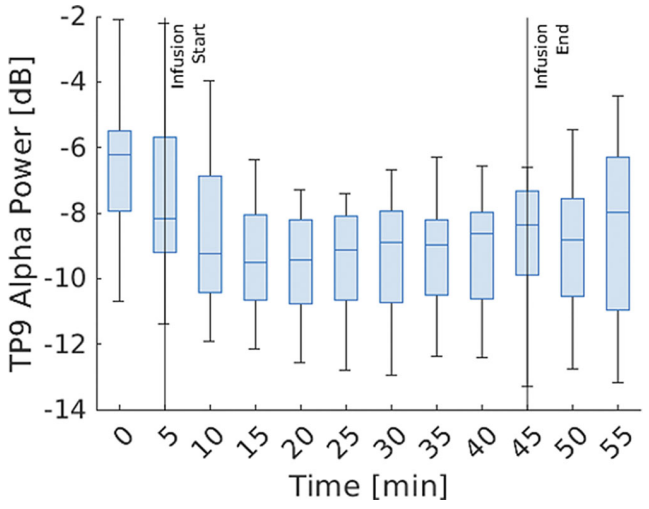
Objectives: Assess the feasibility of identifying EEG correlates of ketamine infusions in a routine outpatient setting with a low-cost, easily usable system.
Methods: The study was carried out at the Oxford Health Foundation Trust Ketamine Clinic (ethics reference 22/EM/0226). N=18 EEG recordings from N=12 patients were collected (5 women, mean age 44, range 33-62, IV dose 0.5-1mg/kg over 40min). 4-channel EEG was collected with a Muse-S headband at 256Hz, from 5min before to 55min after infusion start. 5s epochs were rejected if gyroscope data indicated head movement above 10 deg/s or if amplitude was above 200µV. A spectrogram (4s window, 3s overlap) as well as band-limited power (theta: 4-8Hz, alpha: 8-13Hz, beta: 13-25Hz) were computed. Significance of changes was found with a repeated measures analysis of variance (RM-ANOVA) on power in 5min segments together with post-hoc Tukey's P-values.
Results: Across the ketamine infusion recordings, there was a significant effect of time (F=3.65, P=0.0105) and Channel*Time interaction (F=3.80, P<0.001) on the EEG spectrum. Effects were largest on temporal electrodes, particularly TP9 in the alpha and theta bands (Figure 1, Table 1).

Table 1: Effect sizes (Cohen's d) and FDR-corrected ANOVA P-values for ketamine effects on each EEG channel and frequency band. P<0.05 was considered significant (bold). n.s. = not significant (P>0.2).

Channel / Band	TP9	AF7	AF8	TP10
Theta	**1.16 (P=0.019)**	0.11 (n.s.)	0.11 (n.s.)	0.42 (P=0.113)
Alpha	**1.41 (P<0.001)**	0.12 (n.s.)	0.17 (n.s.)	0.605 (P=0.113)
Beta	1.19 (P=0.112)	0.03 (n.s.)	0.08 (n.s.)	0.21 (n.s.)

Image:



Conclusions: In a routine outpatient setting, sub-anaesthetic ketamine infusions in TRD patients were associated with decreased fronto-temporal EEG alpha and theta power. Future work should assess the potential of low-cost routine EEG, and alpha desaturation specifically, to inform individualised ketamine treatment.

Disclosure of Interest: None Declared

EPP0696

The primary motor cortex of schizophrenia patients show neuronal and subcellular impairments in the right hemisphere – postmortem study

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doi: 10.1192/j.eurpsy.2024.779

Introduction: In mental disorders, very little is known about the cellular and subcellular mechanisms underlying the development of symptoms. Postmortem studies can contribute to understanding these. Our research group collects and studies cortical samples with short postmortem intervals from schizophrenia patients.
Objectives: We investigated primary motor cortical brain samples, to understand the background of motor symptoms in schizophrenia.
Methods: Both hemispheres of primary motor cortices of eight control- and eight subjects with schizophrenia were analysed by immunohistochemistry. We labelled pyramidal cells with SMI32 antibody, which binds to neurofilaments, and parvalbumin (PV) antibody, which labels one type of inhibitory input on these cells, axo-axonic and axo-somatic interneurons, and a proportion of giant pyramidal neurons (Betz cells). We were interested in the size and density of layer 3 and 5 pyramidal cells and Betz cells, the distribution of PV-labelled terminals and the PV expression of Betz cells. Results of the subjects were compared both as a whole and separately per hemisphere.
Results: Most changes were present in the primary motor cortices in the right hemisphere (presumably subdominant). Here, the density of Betz cells and their inhibitory inputs were also reduced. PV-expression of Betz cells was not dependent on the group studied, but we observed that it is decreasing with age. The other investigated characteristics show no significant differences.
Conclusions: Our results suggest that the primary motor cortex may be involved in schizophrenia. Neurodevelopmental, pharmacological and neurodegenerative causes could be involved in this process. Network dysconnectivity is likely to underlie the stronger involvement of the subdominant side, and literature data point also in this direction. We believe that our research method is suitable for the study of the background of other symptoms and may lead to a better understanding of schizophrenia, especially if we could combine our results with clinical research.

Disclosure of Interest: None Declared