reward likelihood being uncertain. This finding has key implications to the design of novel interventions for OCD.

#3101 IMBALANCED BASAL GANGLIA CONNECTIVITY IS ASSOCIATED WITH MOTOR DEFICITS AND APATHY IN HUNTINGTONS DISEASE: FIRST EVIDENCE FROM HUMAN *IN VIVO* NEUROIMAGING

[1]Akshay Nair, [2]Adeel Razi, [1]Sarah Gregory, [1,3]Robb Rutledge, [1]Geraint Rees*, [1]Sarah Tabrizi*. [1]*University College London, London, UK*; [2]*Monash University, Melbourne, AUS*; [3]*Yale University, New Haven, USA*

10.1136/jnnp-2021-BNPA.15

Background The gating of movement in humans is thought to depend on activity within the cortico-striato-thalamic loops. Within these loops, emerging from the cells of the striatum, run two opponent pathways the *direct* and *indirect* pathway. Both are complex and polysynaptic but the overall effect of activity within these pathways is to encourage and inhibit movement respectively. In Huntingtons disease (HD), the preferential early loss of striatal neurons forming the indirect pathway is thought to lead to disinhibition that gives rise to the characteristic motor features of the condition. But early HD is also specifically associated with apathy, a failure to engage in goal-directed movement. We hypothesised that in HD, motor signs and apathy may be selectively correlated with indirect and direct pathway dysfunction respectively.

Methods Using a novel technique for estimating dynamic effective connectivity of the basal ganglia, we tested both of these hypotheses *in vivo* for the first time in a large cohort of patients with prodromal HD (n = 94). We used spectral dynamic casual modelling of resting state fMRI data to model effective connectivity in a model of these cortico-striatal pathways. We used an advanced approach at the group level by combining Parametric Empirical Bayes and Bayesian Model Reduction procedure to generate large number of competing models and compare them by using Bayesian model comparison.

Results With this fully Bayesian approach, associations between clinical measures and connectivity parameters emerge *de novo* from the data. We found very strong evidence (posterior probability > 0.99) to support both of our hypotheses. Firstly, more severe motor signs in HD were associated with altered connectivity in the indirect pathway and by comparison, loss of goal-direct behaviour or apathy, was associated with changes in the direct pathway component of our model.

Conclusions The empirical evidence we provide here is the first *in vivo* demonstration that imbalanced basal ganglia connectivity may play an important role in the pathogenesis of some of commonest and disabling features of HD and may have important implications for therapeutics.

#3113 EVALUATING THE PCL-C AS A MEASURE OF TRAUMA AND PTSD SYMPTOMS IN PATIENTS WITH FUNCTIONAL NEUROLOGICAL SYMPTOM DISORDER

Umamah Yusufi. *Sheffield Institute of Translational Neuroscience (SITrN)*

10.1136/jnnp-2021-BNPA.16

Aims Functional Neurological Symptom Disorder (FNSD) is common and disabling. Historically trauma was considered an essential aetiological factor, but the precise contribution of trauma to the disorder, and resulting disability, remains controversial. The PCL-C is a self-reported screening tool for PTSD symptoms based on DSM-IV criteria. A previous study in patients with FNSD demonstrated high scores on the PCL-C, with a reduction in scores following psychotherapy. However strong correlations with other psychological co-morbidities raised the possibility that the PCL-C may be capturing non-specific distress rather than indicators of previous traumatisation. The present study aimed to investigate (i) whether underlying factors measured by the PCL-C can distinguish specific trauma-memory-related symptoms from less specific emotion-regulation-related symptoms, (ii) the extent to which individual factors correlate with other psychopathology and health-related quality of life (HRQoL) measures and (iii) whether different factors change with psychotherapy, all in patients with FNSD.

Methods An exploratory factor analysis of PCL-C responses from 473 FNSD patients pre-and post-psychotherapy was performed to generate1-4 factor models. The final factor model was determined through confirmatory factor analysis. Relationships between PCL-C factors, measures of comorbidities (depression, somatisation and anxiety) and HRQoL were assessed using regression analysis. Pre- and post-psychotherapy scores were compared.

