Results The most common reasons for patients not accepting a CBT referral were referral to the Trust's inpatient neuropsychiatry ward, or non-attendance at assessment or treatment sessions. We identified a total of 98 patients with mFND and 76 ONP patients treated with CBT. 56.1% of mFND and 56.6% of ONP patients attended all sessions offered. There were no socio-demographic characteristics associated with mFND patients who completed all therapy sessions versus those who dropped out early. Compared to controls, mFND patients were more likely to be female (72.4% v. 44.7%, χ^2 : 13.6, 95% CI: 12.2–41.9, $p=0.001$), unemployed (52.6% versus 35.5%, χ^2 : 5, 95% CI: 2.2–30.8, $p=0.03$), to have a carer (27.6% versus 14.3%, χ^2 : 4.4, 95% CI: 0.9–24.7, $p=0.04$) and to have experienced child sexual abuse (23.8% v 8.2%, χ^2 : 7.3, $p=0.01$). The most common mFND symptom was weakness (26.9%), most frequently in the leg or entire body. Both mFND and ONP patients showed significant improvements in measures of psychological functioning (measured with the CORE-OM, HoNOS-ABI, and PHQ-9), with physical symptoms improving in 49.4% of mFND patients. A logistic regression showed acceptance of psychological formulations prior to CBT ($p<0.02$) was associated with the improvement of physical symptoms in mFND patients.

Conclusions mFND patients treated in a specialist CBT clinic show similar improvements in physical and psychological functioning to ONP patients with positive effects on motor symptoms, distress, depression, general health and social functioning. With little evidence on the effectiveness of CBT in treating motor variants of FND, this study could help inform the design of a pilot RCT.

38 ARE THERE ANY FACTORS THAT PREDICT THE DIAGNOSIS OF EPILEPSY OR PSYCHOGENIC NON-EPILEPTIC SEIZURES (PNES) IN PATIENTS ADMITTED TO A SPECIALIST EPILEPSY UNIT?

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Introduction Differentiating between epilepsy and psychogenic non-epileptic seizures (PNES) can be difficult. Although clearly not a substitute for taking a careful history, certain patient characteristics may assist the clinicians towards diagnosis. The population of patients referred to an epilepsy specialist centre represent a complex and distinct group of patients and it is not clear which factors, if any, could point towards a diagnosis of epilepsy or PNES.

Aims/Objectives To investigate if the diagnosis of epilepsy or PNES is predicted by baseline demographic and clinical variables, including Hospital Anxiety and Depression (HADS) scores and medication prescription, in patients admitted to a specialist adult epilepsy centre.

Methods We conducted an observational retrospective cohort of consecutive patients admitted to the William Quarrier Scottish Epilepsy Centre (WQSEC) over a period of one year (01/09/16–01/09/17). Chosen predictor variables at baseline included: sex, age, employment education or training (EET), Scottish Index of Multiple Deprivation 2016 (SIMD 2016) rank status, attack frequency, length of index admission, number of anti-epileptic agents prescribed, prescription of benzodiazepines, of analgesia, or of psychotropic medications, and

HADS scores. Outcome measures were diagnosis of epilepsy or PNES, from diagnosis made by expert clinicians on discharge from index admission. Because of the presence of dual diagnosis, two multivariable binary logistic regression models were built – one for the epilepsy and one for the PNES diagnosis outcomes.

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

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

39 THE IMMUNOMODULATORY EFFECT OF CLOZAPINE IN PATIENTS WITH TREATMENT RESISTANT SCHIZOPHRENIA: A RETROSPECTIVE COHORT STUDY

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

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

Results Of 188 included patients, 114 (61%) responded to treatment. Response did not significantly vary by ethnicity or age. Mean interval between haematological assessments was 6.9 (*SD* 2.0) days. General linear models revealed a significant increase from baseline for all cell lines in the second

treatment week ($p=0.001$) which persisted for three weeks ($p=0.001$). Group based trajectory modelling indicated that 15% of patients showed a temporary increase in white cell and neutrophil trajectories. Logistic regression revealed that treatment response was not associated with a 'spike' in cell count. However, females were 2.08 times more likely to respond to treatment (95% CI [1.09, 4.08], $p=0.017$) and 2.67 times more likely to exhibit a 'spike' in cell counts following Clozapine initiation (95% CI [1.14, 6.24], $p=0.023$).

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

40 A MULTI-SPECIALITY CONUNDRUM: NEUROPSYCHIATRIC SEQUELAE OF THYROTOXICOSIS

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

On examination in his stupor, he was seen to be lying in bed with his eyes closed, opening them a fraction to voice. He was sometimes able to follow simple commands but this was slow and inconsistent. He was unable to communicate through head

movements or blinking and stared with a fixed expression. There was waxy flexibility of his limbs and arching on the back at regular intervals. Power was normal with flexor plantars. There were no ictal signs (i.e. no nystagmus/gaze deviation/twitching/dystonia). When agitated he was seen to have echopraxia. He consistently had hyperhidrosis and tachycardia.

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

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

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

41 KORSAKOFF'S SYNDROME: NEUROCOGNITIVE DOMAINS IMPAIRMENTS AND POTENTIAL THERAPEUTIC INTERVENTIONS

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Objectives/aims To explore the burden of neurocognitive impairments in a cohort of Individuals with Korsakoff's psychosis requiring current long stay psychiatric care. To consider additional therapeutic interventions to target the health care burden potentially created by these comorbidities.

Abstract 41 Table 1 DSM 5 Neurocognitive Domains

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

*TBI Co Morbidity

** Co Morbid Intellectual Disability