**TMS-EEG indexes abnormal GABAergic signalling in patients with schizophrenia**

Sukhwinder S Shergill1*, Viviana Santoro2, Lorenzo Rocchi3, Meng Di Hou1 and Isabella Premoli2

1Department of Psychosis Studies, Institute of Psychiatry, Psychology and Neuroscience (IoPPN), King’s College London; 2Department of Basic and Clinical Neuroscience, Institute of Psychiatry, Psychology and Neuroscience, King’s College London and 3Department of Clinical and Movement Neurosciences UCL Queen Square Institute of Neurology, University College London

*Corresponding author.

doi: 10.1192/bjo.2021.185

**Aims.** Transcranial magnetic stimulation (TMS) is a non-invasive brain stimulation tool designed to probe the strength of inhibitory and excitatory neurotransmission in the cortex. Combined with electromyography, paired-pulse TMS paradigms have revealed a deficit in inhibition mediated by GABA-A receptors in patients with schizophrenia. Combined TMS-electroencephalography (TMS-EEG) provides a more detailed examination of cortical excitability and may shed more light into the pathophysiology of schizophrenia. Of the various peaks of the TMS-evoked EEG signal, responses at 45 (N45) and 100 ms (N100) likely reflect GABA-A and GABA-B receptor-mediated inhibition, respectively. Responses at 25 ms (P25) are affected by voltage-gated channel ligands, whereas glutamatergic processes may be related to the P70 component. We here aim to systematically investigate the role of these neural processes in patients with schizophrenia by using TMS-EEG.

**Method.** TMS-evoked EEG potentials (TEPs) were recorded in patients with schizophrenia (n = 19) and in age-matched healthy controls (n = 17). 150 TMS pulses at 90% of resting motor threshold were applied over the left primary motor cortex during EEG recording. Differences in TEPs between the two groups were analysed for all electrodes and for time windows corresponding to each TEP (P25: 0.015-0.035 ms; N45: 0.035-0.06 ms; P70: 0.035-0.06 ms; N100: 0.09-0.14 ms) by applying multiple independent sample t-tests. Further, a cluster-based permutation analysis approach was implemented to correct for multiple comparisons.

**Result.** Compared to controls, patients showed amplitude reduction for the P25 (negative and positive cluster; p < 0.001 and p = 0.04, respectively), N45 (negative and positive cluster; p < 0.001 and p = 0.001, respectively) and P70 component (negative and positive cluster; p = 0.04 and p = 0.004, respectively).

**Conclusion.** There results extend on previous literature about impairment of GABA-A receptor mediated inhibition in schizophrenia, as demonstrated by the N45 amplitude reduction, whereas no significant differences in GABA-B index (i.e., N100) were revealed. Our results also showed that, although specific mechanisms underlying P25 and P70 have not been fully elucidated yet, excitatory neurotransmission is altered in this clinical population. To conclude, TMS-EEG may provide a more comprehensive view of the inhibitory and excitatory mechanisms involved in the pathophysiology of schizophrenia.

---

**Cardiovascular risk quantification using QRISK-3 score in people with intellectual disability**

Jamie Sin Ying Ho1*, Vikram Rohra2, Laura Korb1 and Bhathika Perera1

1Haringey Learning Disability Partnership, Barnet; Enfield and Haringey Mental Health Trust and 2North Middlesex University Hospital NHS Trust

*Corresponding author.

doi: 10.1192/bjo.2021.187

**Aims.** The prevalence of cardiovascular diseases (CVD) in people with intellectual disability (ID) is around 14%, higher than the general population. However, CVD risk assessments are not consistently performed. Given the high risk of premature deaths in people with ID, it is important to identify preventable risk factors and follow evidence-based interventions. QRISK-3 is a validated risk-stratification tool, which calculates the 10-year risk of developing a heart attack or stroke (https://qrisk.org/three/index.php). There are no published studies on the use of QRISK-3 in people with ID. This project aimed to

---

**Anxiety levels during COVID 19 pandemic in primary and secondary doctors in UK**

Dr Shweta Mittal1* and Abdalla Abouebeid2

1Nottinghamshire Healthcare NHS Foundation Trust, Bassetlaw Mental Health Services and 2The St. Vincent Practice

*Corresponding author.

doi: 10.1192/bjo.2021.186
understand the use of QRISK-3 in an ID clinic and to quantify individual CVD risks to recommend appropriate management options. **Method.** A cross sectional study was performed on 143 patients open to an ID psychiatry clinic. Patients and carers were sent an accessible information leaflet on this study. Basic demographic data and information on psychiatric diagnoses were collected. Patients were grouped according to the presence of severe mental illness (SMI) defined as schizophrenia, bipolar disorder and other psychotic illnesses. QRISK-3 ≥ 10% was defined as elevated risk in accordance with NICE guidelines. Patients who had a high QRISK-3 score were advised to contact their GP. **Result.** Of 143 patients, 73 (51.0%) had a mild ID and the remaining had a moderate to severe ID. The mean age was 43.3 years, 53.1% were male. Overall, 28 (19.6%) participants had an elevated CVD risk, of whom 16 (57.1%) were not on statins, which is the recommended treatment. The mean QRISK-3 score was 6.31 (standard deviation [SD] 8.95), and the relative risk is 3.50 (SD 7.13). The proportion of QRISK-3 ≥ 10% and mean score were not significantly different in those with SMI, but those with SMI were more likely to be prescribed statins than those without (14 [31.1%] vs 10 [10.2%], p = 0.002). Statins were given to 24 (16.8%) participants, of whom 12 (50%) had elevated CVD risk, 89% had a blood pressure recording within the past 5 years, 87% had height and 88% had weight recorded. 73% had lipid serology results recorded. **Conclusion.** Elevated CVD risk was common in this ID study population, and more than half with elevated QRISK-3 were not on the medical treatment recommended by national guidelines. QRISK-3 could feasibly be implemented in the outpatient setting. Increased routine CVD risk assessment and management should be considered as another measure to reduce morbidity and mortality.

