



**Royal College of Psychiatrists  
Faculty of Neuropsychiatry Annual  
Conference**

**18 September 2020**

**Conference Booklet**



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Dear colleague

It gives us great pleasure to welcome you to our Faculty of Neuropsychiatry's first virtual conference. You will appreciate how challenging the last few months have been. Our original plans for a two-day face-to-face programme had to be radically modified to accommodate our 'new normal'. As a Faculty, we were determined not to let colleagues down and have worked hard to be able to deliver a high quality conference that would support colleagues' education and maintain the success that has been built up over recent years. Despite the complications that came with the COVID-19 pandemic, the Faculty was also keen to turn some of the challenges into real opportunities and extend our audience base to include even more countries from around the world.

As one of the first College Faculties to run a virtual conference, we believe we have curated what promises to be a highly educational and stimulating day. As you will see from the programme, the conference will cover a number of topics of relevance to clinicians and researchers alike.

We are immensely grateful for your ongoing support and wish you a valuable and enjoyable day.

Dr George El-Nimr  
Academic Secretary

Dr Mike Dilley  
Faculty Chair

## **General Information**

### **Accreditation**

This conference is eligible for up to 6 CPD hours on Friday 18 September, subject to peer group approval.

### **Certificates**

Certificates of attendance will be emailed to delegates after the conference.

### **Feedback**

A detailed online feedback form can be found by visiting <https://www.surveymonkey.co.uk/r/N9DGP5W>

All comments received remain confidential and are viewed in an effort to improve future meetings.

If you wish to tweet about the conference use @RCPsych @rcpsychNeuro #NeuroConf20

### **Posters**

Poster viewing is available throughout the conference using the following [link](#)

### **Conference Resources**

Please see the following link to access the [conference resources](#). Webpage.

## **Presentation abstracts and biographies**

(Listed by programme order)

Abstracts and biographies not included here were not available at the time of going to print.

### **Welcome from the Faculty Chair**

Dr Mike Dilley

**Dr Mike Dilley** is a Consultant Neuropsychiatrist in neurorehabilitation and has specialist interests in traumatic and acquired brain injury (including hypoxic-ischaemic brain injury) and severe and complex functional neurological disorder that requires multidisciplinary inpatient rehabilitation.

He is the current Faculty Chair of the Neuropsychiatry.

### **Welcome from the President**

Dr Adrian James

**Dr Adrian James** was elected President of the Royal College of Psychiatrists in 2020.

Adrian is Consultant Forensic Psychiatrist at Langdon Hospital in Dawlish, Devon. He is a former Medical Director of Devon Partnership NHS Trust and founding Chair of the School of Psychiatry at the Peninsular Deanery (2006-2008).

He was the elected Chair of the South West Division of the Royal College of Psychiatrists (2007-2011) and sat on the College Council in this capacity. In 2010 he was appointed Chair of the Westminster Parliamentary Liaison Committee of the Royal College of Psychiatrists.

He was Clinical Director for Mental Health, Dementia and Neurology, working for NHS England South West (2013-2015, interim from 2012-13). He has also acted as a Reviewer and Clinical Expert for the Healthcare Commission and its successor organisation the Care Quality Commission.

He has chaired expert review groups on Integrated Care Systems, Cannabis, Prevent and Learning from Deaths. In addition, he set up the Quality Improvement Committee and the Workforce Wellbeing Committee at the College.

His priorities as President are:

- establishing a pathway to parity for mental health services
- equality and diversity
- sustainability
- workforce wellbeing

## **Plenary 1: Autism Spectrum Disorder**

### **Psychiatric co-morbidity in people with autistic spectrum disorder**

Dr Quinton Deeley

Dr Deeley is a member of the Royal College of Psychiatrists. He undertook medical training at Guys and St Thomas' Hospitals before training in psychiatry on the Maudsley Hospital training scheme. He has a PhD in the cognitive neuroscience of autism and psychopathy. He also has a Bachelor of Arts degree in Theology and Religious Studies from Cambridge University.

### **Autistic spectrum disorder in females**

Dr Michael Craig

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition that is 2-5 times more common in males than in females. It is still poorly understood what causes the 'male-biased' prevalence of ASD. However, aetiological models suggest that the biological female phenotype in general displays a lower vulnerability to ASD than the male phenotype.

The current presentation will review a) evidence for normative variability in the biological phenotype of neurotypical males and females; and b) the putative role for sex hormones and sex chromosomes in this variability.

**Dr Michael Craig** is a Reader in Translational Reproductive and Neurodevelopmental Sciences (TRANS), and has set up and developed the MSc in Clinical Neurodevelopmental Sciences, at the IoPPN, King's College, London. He is Clinical Lead of the *National Autism Unit* at the Bethlem Royal Hospital and the *National Female Hormone Clinic* at the Maudsley Hospital.

His main research interests are a) the effects of the *in utero* environment on neurodevelopment; b) gender differences in neurodevelopment and ageing; and c) neurodevelopmental aspects of antisocial behaviour. His research is currently funded by MRC(UK), NIMH(USA) and the European Commission (EC).

### **Plenary 2: Novel Neuro Imaging Markers**

#### **Positron emission tomography studies on peripheral and central immunity in healthy volunteers and clinically depressed cohorts**

Professor Federico Turkheimer

The relationship between peripheral and central immunity and how these ultimately may cause depressed behavior has been the focus of a number of imaging studies conducted with Positron Emission Tomography (PET). These studies aimed at testing the immune-mediated model of depression that proposes a direct effect of peripheral cytokines and immune cells on the brain to elicit a neuroinflammatory response via a leaky blood-brain barrier and ultimately depression. However, studies conducted so far using PET radioligands targeting the neuroinflammatory marker 18 kDa translocator protein (TSPO) in patient cohorts with depression have demonstrated mild inflammatory brain status but no correlation between central and peripheral immunity.

To gain a better insight into the relationship between heightened peripheral immunity and neuroinflammation, we estimated blood-to-brain and blood-to-CSF perfusion rates for two

TSP0 radiotracers collected in two separate studies, one large cross-sectional study of neuroinflammation in normal and depressed cohorts and a second study where peripheral inflammation in healthy controls was induced via subcutaneous injection of interferon (IFN)- $\alpha$ . In both studies we observed a consistent negative association between peripheral inflammation, measured with c-reactive protein P (CRP), and radiotracer perfusion into and from the brain parenchyma and CSF. Importantly, there was no association of this effect with the marker of BBB leakage S100b, that was unchanged.

These results support a different model of peripheral-to-central immunity interaction whereas peripheral inflammation causes a “stiffening” of the healthy BBB with consequent reduction of small molecule trafficking to and from the blood into the brain and CSF. This effect, on the long term, is likely to disrupt brain homeostasis and induce depressive symptoms. Moreover, given the molecular similarity between the TSP0 ligands and antidepressants, this phenomenon may underlie treatment resistance in depressive cohorts with heightened peripheral status.

**Professor Federico Turkheimer** is Chair in Neuroimaging at King' College London. The aim of his work is to develop in-vivo imaging markers of brain function with particular focus on psychiatric disorders. His group uses Positron Emission Tomography (PET) and Magnetic Resonance Imaging (MRI) to model brain physiology in health and disease. Presently the work is focused on brain immunity and its relationship with the peripheral immune system, brain oxidative metabolism and glymphatic function and their role in depression and psychosis. The group has also great interest in constructing precise models of drug pharmacokinetic action and pharmacodynamics to quantitatively evaluate treatment effects.

### **In vivo neuroimaging to index drug effects: mechanisms and methods**

Professor Mitul Mehta

Pharmacodynamic effect of drugs can be assessed in vivo in humans with neuroimaging. Emission tomography combined with selected ligands can index the uptake and binding at target sites, but is limited to one ligand per study. In contrast, during single studies MRI is effectively multimodal. Markers of brain function sensitive to drug modulation include blood flow and perfusion, functional imaging with tasks, brain connectivity, tracking of activity for rapidly acting drugs, and spectroscopy for brain metabolite levels. These methods can be applied to healthy volunteers, combined with drug models of dysfunction, and patient groups. These methods are particularly suitable to translate evidence from experimental animals, validate theory and provide early indicators of potential efficacy. Examples of utility are glutamatergic models of dysfunction to assess novel therapies, deficits in inhibition, emotional recognition and working memory, and classification of drug with machine learning. Significant challenges exist with MRI methodology such as the limited evaluation of reliability, the modelling of confounds, valid, stratification of individuals and bridging the gap between the modulation of brain systems and clinical outcome. Numerous examples exist to demonstrate the success and future potential of MRI methodologies in the drug development process, which, if correctly applied, can provide a principled basis for some treatment options over others, accelerating the delivery of novel therapeutics for those in need.