Results The best model for the PCL-C comprised of two-factors: factor 1 (intrusive symptoms) explained 55.2% of the variance, whilst factor 2 (emotional dysregulation) explained 7.8% of the variance. Both factors reduced in severity after psychotherapy, but factor 2 reduced by more and correlated more strongly with a decrease in depression and anxiety than factor 1. Changes in depression, anxiety, somatic symptoms and mental HRQoL predicted 61.9% of the change in factor 2, but only 49.2% in factor 1. Improvements in mental HRQoL were strongly associated with a decrease in factor 2, but less so in factor 1.

Discussion The factor analysis revealed the PCL-C represents two correlated but distinguishable symptom clusters in patients with FNSD: intrusive symptoms and emotional dysregulation. Both were elevated in our patient sample. The high level of intrusive symptoms suggests many patients with FNSD experience classical post-traumatic symptoms, which may indicate that subjective traumatic experiences contribute to pathogenesis and resulting disability. These symptoms showed some reduction with psychotherapy, but the greater reduction in emotional dysregulation symptoms and associated improvements in mental HRQoL suggest that psychotherapy may be more effective in reducing the burden of comorbidities and providing better coping strategies than in addressing core symptoms of the underlying disorder.

#3112 ACQUIRED APHANTASIA IN 88 CASES: A PRELIMINARY REPORT

Laurence Knowles, Kealan Jones, Adam Zeman. *University of Exeter Medical School, College House, Exeter, UK*

10.1136/jnnp-2021-BNPA.17

Aims For most of us, visual imagery is a fundamental feature of day-to-day subjective experience. It is thought to play multiple cognitive roles.[1] However, there is widespread variation in the subjective intensity of visual imagery, ranging from

extreme vividness to complete absence. The term aphantasia was coined recently to describe the latter, which is usually lifelong. While rarer, cases of acquired aphantasia can provide mechanistic insight. Isolated cases have long been reported,[2] with some attempts at theoretical synthesis.[3 4] We give a preliminary description of 88 such cases identified from among ~14,000 people contacting us in the wake of publicity surrounding Aphantasia.

Methods Cases were selected from individuals contacting us spontaneously reporting reduced or absent intensity of visual imagery. Contacts were asked to complete two measures of visual imagery, the Vividness of Visual Imagery Questionnaire (VVIQ) and Imagery Questionnaire (IQ).[5]

Results Cases were divided into those in with a strong probability of a neurological cause (n=39), a psychological cause (n=20) and those about which we cannot yet be confident (n=29). Functional aphantasia appears likely to account for some of the cases in the third category. The commonest precipitating events were head injury (n=19), affective disorder (n=17) and stroke (n=13). Other causes included surgery (postoperative) (n=7), drugs (n=6), infectious or inflammatory disease (n=3), neurodegenerative disease (n=3), and seizure disorders (n=2). For subjects completing the VVIQ (n=29), the mean score was 20.1/80 (range 16-32,) indicating marked reduction of imagery vividness. Localizable lesions were predominantly right sided (n=6) and occurred in posterior cortical areas, particularly occipital and parietal, as well as two cases associated with damage to temporal cortex. Some cases reported other impairments, including impaired memory (n=12), prosopagnosia (n=5) and navigational difficulties (n=2). Of cases who reported on their dreams (n=28), around half had lost visual dreaming, a third had preserved visual dreaming and the remainder had visual dreaming of reduced intensity.

Conclusions To our knowledge, this is the largest reported case series of acquired loss of visual imagery. Both neurological and psychological disorders can be responsible for acquired aphantasia. Our series includes cases of probable functional aphantasia. Further detailed analysis of these cases is required.

REFERENCES

1. Pearson. *Nat. Rev. Neurosci*. 2019;**20**:624634.
2. Zago, *et al*. *Cognit. Neuropsychiatry* 2011;**16**:481504.
3. Farah. *Cognition* 1984;**18**:245272.
4. Bartolomeo. *Cortex* 2002;**38**:357378.
5. Zeman, *et al*. *Cortex* 2020;**130**:426440.