**A case of olanzapine-associated rhabdomyolysis**

Valentin Skryabin1*, Michael Zastrozhin1, Dmitry Sychev2 and Evgeny Bryun3

1Moscow Research and Practical Centre on Addictions of the Moscow Department of Healthcare, Russian Medical Academy of Continuous Professional Education of the Ministry of Health of the Russian Federation; 2Russian Medical Academy of Continuous Professional Education of the Ministry of Health of the Russian Federation and 3Moscow Research and Practical Centre on Addictions of the Moscow Department of Healthcare

*Corresponding author.

doi: 10.1192/bjo.2021.188

**Aims.** To describe the case of olanzapine-associated rhabdomyolysis in a 20-year-old patient with a suspected diagnosis of paranoid schizophrenia. **Method.** A 20-year-old male Caucasian patient was admitted to the Psychiatric Department with a one-month history of irritable behavior, talking to himself, persecutory delusions, and poor sleep. He was prescribed oral olanzapine at a dose of 10 mg per day. After two days of olanzapine monotherapy, the patient experienced muscle jerks in the legs. Four days after the initiation of olanzapine treatment, he complained about fatigue and weakness in the lower extremities along with myalgia. Physical examination revealed decreased muscle power with no extrapyramidal symptoms. Blood chemistry showed serum creatine kinase (CK) and serum lactate dehydrogenase (LDH) of 9,725 U/L and 843 U/L, respectively, on day four of the therapy. The Naranjo algorithm score of 6 suggested that olanzapine was the probable cause of rhabdomyolysis. A diagnosis of drug-induced rhabdomyolysis was established from the background of blood tests (increased serum CK and LDH levels), clinical presentation (fatigue and weakness in the lower extremities, muscle jerks, and myalgia), and Naranjo algorithm score of 6 for olanzapine. On suspicion of its contribution to rhabdomyolysis, olanzapine was immediately withdrawn. The patient was referred to the intensive care unit. To prevent acute renal failure, high-volume alkaline diuresis was initiated. After consulting a clinical pharmacologist, the patient’s primary physician decided to perform a pharmacogenetic test to develop an individualized treatment regimen. Pharmacogenetic test results were interpreted using the PGX2 software (Meditsina LLC, Moscow, Russia). The test revealed that the patient was a homozygous mutant for CYP2D6*, which corresponds to CYP2D6 PM phenotype. With this in mind, triluoperazine was prescribed at a daily dose of 10 mg instead of olanzapine as recent data indicate that triluoperazine is metabolized by CYP1A2 and UGT1A4 instead of CYP2D6. Subsequently, the patient recovered well and was discharged without any nephrological sequelae. **Result.** Recent research demonstrates that CYP2D6 is one of the most important isoenzymes implicated in drug metabolism because the CYP2D6 gene is highly polymorphic. Few reports on the association between olanzapine use and rhabdomyolysis have been published to date, and the present case report draws attention to pharmacogenetic testing which allowed the psychiatrist to prescribe another antipsychotic with no risk of rhabdomyolysis. **Conclusion.** The presented case demonstrates that pharmacogenetic-guided personalization of treatment may allow selecting the best medication and determining the right dosage, resulting in the reduced risk of adverse drug reactions and pharmacoresistance.

**Effects of tailored quality improvement programme for effective medication management in high dependency in-patient psychiatry rehabilitation unit**

Hina Tahseen* and Jade Brown

Delfryn House, Cygnet Health Care

*Corresponding author.

doi: 10.1192/bjo.2021.190

**Aims.** To determine the effects of a tailored quality improvement programme for effective medication management including a reduction in prescription and administration errors in oral and depot psychotropic medication, patient education on medication and implementation of policies and guidelines. **Background.** Medication errors are common in hospital admissions and pose a threat to patient safety (Buckley et al. 2013). Medication errors may occur in different stages of the patient treatment process such as during prescribing, transcribing, preparing, dispensing, administration, and monitoring (Wang et al. 2015). In addition to these, for the detained mental health patients, the Mental Health Act 1983 legislation requires up-to-date treatment certificate compliance (Wales. Welsh Assembly 2008). A Quality Improvement programme to improve safe medication prescription and administration was designed for the patients admitted in Delfryn House, a mental health high dependency rehabilitation unit. **Method.** Using Plan-Do-Study-Act (PDSA) quality improvement methodology, a medication management committee was created under the leadership of Specialty doctor and Head of Care (HOC), and comprising of the consultant psychiatrists, specialty doctor, heads of care (ward managers), senior nurses,