**Professor Mitul Mehta** completed his PhD at the University of Cambridge before training in Neuroimaging as an MRC Fellow at Imperial College and then as a Wellcome Trust



Awardee at King's College London. The Neuropharmacology group has developed assays for brain modulation, methods for testing drug effects on brain function and applied these to existing and novel compounds in healthy volunteers and patient groups. Recently, Prof. Mehta was appointed Director of the Centre for Innovative Therapeutics in order to further interactions with industry to accelerate diagnostic and therapy development for patient benefit.

### **Plenary 3: Neuropsychiatry of Parkinson's Disease**

#### **Understanding dementia and hallucinations in Parkinson's disease**

Dr Rimona Weil

Dementia is common and debilitating in Parkinson's disease, affecting half of all patients within 10 years of diagnosis; and hallucinations are a harbinger of Parkinson's dementia, being the strongest predictor for nursing home admission. But the mechanisms underlying dementia and hallucinations are still unclear. Evidence is starting to emerge that patients with Parkinson's disease who have visual dysfunction develop dementia more rapidly. This talk will present some of our recent work using advanced neuroimaging to detect early changes in Parkinson's dementia, and shedding light onto mechanisms for hallucinations in Parkinson's disease.

**Dr Rimona Weil** is a Consultant Neurologist at the National Hospital for Neurology and Neurosurgery and a Clinician Scientist. She runs clinics that manage patients with Parkinson's dementia and Dementia with Lewy Bodies. She studied medicine at Downing College, Cambridge and at UCL, and undertook a PhD in neuroscience at the Wellcome Trust Centre for Neuroimaging, UCL. She was awarded a post-doctoral UCL Excellence Fellowship to study visual changes in Parkinson's disease. Currently, she runs a Wellcome-funded longitudinal study on predictors of dementia in Parkinson's disease, using neuroimaging, retinal and cognitive markers.

#### **Parkinson's disease: mood, psychosis, impulse control and deep brain stimulation**

Professor Eileen Joyce

Parkinson's disease is now recognised as a neuropsychiatric syndrome and not only a movement disorder. Psychological and behavioural symptoms are common and can present at all stages of the illness. They can be understood with reference to current knowledge of the underlying neuropathology, how it spreads throughout the CNS, and how this affects neural systems involved, not only in motor function, but also in emotion and reward processing as well as impulse control. This presentation will provide an update on the neuropsychiatric presentations of PD and their management. Outstanding gaps in service provision will be discussed.

**Eileen Joyce** is a Professor of Neuropsychiatry at The Institute of Neurology, University College London. Her current research focuses on interventions for neuropsychiatric disorders such as schizophrenia, OCD and Tourette's syndrome and their mechanisms of action. Her clinic work includes the management of complex neuropsychiatric disorders such as conversion disorder, Parkinson's disease and Tourette's syndrome. She is involved in clinical studies of deep brain stimulation and neurosurgical ablation for severe mental illness.

Professor Joyce obtained her first degree in experimental psychology and PhD in dopamine psychopharmacology from the University of Cambridge. She then went on to study medicine also at Cambridge. She trained in psychiatry at the Bethlem and Maudsley Hospitals and spent several years as a research worker at the Institute of Psychiatry, where she was a Wellcome Trust Lecturer in Mental Health. This was followed by time at the USA National Institutes of Health. Before moving to UCL/UCLH, she was Professor of Neuropsychiatry at Imperial College London before moving to UCL.

She is past Chair of the Faculty of Neuropsychiatry, Royal College of Psychiatrists.

## **Plenary 4: Human Decision Making: The Science**

### **Neurobehavioural substrates of addiction: a translational perspective**

Professor Jeffrey Dalley

Compulsion is a core component of addiction, defined by drug seeking and use that persists despite growing personal harm. However, for reasons that are not well understood, only a small proportion of individuals exposed to addictive substances develop compulsive drug use. Studies in humans indicate a high co-morbidity of addiction with the traits of impulsivity, novelty-/sensation-seeking and cognitive inflexibility. However, because both pre-existing and drug-induced abnormalities in the brain may contribute to compulsive drug use, it is not always possible to unambiguously define brain vulnerability markers of addiction from research in humans alone. This talk considers a broader translational approach to investigate neural vulnerability mechanisms in addiction, including high resolution magnetic resonance imaging in rodents, behavioural genetics, and computational neuroscience.

**Jeff Dalley** is a Professor in the Department of Psychology and the Department of Psychiatry at Cambridge University. He is also the Director of Studies in Neuroscience and Psychology at St Catharine's College in Cambridge as well as the Editor-in-Chief of *Brain and Neuroscience Advances*, a society journal owned by the British Neuroscience Association. His research spans the fields of behavioural and cognitive neuroscience, psychopharmacology, and the translation of basic advances in neuroscience to a range of brain disorders, including drug addiction.

### **The readiness potential in the debate about conscious free will**

Dr Aaron Schurger

The readiness potential (RP) is a slow buildup of neural activity in pre-motor areas that precedes the onset of a self-initiated movement by up to one second or more. Contemporary neuroscientific accounts of human volition lean heavily on the RP as a temporal marker of the brain processes leading up to movement onset, even though the precise nature of the RP remains unclear. Libet (1983) used the RP to argue against the possibility of conscious free will because the RP is apparent hundreds of milliseconds before subjects report having been aware of the conscious decision to act. Although the precise relationship between the RP and conscious volition remains highly controversial, the interpretation of the RP as a signature of "planning and preparation of volitional acts" has held strong for decades. However, recent evidence has begun to cast doubt on that interpretation and at least one alternative account of the RP has begun to gain traction. I

will discuss this alternative account of the RP, and its impact on our understanding of human volition. A small change in the way that we interpret the RP can have a profound impact on how we interpret data that seem to bear on the question of conscious volition. Whatever role the RP ends up playing in action initiation, I will argue that we need to clearly understand the nature of movement-preceding neural activity before we can conclude anything about conscious volition. The data that we have to date suggest that the conscious feeling of intending to act is cobbled together from neural activity that happens before, during, and just after action initiation.

Coming from a background in computer science, **Aaron Schurger** earned his PhD in psychology and neuroscience at Princeton University under the guidance of Jonathan D. Cohen and Anne Treisman. After that he joined the research team of Stanislas Dehaene at the NeuroSpin research center near Paris, France as a post-doc, and then as a senior researcher working with Olaf Blanke and José del R Millán at the EPFL in Lausanne, Switzerland. He currently holds a dual appointment as principal investigator with the French National Institute for Health and Medical Research (INSERM), and as assistant professor in the Department of Psychology and the Brain Institute at Chapman University in California. Schurger's research focuses on the neural signatures of subjective experience and the neural antecedents of self-initiated movement. In 2013 Schurger was awarded the William James Prize from the Association for the Scientific Study of Consciousness (ASSC) and in 2015 was awarded the BMI-Kaloy prize from the Kaloy Foundation for his work on the influence of spontaneous fluctuations in brain activity on self-initiated movement. Schurger uses a variety of techniques in his research including behavioral psychophysics, neuroimaging, computational modeling, machine learning, and brain-computer interfaces.

## **Poster exhibition**

(alphabetically by surname)

### **1. Acute psychotic presentation in syphilis- the great imitator is back**

***Dr Bethan ap Rees; Dr Mudasir Firdosi***

Neurosyphilis presenting with psychiatric symptoms is now an uncommon diagnosis since the introduction of antibiotic therapy. This case is a patient with a diagnosis of Human Immunodeficiency Virus (HIV) and neurosyphilis who presented with psychosis and responded well to treatment with antibiotics and antipsychotics.

We discuss the challenges in management of psychosis in neurosyphilis due to the lack of guidelines, and also discuss the importance of psychiatrists including neurosyphilis in their diagnosis of patients presenting in similar ways, and offering serological testing for syphilis in such patients. This is most important within the men who have sex with men (MSM) population and those with a diagnosis of Human Immunodeficiency Virus (HIV) due to the increased prevalence of syphilis within these populations.

