Results For three, wheelchair-dependent patients with FND and FEVD admitted to The Wolfson Neuro-rehabilitation Centre for 12-24 week inpatient treatment, our approach resulted in two walking independently and one with supervision. Care needs were reduced and wheelchair dependence was eliminated. Patients reported improvement in independence and quality of life with one patient returning to part-time employment as a PA (See Table 1).

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

Objectives/aims The importance of identifying emerging rather than widespread amyloid pathology is highlighted by evidence showing that rapid amyloid accumulation associates with cortical atrophy, cognitive deficits and tau deposition even where individuals are in the 'amyloid negative' range. The study aimed to test the hypothesis that among cognitively healthy individuals distinct groups exist in terms of amyloid and phosphorylated tau accumulation rates. We also hypothesised that membership to the faster accumulator group can be predicted (using age, genetics, cognition and hippocampal volume). Finally we aimed to identify time points of significant increase in the rate of AD protein accumulation and the associated predictors.

Methods The analysis reports data from 263 individuals from the BIOCARD NIH-funded study who had a mean of 2.23 cerebrospinal fluid (CSF) measurements since the study began in 1995. We used latent class mixed-effect models to identify distinct classes in terms of amyloid and p-tau accumulation rates. To investigate non-linear changes of AD-related CSF biomarkers we used generalised additive modelling. For both analyses demographic, genetic (APOE4 genotype) as well as functional correlates in LE.

Methods We analysed acute neuroradiological reports, clinical notes, scores on post-acute neuropsychological tests and self-administered questionnaires on mood, emotion, and affect (including the Centre for Neurologic Study-Lability Scale; CNS-LS), along with structural MRI and resting-state fMRI datasets in relation to emotional lability in a large cohort of patients (n=36) that had received a neurological diagnosis of LE, presented with focal hippocampal structural abnormalities in the acute phase, and post-acute hippocampal atrophy andthalamic volume reduction.

Results Emotional lability was present in 50% of the patients. It was associated with increased tearfulness compared with non-labile patients and healthy controls, whereas no patient presented with labile laughter (CNS-LS). Patients with emotional lability (n=18) did not differ from those without (n=18) in any demographic or clinical details in their acute or post-acute presentation (autoantibodies, immunosuppressive therapy, seizures, antidepressant medication, age at or delay from symptom onset), or in residual depression, anxiety, impulsiveness, memory impairment, or executive dysfunction, or in hippocampal and thalamic volumes. Instead, the presence and extent of emotional lability across patients was associated with reduced resting-state hemodynamic activity in and hippocampal functional connectivity with regions in the inferior and superior parietal lobules.

Conclusions We present the first investigation of persistent affective dysregulation in LE. Emotional lability is common following LE, but is not a manifestation of depression, anxiety, impulsiveness, or executive dysfunction. The type of

Abstracts

31 PREDICTION OF EMERGING AMYLOID AND TAU PATHOLOGY IN COGNITIVELY HEALTHY INDIVIDUALS: A LONGITUDINAL CSF STUDY

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Objective/Aims Autoimmune limbic encephalitis (LE) is commonly associated with cognitive and psychiatric disturbances at the acute stage of the disease, and with residual episodic memory impairment. While behavioural and psychiatric symptoms generally dissipate post-acutely, very little is known about the profile of persistent neuropsychiatric symptoms. In particular, emotional lability represents an elusive entity that may be missed as a manifestation of comorbid mood or personality change and can have disabling consequences, due to the stigma attached to the loss of emotional control. We aimed to assess the post-acute profile of emotional lability and its neuroanatomical correlates in LE.

Methods We analysed acute neuropsychological reports, clinical notes, scores on post-acute neuropsychological tests and self-administered questionnaires on mood, emotion, and affect (including the Centre for Neurologic Study-Lability Scale; CNS-LS), along with structural MRI and resting-state fMRI datasets in relation to emotional lability in a large cohort of patients (n=36) that had received a neurological diagnosis of LE, presented with focal hippocampal structural abnormalities in the acute phase, and post-acute hippocampal atrophy and thalamic volume reduction.

Results Emotional lability was present in 50% of the patients. It was associated with increased tearfulness compared with non-labile patients and healthy controls, whereas no patient presented with labile laughter (CNS-LS). Patients with emotional lability (n=18) did not differ from those without (n=18) in any demographic or clinical details in their acute or post-acute presentation (autoantibodies, immunosuppressive therapy, seizures, antidepressant medication, age at or delay from symptom onset), or in residual depression, anxiety, impulsiveness, memory impairment, or executive dysfunction, or in hippocampal and thalamic volumes. Instead, the presence and extent of emotional lability across patients was associated with reduced resting-state hemodynamic activity in and hippocampal functional connectivity with regions in the inferior and superior parietal lobules.

Conclusions We present the first investigation of persistent affective dysregulation in LE. Emotional lability is common following LE, but is not a manifestation of depression, anxiety, impulsiveness, or executive dysfunction. The type of

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emotional lability seen in LE is semilogically distinct from pseudobulbar affect observed in other neurological diseases. While LE is characterised by focal hippocampal atrophy, functional abnormalities in regions interacting with the hippocampus may provide a more parsimonious explanation of emotional lability than the volume of medial temporal lobe structures. Functional abnormalities in parietal regions supporting perspective taking and social-affective processing may compromise patients’ emotion regulation.

### Objectives/Aims
This aim of case report is to discuss the clinical conundrum and diagnostic challenges in a young patient presenting with First Episode Psychosis. Investigations revealed Extensive Leukencephalopathy due to a rare metabolic disorder- 3-methylglutaconic aciduria (3-MGA) type IV. Several studies have shown that 3-MGA type IV can present with psychosis, epilepsy or depression as part of the spectrum of symptoms. The role of Organic Brain condition in the onset of first episode psychosis in this patient is discussed in this report.

### Methods
A 23-year-old female patient was admitted under Section 2 of the Mental Health Act, she was reflecting auditory hallucinations and paranoid delusions. Investigations revealed Extensive Leukencephalopathy due to a rare metabolic disorder- 3-methylglutaconic aciduria (3-MGA) type IV confirmed on urine testing. The patient was admitted to the Neurology service and an EEG showed a mild degree of general cerebral dysfunction without interictal epileptiform activity. There was no correlation found between her clinical symptoms of acute onset psychosis and her diagnosis of 3-MGA Type IV.

### Results
MRI scan showed extensive diffuse leukencephalopathy. Comparison to MRI scan done 6 years previously did not show any change or progression to the white matter lesions. An EEG showed a mild degree of general cerebral dysfunction without interictal epileptiform activity. There was no correlation found between her clinical symptoms of acute onset psychosis and her diagnosis of 3-MGA Type IV. She was commenced on Aripiprazole and her presentation improved significantly. Both Auditory hallucinations and Paranoid delusions improved considerably, with moderate improvement in mood, affect and apathy. Some Catatonic symptoms persisted but were less intense. She was given a diagnosis of Undifferentiated Schizophrenia under ICD 10, as she displayed features of Paranoid, Catatonic and Catatonic without clear predominance of particular subtype of Schizophrenia. She was discharged home with follow-up from Neuropsychiatry and community Mental health teams. She continues to be investigated for the genetic cause of 3-methylglutaconic aciduria Type 4.

### Conclusions
To conclude, although often metabolic disorders, including 3-methylglutaconic aciduria, can present with psychosis, it is prudent to establish a causative link in order to manage appropriately and effectively.

### References