#3079 INVESTIGATING THE FEASIBILITY OF AUTOMATING THE DIFFERENTIAL DIAGNOSIS OF TRANSIENT LOSS OF CONSCIOUSNESS

[1]Nathan Pevy, [2]Heidi Christensen, [3]Traci Walker, [4]Markus Reuber. *[1]Sheffield Institute of Translational Neuroscience; [2]The Department of Computer Science; [3]The Division of Human Communication Sciences; [4]Academic Neurology Unit, Royal Hallamshire Hospital*

10.1136/jnnp-2021-BNPA.18

Background There are three common causes of Transient Loss of Consciousness (TLOC), syncope, epileptic and psychogenic nonepileptic seizures (PNES). Many individuals who have experienced TLOC initially receive an incorrect diagnosis and inappropriate treatment. Whereas syncope can be distinguished from the other two causes relatively easily with a small number of yes/no questions, the differentiation of the other two causes of TLOC is more challenging. Previous qualitative research based on the methodology of Conversation Analysis has demonstrated that epileptic and nonepileptic seizures are described differently when patients talk to clinicians about their TLOC experiences. One particularly prominent difference is that epileptic seizure descriptions are characterised by more formulation effort than accounts of nonepileptic seizures.

Aim This research investigates whether features likely to reflect the level of formulation effort can be automatically elicited from audio recordings and transcripts of speech and used to differentiate between epileptic and nonepileptic seizures.

Method Verbatim transcripts of conversations between patients and neurologists were manually produced from video and audio recordings of interactions with 45 patients (21 epilepsy and24 PNES). The subsection of each transcript containing the patients account of their first seizure was manually extracted for the analysis. Seven automatically detectable features were designed as markers of formulation effort. These features were used to train a Random Forest machine learning classifier.

Results There were significantly more hesitations and repetitions in descriptions of first epileptic than nonepileptic seizures. Using a nested leave-one-out cross validation approach, 71% of seizures were correctly classified by the Random Forest classifier.

Conclusions This pilot study provides proof of principle that linguistic features that have been automatically extracted from audio recordings and transcripts could be used to distinguish between epileptic seizures and PNES and thereby contribute to the differential diagnosis of TLOC. Future research should explore whether additional observations can be incorporated into a diagnostic stratification tool. Moreover, future research should explore the performance of these features when they have been extracted from transcripts produced by automatic speech recognition and when they are combined with additional information provided by patients and witnesses about seizure manifestations and medical history.

#3085 RELATIONSHIP BETWEEN VARIANT CONNECTIVE TISSUE (HYPERMOBILITY) AND AUTISM SENSORY PROCESSING: EXTERNALLY ORIENTED THINKING AS A MEDIATOR

[1,2]GK Savage, [1,2]JLL Csecs, [1,2]G Davies, [1,2,3]HD Critchley, [1,2]JA Eccles. *[1]Department of Neuroscience, Brighton and Sussex Medical School, UK; [2]Sussex Partnership NHS Foundation Trust, Sussex, UK; [3]Sackler Centre for Consciousness Science, University of Sussex, UK*

10.1136/jnnp-2021-BNPA.19

Objectives/Aims Autism is a neurodevelopmental condition characterised by differences in sensory processing, social communication and restricted/repetitive behaviors. Joint hypermobility is a common connective tissue variant, reportedly overrepresented in Autism. Alexithymia is a personality construct characterised by altered emotional awareness which has notably high rates of overlap with autism spectrum disorder. This study tested whether hypermobility was associated with autistic traits and examined alexithymia as a mediator of this association.

Method Forty-two people underwent eligibility assessment for a study of joint hypermobility and anxiety (ISRCTN17018615). Hypermobility was assessed using both the Brighton Criteria for Joint Hypermobilty Syndrome (JHS) and 2017 Hypermobile Ehlers Danlos Syndrome (hEDS) Criteria. Participants completed the Ritvo Autism Asperger