## **2. Sensory symptoms in body-focused repetitive behaviours and premonitory urges to tic in Tourette syndrome: an overlap?**

**James Badnoch\***; Tamara Searle\*; Iona Watson\*; Andrea E. Cavanna (joint lead authors)

Tourette syndrome (TS) is a neurodevelopmental condition characterised by multiple motor tics plus at least one phonic tic. Sensory symptoms are thought to play a key role in the clinical phenomenology and pathophysiology of TS, as most patients report premonitory urges driving tic expression. Interestingly, sensory symptoms have also been reported in other conditions characterised by repeated behaviours. This narrative review explores the nature of sensory symptoms reported by patients with body focussed repetitive behaviours (BFRBs) and restless legs syndrome (RLS) in comparison to TS. A sense of mounting inner tension and reinforcement mechanisms driven by gratification and relief on expression of the tic or behaviour appear to be implicated across all conditions. Moreover, subjective urges can be temporarily suppressed by patients with TS and selected BFRBs, whereas patients with RLS tend to report dysaesthesia more frequently than a suppressible urge to move. The observed similarities in the clinical phenomenology of the urges across these conditions raise the possibility of a comparable underlying pathophysiology. Preliminary findings suggest a possible shared role for the insula, the basal ganglia (putamen), and the posterior cingulate cortex. An improved understanding of the pathophysiological aspects shared by repetitive behaviours driven by sensory symptoms and premonitory urges to tic could allow for the development of more effective treatment options.

### **3. Tics in patients with encephalitis**

**James Badenoch\***; Tamara Searle\*; Iona Watson\*; Andrea E. Cavanna (\* joint lead authors)

Movement disorders have been described in the context of different types of encephalitis. Among hyperkinetic manifestations, tics have sporadically been reported in cases of encephalitis resulting from a range of aetiologies. This review aimed to assess the prevalence and characteristics of tics in patients with encephalitis. We conducted a systematic literature review of original studies on the major scientific databases, according to the standards outlined in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. In addition to the established association between tics and encephalitis lethargica, our literature search identified reports of tics in patients with immune-mediated pathologies (including autoimmune encephalitides affecting the N-methyl-d-aspartate receptor, voltage gated potassium channels, and glycine receptors) and infective processes (ranging from relatively common viral pathogens, such as herpes simplex, to prions, as in Creutzfeldt-Jakob disease). Tics were most commonly reported in the post-encephalitic period and involvement of the basal ganglia was frequently observed. The association of new-onset tics and encephalitis, in the background of other neuropsychiatric abnormalities, has practical implications, potentially improving the detection of encephalitis based on clinical features. Future research should focus on the categorisation and treatment of hyperkinetic movement disorders associated with encephalitis.

#### **4. Pharmacotherapy for tics in adult patients with Tourette syndrome and other tic disorders**

**James Badenoch**; *Andrea E. Cavanna*

Background: Tourette syndrome (TS) and persistent motor/vocal tic disorders are neurodevelopmental conditions characterised by the chronic presence of motor and/or vocal tics. Patients with TS often present with co-morbid disorders, especially attention-deficit and hyperactivity disorder (which tends to improve after childhood), and obsessive-compulsive disorder (which can persist in adulthood). We set out to explore pharmacotherapy for tics in adult patients with TS and persistent motor/vocal tic disorders, as well as its relationship with the presence of co-morbid conditions.

Methods: We retrospectively reviewed the clinical characteristics and pharmacotherapy of 192 adult patients with TS (n = 187), persistent motor tic disorder (n = 3) and persistent vocal tic disorder (n = 2) attending a specialist clinic in the UK.

Results: Anti-dopaminergic medications (n = 65) and alpha-2-agonists (n = 50) were the most commonly prescribed pharmacotherapy for tic management. A sub-group analysis revealed that co-morbid obsessive-compulsive disorder and sub-threshold obsessive-compulsive behaviours were significantly more common in patients treated with anti-dopaminergic medications than patients taking alpha-2-agonists ( $p = 0.013$  and  $p = 0.047$ , respectively).

Conclusions: The use of pharmacotherapy options for tic management observed at a specialist clinic for adults with TS reflects guideline recommendations. We found that the presence of co-morbid obsessive-compulsive disorder/behaviours correlates with the choice of anti-dopaminergic medications over alpha-2-agonists, in line with available evidence on the efficacy of anti-dopaminergic medications for the treatment of specific tic-related behavioural symptoms.

## **5. Psychological distress after subarachnoid haemorrhage: a systematic review and meta-analysis**

**M Bartlett<sup>1</sup>, D Bulters<sup>2</sup>, R Hou<sup>1,1</sup>** *Department of Psychiatry within Medicine, University of Southampton, Southampton, UK; <sup>2</sup> Department of Neurosurgery, University Hospital Southampton, Southampton, UK*

**Background:** Subarachnoid haemorrhage (SAH) is a devastating condition, with high rates of death and disability. Psychological distress is a common, long-term complication of SAH. However, the prevalence rates of the condition vary widely across studies and there has been no systematic review and meta-analysis conducted to evaluate available evidence.

**Aims:** The primary objective was to determine the pooled prevalence of psychological distress including anxiety and depression in patients after SAH. The secondary objective was to evaluate risk factors for psychological distress after SAH.

**Methods:** Multiple databases including EMBASE, Medline, PsychInfo, and Web of Science were searched for publications before 1st December 2019. Screening, data extraction, and quality assessment were undertaken following the PRISMA guidelines for preferred reporting of systematic reviews and meta-analysis. The PICOSS framework was used to guide the search strategy. Data were extracted and analysed separately for anxiety and depression at short and long term follow up time points. A random-effects model was used to calculate pooled prevalence rates. Meta-analysis was conducted using Comprehensive Meta-analysis software. The review protocol was registered on PROSPERO (ref: CRD42020182594).

**Results:** 42 studies involving 5,950 patients reporting anxiety and 64 studies involving 8,834 patients reporting depression were included in this review. The pooled short term (<3 years) and long term (≥3 years) prevalence rates of anxiety were 31.4% (95% CI: 23.6%, 40.4%) and 40.4% (95% CI: 31.6%, 49.8%), respectively, whereas the pooled short term and long term prevalence rates of depression were 25.2% (95% CI: 17.8%, 34.5%) and 35.8% (95% CI 28.6%, 43.6%), respectively. Gender and pre-existing psychiatric conditions were identified as potential risk factors for psychological distress after SAH.

**Conclusions:** This is the first systematic review and meta-analysis evaluating psychological distress after SAH. The findings highlight the need for appropriate assessment and management of these comorbid conditions in individuals after SAH. Further research is warranted to examine underlying mechanisms such as the neuroinflammatory hypothesis and identify modifiable risk factors which may offer new intervention approaches to benefit SAH patients.



## **6. Beta-frequency electrophysiological bursts: BOLD correlates and relationships with psychotic illness**

**Paul M. Briley;** *Elizabeth B. Liddle; Molly Simmonite; Marije Jansen; Thomas P White; Vijender Balain; Lena Palaniyappan; Richard Bowtell; Karen J. Mullinger & Peter F. Liddle*

**Background:** The post movement beta rebound (PMBR) is a transient increase in power in the beta frequency band (13-30 Hz), recorded with methods such as electroencephalography (EEG), following the completion of a movement. PMBR size is reduced in patients with schizophrenia and inversely correlated with severity of illness. PMBR is also inversely correlated with measures of schizotypy in non-clinical groups. Therefore, beta-band activity may reflect a fundamental neural process whose disruption plays a key role in the pathophysiology of psychotic illness. Recent work has found that changes in beta power reflect changes in the probability-of-occurrence of transient bursts of beta-frequency activity. Understanding the generators of beta bursts could help us to unravel the pathophysiology of psychotic illness and thus identify novel treatment targets.

**Methods:** EEG data were recorded simultaneously with BOLD data measured with 3T functional magnetic resonance imaging (fMRI), whilst participants performed an n-back working memory task. We included seventy-eight participants – 32 patients with schizophrenia, 16 with bipolar disorder and 30 healthy controls. Beta bursts were identified in the EEG data using a thresholding method and burst timings were used as markers in an event-related fMRI design convolved with a conventional haemodynamic response function. A region of interest analysis compared beta-event-related BOLD activity between patients and controls.

**Results:** As expected, the PMBR, measured in the EEG data, was smaller in patients than controls. In patients, smaller PMBR size was associated with poorer functioning (as measured by GAF and SOFAS scores), poorer performance on a cognitive task (digit symbol substitution), and greater persistence of symptoms of disorganisation. The positive BOLD correlates of beta bursts included inferolateral pre/post-central gyri and superior and transverse temporal gyri, as well as superior pre/post-central gyri and left cerebellum. The negative BOLD correlates of beta bursts differed between task and rest blocks and represented a phasic inhibition of areas that were tonically active during the respective block. Despite the smaller PMBR in patients measured with EEG, BOLD responses to beta bursts were significantly greater in both amplitude and extent in patients than controls.

