2. Hoeritzauer I, Pronin S, Carson A, Statham P, Demetriades AK, Stone J. The clinical features and outcome of scan-negative and scan-positive cases in suspected cauda equina syndrome: a retrospective study of 276 patients. *J Neurol* 2018;**265**:2916–26.
3. Leue C, Kruimel J, Vrijens D, Masclee A, Van Os J, Van Koeveeringe G. Functional urological disorders: A sensitized defence response in the bladder-gut-brain axis. *Nat. Rev. Urol.* 2017;**14**:153–63.

12 FEIGNED OR FUNCTIONAL?

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

10.1136/jnnp-2019-BNPA.12

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.

13 HYPERMOBILITY AND AUTONOMIC DYSFUNCTION: INSIGHTS FROM BENCH TO BEDSIDE

Jessica Eccles. *Sussex Partnership NHS Foundation Trust and Brighton and Sussex University Hospitals NHS Trust*

10.1136/jnnp-2019-BNPA.13

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 autonomic, 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.

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 Hypermobility 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.

14 THE LANGUAGE DISORDER IN SEMANTIC DEMENTIA: DOES IT MATTER WHICH LANGUAGE YOU SPEAK?

Karalyn Patterson. *University of Cambridge*

10.1136/jnnp-2019-BNPA.14

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.

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.

REFERENCES

1. Fushimi, T, Komori, K, Ikeda, M, Lambon Ralph, MA & Patterson, K. (2009). The association between semantic dementia and surface dyslexia in Japanese. *Neuropsychologia*. 2009;47:1061–1068.
2. Patterson, K & Fushimi, T. Organisation of language in the brain: Does it matter which language you speak? *Interdisciplinary Science Reviews* 2006;31:201–216
3. Ratnavalli, E, Narayana, J & Patterson, K (2019). Differential deterioration of languages in bilingual patients with semantic dementia: Implications for language representation in bilinguals. Under review.

Members' Platform Presentations

15 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. *Research Fellow, Institute of Population Health, Trinity College Dublin*

10.1136/jnnp-2019-BNPA.15

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.

16 EFFECT OF METHYLPHENIDATE ON RISK PREFERENCE IN ATTENTION DEFICIT HYPERACTIVITY DISORDER

¹Alekhyia Mandal*, ²Arjun Sethi, ²Neil A Harrison, ¹Valerie Voon. ¹Department of Psychiatry, University of Cambridge; ²Department of Psychiatry, University of Sussex

10.1136/jnnp-2019-BNPA.16

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