is equal evidence of sympathetic responses, and it is likely that neurologists of the mid twentieth century were often less cautious and more candid in their remarks. Whilst the management of hysteria was not seen as a neurologist's job, patients were regularly referred to psychiatry, signifying an acceptance of the legitimacy of the condition.