**Discussion:** The most prominent clusters of BOLD activity associated with beta bursts included areas involved in the motor execution and sensory processing of speech sounds and language. Given the nature of the n-back working memory task, which involves remembering the names of previously-seen letters and thus storing their phonological representations, these results are consistent with a recent theory that beta bursts mediate reactivation of latent content-specific representations in working memory. The increased BOLD response associated with beta bursts in patients, despite reduced PMBR, could reflect inefficiency of the cortical communication underpinned by beta bursts, or it may indicate that the content-specific representations re-activated by beta bursts are less reliably defined in psychosis. This may underlie the working memory deficits and loosening of associations characteristic of psychotic illness.

## **7. Investigating the dopaminergic mechanisms underlying motivation using designer receptors exclusively activated by designer drugs (DREADDS)**

**Ankit Chadha**; Amol Joshi; Chiara Toschi; Jeffrey W. Dalle

The roles of dopamine are of great relevance in modern psychiatry, with the neurotransmitter being implicated in a plethora of functions not limited to the expression of impulsivity and motivational responding. In recent years, the relationship between motivation and Ventral Tegmental Area (VTA) dopamine (DA) neuron activity has been enhanced by an incredibly transformative tool in behavioural neuroscience research known as chemogenetics. This study aimed to validate the use of chemogenetics in rats by suppressing activity in VTA-dopamine (VTA-DA) neurons projecting to the medial nucleus accumbens shell (AcbSh) and observing the resultant performance changes in locomotor and progressive-ratio paradigms. It also sought to correlate these findings with histological evidence gathered from immunohistochemical staining of mCherry, TH and c-Fos, paving the way for the use of DREADDs in future studies exploring the role of dopamine in impulsivity. The principal findings were that while chemogenetics substantially reduced locomotor activity, there was an absence of a significant change in performance in the PR paradigm. These effects were thought to be attributable to the lack of dopamine in the medial VTA-AcbSh pathway, along with inadvertent targeting of an inhibitory VTA-AcbSh GABA-ergic system. Overall therefore, the study concludes that future studies in the field of impulsivity may employ DREADDs technology as a suitable tool to modulate DA neuron firing in richly-innervated target pathways.

## **8. Associations between baseline cortisol and trajectory of symptom improvement in depressed adolescents receiving psychological therapy.**

*Ankit Chadha; Paul Wilkinson; Ian Goodyer*

A recent meta-analysis of small and heterogenous studies suggested high levels of peripheral circulating cortisol may predict poor response to psychological therapy. We tested the association between morning (n=112) and evening (n=166) salivary cortisol levels and self reported symptom response to psychological therapies in depressed adolescents engaged in a randomized controlled trial. High evening but not morning cortisol levels were associated with a slower initial response to treatment ( $p=0.022$ ). This effect was not significantly different across the three therapies. High evening cortisol may impair a depressed adolescent's ability to use psychological therapy.

## **9. A single administration of 'microbial' D-alanine to healthy volunteers augments reaction to negative emotions: a comparison with D-serine**

*Liliana P Capitaó; **Jessica Forsyth**; Mia A Thomaidou; Mark D Condon; Catherine J Harmer; Philip WJ Burnet*

Background: Activation of the glutamate N-methyl-D aspartate receptor with its co-agonist D-serine has been shown to improve subjective mood in healthy volunteers. D-alanine is another potent N-methyl-D-aspartate receptor co-agonist which arises from the natural breakdown of host gut microbes, and is predominantly sequestered in the pituitary. This raises the question as to whether D-alanine may also impact emotion, and whether this influence is through the neuroendocrine stress response.

Aims: The current study explored the effects of D-serine and D-alanine on emotional processing, cognition and the levels of the stress hormone cortisol in healthy volunteers.

Methods: In a double-blind, placebo-controlled randomised study, participants (n=63) received a single oral dose of either D-serine, D-alanine (60 mg/kg) or placebo and then performed the Emotional Test Battery and N-back task (two hours post-administration) and provided saliva samples at fixed intervals.

Results: Subjects administered with D-alanine were faster at identifying facial expressions of fear, surprise and anger, and at categorising negative self-referential words. Participants on D-alanine also showed a trend to recall more words than placebo in a memory task. D-serine did not have any meaningful effects in any of the tasks. Neither amino acid had a significant effect on salivary cortisol or working memory.

Conclusion: This study is the first to suggest that D-alanine can modulate emotional cognitive processing after a single dose. The lack of findings for D-serine nevertheless contrasts a previous study, emphasising a need for further investigation to clarify discrepancies.

## **10. Reducing risk of falls and fractures in old age psychiatry patients: Are we checking vitamin D?**

**Renate Fromson;** *Ashling Ramdin*

Introduction: Vitamin D is essential in maintaining bone integrity and function of the neuromuscular system (1). Its deficiency is a significant factor in metabolic bone disease (1) and is linked to an increased risk of falls and fractures (2). The impact on morbidity and mortality in the elderly population contributes significantly to healthcare costs. NICE advises treating vitamin D deficiency (<25nmol/L) and insufficiency (25-50nmol/L with additional risk factors) (3). Screening for medical comorbidities in psychiatric patients is well practised; however vitamin D is not routinely assessed on old age psychiatry wards.

Methods: Retrospective study of patients admitted between June 2019 and January 2020 to Garnet Ward, Highgate Mental Health Centre. Demographics and blood results were recorded using hospital software. Patients were stratified by falls risk: low (no previous falls, no co-morbidities increasing falls risk), medium (one previous fall, no co-morbidities) and high ( $\geq 1$  previous fall,  $\geq 1$  co-morbidity). Vitamin D levels were classified as deficient or insufficient based on NICE guidelines.

Results: 44 patients were admitted; 25 female, with an age range of 56-95. 26 patients (59%) were over the age of 70. 19 were high falls risk, 10 medium and 15 low. 19 patients (43.2%) had vitamin D checked on admission. The range was 13-131 nmol/L. 9 patients (47%) had vitamin D < 25, 6 (32%) between 25-50 and 4 (21%) > 50. Everyone with a vitamin D of < 25 or 25-50 was prescribed colecalciferol. Conclusion Our patient demographic is elderly and co-morbid, with a majority at high falls risk. Only 43% of patients had Vitamin D checked on admission. Where it was checked and was low, colecalciferol was prescribed. We advise including Vitamin D as part of routine admission bloods for every patient admitted to the ward.

Further investigations could include a routine FRAX score.

References:

1. <https://www.ncbi.nlm.nih.gov/pubmed/8642450>
2. <https://asbmr.onlinelibrary.wiley.com/doi/full/10.1359/jbmr.2002.17.5.891>
3. <https://cks.nice.org.uk/vitamin-d-deficiency-in-adults-treatment-and-prevention#!scenario>

## **11. Electroencephalography (EEG) Signatures of Induced Auditory Verbal Hallucinations (AVH)**

**Danish Hafeez;** *Ms Isabel O'Rourke, Masters Student; Dr Bo Yao, Lecturer; Dr Andrew Marshall, Consultant Neurophysiologist*

Introduction: Auditory hallucinations remain the most common type of hallucination in schizophrenia, but the mechanisms underlying it remain largely misunderstood. Research indicates that misattribution of inner speech may explain auditory hallucinations and this attribution process. This study's objective was to induce auditory verbal hallucinations (AVH) in healthy participants.

Method: AVH were induced using a speech detection paradigm simultaneously coupling the auditory and visual phrase "The grass is green" in white noise. Sixty participants completed the speech detection paradigm (Experiment 1) and those in the top half of AVH rate distribution were invited to repeat the paradigm with EEG (Experiment 2).

Results: Experiment 1 showed AVH rates in healthy participants are greater than 0%. EEG during experiment 2 showed increased evoked power during AVH compared to correctly recognising the absence of auditory stimuli in gamma (42-60Hz) and low frequency wave (3-5Hz) oscillations. In addition, an increase in inter-trial phase coherence was found in the beta (20-27Hz) and gamma (43-53Hz) frequency bands.

Conclusion: This study reaffirmed that AVH can be induced in healthy participants. The increased evoked power during AVH is indicative of inner speech processing consistent with misattribution of inner speech models. The increased phase locking is suggestive of predictions facilitating inner speech attribution. Future studies could examine AVH in a greater range of hallucination-proneness, counterbalancing speech recordings of both genders.

## **12. Evaluation of an alcohol-related brain injury (ARBI) diagnostic service pilot**

**Kaanthan Jawaah;** *Mohammad Zia Ul Haq Katshu; James Ellison; Mohan Rathnaiah; David Rhinds*

**Aim and hypothesis:** To pilot an ARBI diagnostic service within a community substance misuse team (delivered in partnership with the NHS by a 3rd sector organisation).

**Background:** Alcohol-related brain injury (ARBI) is an umbrella term encompassing alcohol-related cognitive impairment, alcohol-related 'dementias', Wernicke's encephalopathy and Korsakoff's syndrome. It is the result of prolonged and harmful alcohol misuse, developing through direct neuronal damage from alcohol as well as chronic deficient states of vitamin B1 (thiamine). Anecdotally, it is felt that those affected by ARBI can struggle to access the necessary expertise for diagnosis and ongoing management.

**Methods:** A widespread stakeholder engagement process led to the development of a standard operating procedure for the pilot service, utilising a process mapping technique. The service received referrals between September 2018 and January 2019. During this time, assessments were carried out in line with the standard operating procedure by the authors. Referrers were also asked to complete a questionnaire for their views on the service.

**Results:** Referrals were received from several sources within the host third sector organisation. Heterogeneity was seen in presentations and diagnoses made were not limited to ARBI. Referrers spoke highly of the service and how it had positively benefitted their patients going forwards, providing clarity around diagnoses and thus being able to access appropriate support going forwards. There was a clear need demonstrated for such a service.

**Conclusions and Next Steps:** Anecdotally patients with ARBI are viewed as a marginalised group who struggle to access the necessary expertise for diagnosis and ongoing management. This service pilot was successful in filling that gap and demonstrated a proof of concept. Work was undertaken with system partners to devise an acute hospital 'in-reach' pathway embedded with a liaison psychiatry team. This pilot started in February 2020 and was due to run for 4 months to quantify need, but unfortunately was curtailed owing to the COVID-19 pandemic. In the one month that it ran it was very well received and operationally deliverable. It remains an 'off the shelf' solution for when circumstances allow its resumption.

### **13. Application of Actigraphy in Functional Movement Disorders: A Review of the Literature**

*Dr Claire Jones, ST6 Liaison Psychiatry*

**Background:** Actigraphy and other wearable technology is increasingly being utilised in research and clinical applications for neurological disorders, in particular in organic movement disorders. However, little is known about the use of actigraphy in functional movement disorders (FMD), which are recognised as common and disabling conditions. This review examines the scope and quality of the literature into the use of actigraphy in FMD.

**Methods:** A systematic literature review was performed using a comprehensive search strategy for FMD and use of actigraphy or other wearable technology. The identified studies' methodological quality was assessed. Data was extracted on study participants, setting, design and methods, outcomes measured and the validity and reliability of results.

**Results:** 13 studies were included for data extraction and synthesis. All of these focused on functional tremor. The studies retrieved were small, observational and heterogeneous in design with no randomised controlled trials identified. Whilst the power, statistical significance and generalisability of the studies was low, available research did provide proof of principle for the use of actigraphy in both diagnosing and monitoring symptoms of functional tremor over time, and attempted to identify underlying pathophysiological mechanisms. None of the research considered actigraphy for the treatment of FMD.

**Conclusions:** Actigraphy represents a potentially important advancement in the understanding, diagnosis and monitoring of FMD but the reliability, validity and clinical utility of such instruments in this patient group remain to be established.



#### **14. Visual hallucinations in organic psychosis: a meta-analysis**

*Maria Teixeira-Dias; Amber Kaur Dadwal*

**Background:** Distinguishing an 'organic' from a 'non-organic' cause of psychosis is critical, as appropriate treatment may reverse the underlying cause. At present there are no agreed 'red flags' to help a clinician identify a patient likely to have an organic cause based on their psychiatric features. The literature suggests that visual hallucinations may occur more commonly in organic psychosis than non-organic psychosis and this meta-analysis aimed to analyse this association. Additionally, the content of visual hallucinations was also explored.

**Methods:** PubMed, OVID, MEDLINE, Embase, PsychINFO and Global Health databases were searched without date restrictions using the keywords "Psychos\*" AND "Schizophreni\*" AND "Visual hallucinat\*". The inclusion criteria for the meta-analysis were: a) case-control studies in English b) with patients diagnosed with organic psychosis and compared to a non-organic psychosis control group and c) reporting the presence of visual hallucinations in both cohorts.

**Results:** Fifteen studies comprised of 844 organic psychosis patients and 871 non-organic psychosis patients were eligible for inclusion and had their data extracted. The presence of visual hallucinations were found to occur more frequently in organic psychosis compared to non-organic psychosis (odds ratio = 2.49, 95% CI = 1.47, 4.23). The content of visual hallucinations was reported in 4 studies (158 organic psychosis patients and 52 non-organic psychosis patients). The most common type of visual hallucination was of people, reported in 76 (48.1%) organic patients and 31 (59.6%) non-organic patients. There was a significantly higher occurrence of inanimate visual hallucinations in patients with organic psychosis compared to those with non-organic psychosis ( $\chi^2(1) = 6.545, p = 0.011$ ).

**Conclusion:** Visual hallucinations may serve as a 'red flag' in organic psychosis and aid in the distinction from non-organic psychosis. Future research could investigate distinguishing features of animate and inanimate visual hallucinations in both groups

## **15. Cognitive deficits described in people with Tourette Syndrome**

**Dr Rabia Khan**

*Abbreviations: TS: Tourette Syndrome, ADHD: Attention deficit hyperactivity disorder, OCD: obsessive compulsive disorder*

**Introduction:** TS is a neurodevelopmental disorder starting before the age of 18 years characterised by the presence of at least one vocal and multiple motor tics lasting for a year and not explained by medications or another medical condition. The neurological basis of TS is not clearly understood. It is generally agreed that a wider dysfunction in basal ganglia, frontostriatal circuitry and cortical-striatal-thalamic-cortical (CSTC) circuitry can lead to this disorder possibly leading to disinhibition and executive function deficits. Thereby prompting the exploration of cognitive deficits in TS.

**Method:** The review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) checklist. Two databases PsychINFO and EMBASE from 2014 – present (last 5 years) were used for a comprehensive search strategy using Cheshire and Wirral NHS Foundation Trust Healthcare and Management Databases. The key words were '(Tourette\*)' OR '(TS\*)' OR '(TS)' AND '(cognitive\*)' AND '(deficit\*)' OR '(disorder\*)' OR '(function\*)'. Records were limited to those in English language.

**Results:** While some studies show no difference in cognitive functioning in patients with TS others show some associated decline. The discrepancies seem to be associated with methodological differences such as sample size and varied neuropsychological assessment tools used to explore various cognitive sub processes.

The studies considered the prevalence of other co-morbid conditions like ADHD, OCD and impulse control disorders and tried to minimise their confounding effects via their adopted methodology and statistical analysis. An interesting aspect was the notion where TS with and without comorbid ADHD were represented as two distinct types.

**Conclusion:** Cognitive deficits in TS is still a less understood topic that will need further research using larger samples and valid neuropsychological assessment tools. Neuroimaging studies to compound the neuropsychological testing can yield more conclusive results.

## **16. Cognitive symptoms and memory worry in young adults**

**Lachlan King;** *Eilidh McClure; Laura McWhirter; Craig Ritchie; Jon Stone; Alan Carson*

**Background:** Increasing numbers of people present to health care services with memory concerns. Many do not have dementia due to degenerative brain disease. Some may be in prodromal states that will progress to dementia; others have Functional Cognitive Disorders. Others may describe symptoms that are within normal experience.

**Objectives:** We aimed to establish the frequency of memory worry and cognitive symptoms in healthy adults.

**Method:** A survey distributed through social media included multiple-choice questions about frequency of cognitive lapses, beliefs about own memory-efficacy, and worry about memory symptoms. The survey incorporated the Schmidtke and Metternich (2009) short functional memory disorder (FMD) inventory. Respondents were excluded who reported having received a diagnosis of any memory-related cognitive disorder.

**Results:** 127 participants, aged 18-60 (mean = 27) completed the survey. Experience of cognitive lapses was common, with respondents experiencing, on average, 13 of a list of 18 lapses at least once per month. The two most commonly experienced lapses were absent-mindedness and word-finding issues, both with 57% of respondents experiencing these at least weekly. 26% reported that their memory was 'poor' or 'fair', and 39% reported worry about their memory. 89% of this healthy cohort scored within the functional memory disorder range on the short FMD inventory.

**Conclusion:** Cognitive symptoms, and concern about these symptoms, is common in young adults and does not necessarily indicate that a cognitive disorder is present. In designing clinical services which incorporate early detection of disease biomarkers it is imperative that efforts are also made to better understand overlaps and boundaries between normal and pathological cognitive failure.

## 17. Neuropsychiatric complications of COVID-19 from the UK-wide CoroNerve study: diverse presentations illustrated by case vignettes

**McCausland, B.**<sup>\*1,2,3</sup>, Jeyanantham, A.<sup>\*1</sup>, Varatharaj, A.<sup>1,2</sup>, Kneen, R.<sup>4,5,6</sup>, Pett, S.<sup>7,8</sup>, Thomas, R.H.<sup>9,10</sup>, Galea, I.<sup>1,2</sup>, Amin, J.<sup>1,3</sup>, Michael, B.D.<sup>4,5,11</sup>, Joyce, E.<sup>12</sup>, Carson, A.C.<sup>13</sup>, Pollak, T.A.<sup>14</sup>, Nicholson, T.R.<sup>14</sup>. CoroNerve Study Group.

We would like to particularly thank contributing authors Collin, G.<sup>15</sup>, Daher, M.<sup>16</sup>, Serra-Mestres, J.<sup>17</sup> and Sivananthan, A.<sup>18</sup> for sharing their case studies with the Coronerve Study Group.

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**Introduction/Background:** Concerns regarding potential neurological and psychiatric complications of COVID-19 are being increasingly reported. Comprehensive characterisation of these clinical syndromes is crucial to allow selection and evaluation of potential therapies. The aim of this study was to investigate the breadth of cerebral complications of COVID-19 across the UK. We present data on the neuropsychiatric complications of COVID-19, alongside four psychiatric case vignettes which capture the diversity of these complications.

**Methods:** During the exponential phase of the pandemic, we developed an online network of secure rapid-response case report notification portals across the spectrum of major UK neuroscience bodies, comprising the Association of British Neurologists (ABN), the British Association of Stroke Physicians (BASP) and the Royal College of Psychiatrists (RCPsych),

representing neurology, stroke and psychiatry respectively. Physicians were encouraged to report cases prospectively and we permitted recent cases to be notified retrospectively when assigned a confirmed date of admission or initial clinical assessment, allowing identification of cases that occurred before notification portals were available. Initial data were inputted by reporting clinicians who were then sent case report forms (CRFs) for detailed case reporting [ongoing at time of submission]. Here we select four cases from this preliminary data to explore in more depth, by contacting the referring clinicians for more detailed case histories and formulations.

Results: Overall, 153 cases were reported. 25 patients (16%) were referred through the RCPsych portal. Data on reporting physician specialty were available for 150 patients: 26 (17%) were psychiatrists or neuropsychiatrists. Complete clinical datasets were available for 125 (82%) of 153 patients.

In total, 39 (31%) of 125 patients presented with altered mental status, comprising nine (23%) patients with unspecified encephalopathy and seven (18%) patients with both clinical symptoms or signs of encephalopathy and evidence of CNS inflammation meeting the clinical case definition for encephalitis. The remaining 23 (59%) patients with altered mental status fulfilled the clinical case definitions for psychiatric diagnoses as classified by the notifying psychiatrist or neuropsychiatrist. Only two (9%) of 23 patients had exacerbations of existing enduring mental illness. Ten (43%) of 23 patients with neuropsychiatric disorders had new-onset psychosis, six (26%) had a neurocognitive (dementia-like) syndrome, and seven (30%) had another psychiatric disorder, including one case of catatonia and one case of mania.

We present four case vignettes in this poster: 1. New-onset psychosis, 2. New-onset neurocognitive disorder, 3. New-onset mania and 4. New-onset catatonia.

Conclusions: To our knowledge, this is the first nationwide, cross-specialty surveillance study of acute neurological and psychiatric complications of COVID-19. Infection with COVID-19 has been shown to exacerbate pre-existing psychiatric conditions or result in *de novo* psychiatric syndromes, including psychosis, dementia, mania and catatonia. This study highlights the impact of this respiratory virus on the central nervous system, the often-severe impact on patients' lives, and the necessity for multidisciplinary involvement across the neurosciences in managing this patient group.

## **18. A survey of psychiatry trainees to identify essential assessments and investigations in first episode psychosis**

**Hamilton Morrin;** *Claire Carstairs; Timothy Nicholson; Graham Blackman*

**Introduction:** Psychosis is often described as a loss of contact with external reality consisting of delusions, hallucinations, and disorganized speech and behaviour. An episode of psychosis can be considered the result of a primary psychiatric disorder, or secondary to an organic cause. Currently there is a lack of international consensus on essential investigations for first episode psychosis.

**Methods:** An online Qualtrics survey was sent out to registrar grade psychiatrists at South London and Maudsley (SLaM) NHS Foundation Trust during March - April 2020. Clinicians were asked to rate the importance of a range of clinical features in screening for an organic cause of psychosis. They were also asked to rate the importance of including certain investigations as part of routine assessment for first episode psychosis. For both tasks, responders rated a number of predefined items, based on a review of the literature, using a five point Likert scale from 1 (essential) to 5 (should not be performed/included). Mean response index for each item was calculated to rank clinical features and investigations in order of importance.

**Results:** The survey was completed by 20 trainees. All agreed that information collected on initial assessment were informative in screening for organic causes of psychosis. The highest ranked clinical features were altered consciousness (1.1), cancer (1.1), neurological disorder (1.1), brain injury (1.1), delirium (1.1), focal neurology (1.1), immune deficiency (1.2), unexplained weakness (1.2), autoimmune disease (1.3) and signs of infection (1.3). All respondents agreed that patients with first episode psychosis should undergo a routine set of investigations. The highest ranked investigations were neurological exam (1.1), full blood count (1.1), urea and electrolytes (1.1), liver function tests (1.2), inflammatory markers (1.2), general physical exam (1.3), calcium (1.3), thyroid function (1.3), urine drug screen (1.3), and lipid profile (1.4).

**Conclusion:** Among psychiatrists within a large Mental Health Trust, there was consensus that screening for organic causes in a patient presenting with psychosis is essential. In particular, altered consciousness, delirium, focal neurology, or history of cancer, neurological disorder or brain injury were considered most important. There was also agreement that routine investigations should be performed in all patients, with neurological exam and blood tests (for full blood count and urea and electrolytes) deemed most essential.

## **19. An audit of inpatient resources used in organic neurological versus functional neurological disorders**

**Hannah Newman;** *Timothy Greig; Michael O'Gara*

**Objective** To assess the incidence of functional neurological symptoms (FNS) in patients admitted to a neuroscience centre in South West England, and to compare their length of stay and number of inpatient investigations to other neurological presentations. **Background** Patients with FNS may be admitted to hospital with neurological symptoms such as weakness, seizures or tremor. There is no good data on how often patients are admitted to hospital due to FNS, or the utilisation of inpatient investigations for this group. Derriford hospital has an acute neurology service that sees all patients admitted to inpatient bed with neurological symptoms without first being seen by the medical team. This provides an opportunity to identify the frequency of admissions to hospital with FNS, as neurologists are best placed to identify these features.

**Methods:** Data was collected over a 4-month period. Patients seen on the acute neurology take were divided into groups based on the assessing consultant neurologist's impression of their current presentation as: fully explained by organic neurological disease; partially by organic neurology with functional features; entirely functional; or partially or fully due to other medical pathology. Organic neurology was further subcategorised into stroke and other pathology. The length of stay, number of neurological investigations conducted (CT and MRI scans, lumbar punctures and neurophysiology tests) were recorded for each patient and the groups compared.

**Results:** 193 patients were included in the analysis, taken from 43 days over 4 months. 6.7% were entirely functional and 11.9% partially organic neurological with functional features. 72.5% were fully explained by organic neurological disease. 4.7% were partially explained by neurological disease and partially by other medical pathology, and 4.1% were fully explained by non-neurological medical disease.

Length of stay was 3.7 days for functional presentations, 4.95 days for partially functional, 8.44 days for fully organic neurological (6.9 days when stroke diagnoses excluded), 7.77 days for partially medical, and 9.5 days for fully medical presentations. The average number of investigations was 1.6 for fully functional presentations, 1.7 for partially organic neurological with functional features, 2.1 for organic neurological, 1.6 for partially neurological & partially medical, and 2.0 for fully non-neurological medical presentations.

**Conclusions:** Functional neurological symptoms comprise a significant percentage of patients admitted to hospital with neurological symptoms. All together patients presenting with functional features accounted for 18.6% of neurological presentations to a neurosciences centre, representing a significant number of hospital bed days and inpatient investigations.

## **20. The pregabalin paradox: examining the risk of visual hallucinations persisting post-cessation**

**Dr Ashy Rengit;** *Dr Amit Bhardwaj, Liaison Psychiatry, Kent & Medway NHS & Social Care Partnership Trust*

Objectives: Prescribing pregabalin carries the risk of ocular effects – blurred vision, diplopia, and rarely, visual hallucinations. This case report considers the possibility that pre-morbid ocular pathology heightens the risk of developing longterm pregabalin-induced visual hallucinations.

This is in contrast to current literature on the subject, which suggests that –

- Pregabalin induced visual hallucinations remit following treatment cessation
- Pregabalin has a role in treating visual hallucinations associated with poor vision e.g. Charles Bonnet syndrome.

Methods: Ward documents, and electronic records were utilised for the case details. A literature search was undertaken using Pubmed and Google Scholar, using the terms “visual”, “hallucinations” and “pregabalin”.

Case: Mrs B. - a 64 year old lady, was referred to liaison psychiatry for complex visual hallucinations, persisting after cessation of pregabalin initially prescribed for diabetic neuropathy. According to her records, she had been on a rapid dose escalation regimen –

- Initially 25mg twice a day – to be increased by 50mg each day for 7 days to 150mg BD
- Pregabalin then increased to 300mg daily in divided doses over the following week (If needed, pregabalin was due to be increased to 600mg daily in divided doses)

She described bizarre experiences of seeing ‘small running children, and baby blankets for sale’ on the adult cardiology ward, where she had been admitted following cardiac stenting for myocardial infarction. Visual hallucinations were exacerbated in poorly lit environments, typically in the evenings, and she reported these images vanished if she blinked or moved. There was no evidence of confusion, agitation or disorientation noted during medical admission.

Mrs B. relayed that she found these experiences pleasant, albeit worrying, as she retained insight that these images were unrealistic. She also reported ‘seeing more shadows’ in her central vision, attributing this to recent diagnosis of macular degeneration; which was untreated due to longstanding keratoconus. She remarked on loss of function due to blurred vision during the course of using pregabalin. In addition, her history conveyed a high risk of retinopathy due to marginally controlled diabetes, which had resulted in nephropathy and toe amputation.

In terms of mental health history - Mrs B. had been treated for long-term social anxiety and depression using citalopram in primary care. When seen, there was no evidence of acute mood changes or additional psychotic symptoms. Although she had a history of gradual, mild cognitive impairment – she was fully orientated with no evidence of Parkinsonian features to suggest Lewy Body Disease as a potential cause of visual hallucinations.



Discussion: Overall, current research on this topic appears to be limited to various case reports illustrating the varying relationship between pregabalin and visual hallucinations. A proportion of the literature suggests that pregabalin use correlates with development of visual hallucinations in individuals without ocular comorbidity, and side effects remit on cessation of treatment (Mousailidis et al., 2020) The risk of adverse effects appears to be dose-dependent, with rapid dose escalation leading to greater likelihood of visual phenomena (Pedroso et al., 2012)

Commencing pregabalin treatment at high doses has also been noted to increase the risk of visual hallucinations. The pathophysiology is yet to be elucidated, but it has been suggested that certain individuals are more predisposed, or sensitive, to changes in neurotransmitter potentiation following pregabalin administration (Olaizola et al., 2006) This is in contrast to research which suggests that high-dose pregabalin is effective for reducing visual hallucinations, secondary to optic nerve atrophy in Charles Bonnet Syndrome (a disorder whereby loss of visual acuity increases the risk of visual hallucinations) (Bundle et al., 2013)

However, it appears that low-dose pregabalin is preferred in elderly patients for visual hallucinations associated with dementia (Cinar et al., 2011) – although it is important to note that these effects are not evidenced by systematised research trials, and remain an off-license treatment option (De Souza et al, 2016) Finally, more limited research considers the possibility that pregabalin withdrawal could contribute to hallucinatory experiences post-treatment (Gundogmus et al., 2018) It is however notable, that such hallucinatory experiences are multi-modal, commence following pregabalin cessation and do not exclude other confounding risk factors for psychotic disorder.

Conclusion: The literature reviewed suggests that there is no consistent aetiological pathway identified for pregabalin-related visual hallucinations. However, it does imply that certain patient groups may be more likely to develop adverse visual phenomena secondary to pregabalin use.

As pregabalin is a recognised risk factor for reduced visual acuity - we would like to consider the possibility that individuals with ophthalmic co-morbidities are at greater risk of accelerated vision loss following its use. This arguably also places such patients at greater risk of Charles Bonnet syndrome, which presents with visual hallucinations secondary to acute decline in eyesight. We would therefore like to propose that pregabalin requires greater caution when prescribing and monitoring effects in this population.

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## **21. The Lateralisation of Language in the Brain: The Limitations of Current Literature**

***Shaheen Sardar***

The lateralisation of language to the left hemisphere in the majority of individuals who are right handed has thought to have been established, however, this arrangement does not currently have a widely accepted theoretical explanation. This may be due to the limitations of the field; although the left hemisphere is most commonly dominant, it is not a universal finding. Anatomical studies examining brain regions that correlate to language lateralisation have shown mixed findings. Whilst several studies have shown a high prevalence of perisylvian white matter volumes with asymmetry concentrated on the left (indicating language networks as being a more likely anatomical substrate for lateralisation), there have been numerous studies indicating that leftward lateralisation of these regions (such as the temporal planus) is not correlated with language lateralisation. Thus, this analysis of the current literature aimed to identify the limitations associated with the study of lateralisation of the language networks within the brain through neuropsychiatric evidence and to further explore to what extent language function fits this set model of lateralisation to the left hemisphere.

## **22. A case of undetected Temporal Lobe Epilepsy- presenting with autonomic symptoms**

Dr A Zia; **Dr Anu Sharma**

This is a case of a 58 year old female who lived alone and was suffering from mild Learning disability and Epilepsy (generalised tonic-clonic seizures and absence seizures-which were diagnosed at the age of eight years). She also had some ASD traits. Her epilepsy had been well-controlled for many years on valproate 600 mg bd.

She also suffered from Raynauds phenomenon, and could not bear hot weather- there were reports of her collapsing during heat wave.

She also had bereavement 1 year ago, when her partner of 18 years died unexpectedly- he died in a pool of blood, when his lung tumour ruptured and he had a massive bout of haemoptysis. Later on, ambulance crew had to break the door to gain access to the body. She witnessed all of this and was noted to be suffering from anxiety and low mood after the incidence, for which she was referred to us.

There was also a mention of other episodes lasting for a few minutes, where she was breathing heavily, foaming at the mouth, looking angry and making grunting noises and she would hit herself. These episodes last for up to ten minutes, and she did not have any recollection of these episodes. There was no loss of consciousness, tongue-biting, cyanosis or incontinence.

The general opinion of the MDT was that she is suffering from dissociative features. With limited coping skills and limited emotional expressivity (LD and ASD)- she was not able to cope with the trauma of her partner's death, and was also struggling to come to terms with the circumstances surrounding the death.

However, and EEG was still requested to rule out any frontal lobe activity.

Her EEG showed intermixed theta (4-7Hz) and delta waves (2-3 Hz) over both hemispheres, maximally over the temporal regions. In addition, fairly frequent irregular slow waves (2-3 Hz), upto 65 microV, appeared over the temporal regions. During hyperventilation (for 3 minutes)- a small increase in slow activity was seen, maximally over temporal regions. It was clear that the episodes (described above) that she was suffering from were manifestation of ongoing TLE, presenting with autonomic hyperarousal.

Her medication was reviewed and her valproate was increased by 200 mg, noct. Although, valproate was not the choice medication for TLE, but since she lived alone, there were concerns regarding the feasibility to monitor side effects during the lock down period (she had been tolerating valproate for decades without any side effects) - it was reasonable to increase her valproate, rather than to introduce another AED.

She was offered art therapy to enable her to deal with the trauma. Also, currently, there are plans of moving her to a supported accommodation.

### **23. The Procognitive Effects of Ketamine – An analysis of Ketamine for reduction of Alcoholic Relapse trial memory data**

**Mr Andi Stanescu;** *Professor Celia Morgan*

**Aims:** Over the years ketamine has been used as an effective dissociative anaesthetic and more recently has shown potential as an antidepressant in people whose low mood has not lifted as a response to first-line treatments. There is evidence that ketamine may be beneficial for memory, with the proposed mechanisms being those of synaptogenesis and neurogenesis. This randomised, double blind, placebo-controlled study examined whether participants who were randomised to receive three weekly ketamine infusions showed improvements in working memory performance relative to those who received placebo infusions. We additionally explored the link between cognitive function at follow-up and abstinence from alcohol.

**Methods:** Participants attended the hospital for ten testing sessions, of which three were infusion sessions. They completed a mnemonic similarity task before and after each infusion, once during the final lab session (which did not include an infusion), as well as once during the three and six month follow-up sessions. They also completed a forwards and backwards digit span task on four occasions – at the baseline session, the final lab session, and the two follow-up sessions.

**Results:** Our findings do not support our main hypothesis that ketamine infusions have a procognitive effect, as measured by the mnemonic similarity task and digit span performance, as we found no differences between the ketamine and placebo group, either acutely or at follow-up. We also did not find a significant link between abstinence from alcohol and performance on the mnemonic similarity task or digit span task.

**Conclusion:** This suggests that ketamine does not affect working memory acutely or in the longer term as no positive effects were found. We did not identify any significant detrimental cognitive effects of ketamine either, as similar results were observed for the placebo group, in contrast with findings from studies which found that ketamine has a detrimental effect on memory. It is possible that the negative effects become apparent at higher or more frequent doses than those received by the participants in our study.

## **24. Fregoli syndrome in primary and secondary psychosis: A case-level meta-analysis.**

*Maria Teixeira-Dias; Amber Kaur Dadwal*

Background: Fregoli syndrome is a delusion of misidentification characterised by the misidentification of an individual as someone who the patient has an emotional link to. This syndrome can be present in psychotic episodes of primary or secondary nature. The aim of this case-level meta-analysis was to compare the neuropsychiatric characteristics of Fregoli syndrome in primary and secondary psychosis.

Methods: Five databases were searched for any reports of Fregoli syndrome. The patients' and the psychotic episodes' details alongside the co-occurring neuropsychiatric features and treatment responses were extracted. A risk of bias assessment was carried by scoring the methodological quality of all case studies. Odds ratios (OR) were used to compare the clinical features' frequency between primary and secondary psychoses. This meta-analysis followed the PRISMA guidelines.

Results: A sample of 67 patients with Fregoli syndrome was found from the review of 561 articles: 47 with primary psychosis and 20 with secondary psychosis. Results showed that patients in the secondary psychosis group were significantly more likely to present with Fregoli delusion as part of a first-episode psychosis (OR = 7.04,  $p = 0.03$ , 95% CI [1.07, 60.49]) and to have neuroimaging abnormalities (OR = 10.66,  $p = 0.007$ , 95% CI [1.51, 108.87]). Additionally, 67% of the brain lesions reported were in the right hemisphere, 50% in the temporal lobes and 42% in the frontal lobe. No statistical difference between the other analysed clinical features was found.

Fregoli syndrome is more likely to be reported in a first-episode psychosis and to be connected to neuroimaging abnormalities in secondary psychosis when compared to primary psychosis. Neuroimaging abnormalities in these cases are seen predominantly on the right hemisphere. This is the first meta-analysis investigating the features of Fregoli syndrome in primary and secondary psychosis. These results are discussed and potential clinical applications are suggested.

## **25. Understanding functional seizures: Are functional/dissociative seizures “dependent on idea”?**

**Jakov Tiefenbach**; *Mhairi Paterson; Emily Rose; Shona Scott; Ingrid Hoeritzauer; Laura McWhirter; Alan Carson; Jon Stone*

**Background:** It has been suggested for over 100 years that patterns of neurological symptoms and signs in functional neurological disorders (FND) may be shaped at a neural level by underlying ideas or preconceptions how neurological symptoms present. This study used experimental simulation in healthy volunteers to obtain a picture of common widespread ideas about seizures, with a view to compare with features commonly observed in functional and epileptic seizure disorders.

**Methods:** Sixty healthy volunteers recruited from non-medical student and adult populations were instructed to simulate an epileptic seizure. The episodes were video-recorded and assessed by three qualified markers for the presence of clinical features commonly observed in functional seizures (FS), epileptic seizures and syncope. In addition, 10 randomly selected media depictions of epileptic seizures were analysed.

**Results:** Simulated seizures were hyperkinetic (83%), hypokinetic (7%) or staring (10%). 52% had their eyes open and 45% eyes closed. Tremor was observed in 70%, while clonic jerking was only present in 17%. The majority of volunteers maintained a normal or floppy body posture. Head shaking side-to-side was observed in 38%, whilst guttural vocalisation and tearfulness was absent in all volunteers.

**Discussion:** Simulated seizures have some similarities to FS, but also some important differences. Subjective seizure experiences in people with FS, not captured by this experimental simulation, remain a core determinant of semiology. Common ideas about seizure semiology may be shaped by witnessing functional seizures in others, including media depictions.

## **26. Relationships Between Self-Reported Mental Health Difficulties, Autistic Traits and Gender: a Cross-Sectional Population-based Study**

**John H Ward**; *Simone Capp*; *Professor Francesca Happé*; *Dr Grainne McLoughlin*

Background: internalising disorders in autism spectrum disorder (ASD) are common and have serious consequences for autistic people. ASD is under-recognised in girls, and therefore the relationship between autistic traits and internalising disorders may be different to that seen in boys. Methods: 556 people (mean age  $22.4 \pm 0.97$  years) taken from the Twins Early Development Study (TEDS) undertook online and in-person neuropsychological testing. Measures of autistic traits (the Social Responsiveness Scale 2nd Edition (SRS-2) and the Autism Diagnostic Observation Schedule 2 (ADOS) Module 4) were examined with respect to depression and anxiety (measured via the Mini International Neuropsychiatric Interview (MINI)). Results: There were statistically significant correlations between autistic traits and the number of MINI diagnoses endorsed, and this relationship was statistically significantly stronger for girls than boys ( $p < .0001$ ). Furthermore, binary logistic regression revealed significant interactions between being female and autistic traits (from ADOS but not from SRS-2) in relation to MINI anxiety disorders and major depressive disorder (any MINI anxiety disorders odds ratio for girls = 1.56 (95% CI 1.22-2.00,  $p < .0001$ ); major depressive disorder odds ratio for girls = 1.5 (95% CI 1.23-1.85,  $p < .0001$ )). Conclusion: The current results provide evidence that the relationship between autistic traits and mental health difficulties is different for males and females. Furthermore, they suggest a difference in this relationship depending on whether autistic traits are measured by self-report (SRS-2) or behavioural observation (ADOS). Further research examining putative mechanisms for these gender differences and establishing causal directions is needed.



## **27. Vitamin D screening in neuropsychiatry: results from a service evaluation in an inpatient unit**

*Dr Alexa Zhang; Dr Jacqueline Foong*

Objectives: To evaluate the frequency of vitamin D screening and serum vitamin D levels in patients admitted to a neuropsychiatric inpatient unit.

Method: A retrospective case note review was carried out on 49 consecutive patients admitted to a tertiary neuropsychiatric unit over a 6-months period. 20 patients had organic neuropsychiatric disorders and 29 patients had functional neurological disorders. Clinical characteristics and vitamin D screening rates and outcomes were evaluated.

Results: 32 (65%) of 49 patients were screened for serum vitamin D levels during their admission. 23 (74%) of 32 patients were found to have suboptimal vitamin D levels. 76% and 55% of all patients reported fatigue and pain respectively. 76% of FND patients had co-morbid depression while 20% of organic patients had depression and 60% had psychosis. 20% of patients were taking vitamin D supplements before admission and 22% of all patients were started on treatment after screening during admission.

Conclusion: Suboptimal vitamin D levels are common in neuropsychiatric inpatients. We suggest that routine screening of vitamin D should be considered in this population for earlier treatment and to improve neuropsychiatric treatment outcomes. Further prospective studies using larger samples are recommended to confirm our findings.

















