(thrombolysis or thrombectomy) for patients in each deprivation quintile compared to the least deprived quintile. Results: We identified 57,709 patients (median age 74 years; 45.9% female). Compared to patients in the least deprived quintile, those with higher deprivation were younger and more likely to have hypertension and diabetes, but less likely to have atrial fibrillation. Compared to patients in the least deprived quintile, fewer patients in the very deprived quintile (17.9% vs 19.6%, aOR 0.88, 95%CI [0.82,0.95]) and in the most deprived quintile (16.6% vs 19.6%, 0.77 [0.71,0.83]) received revascularization treatments. Conclusions: Our results suggest disparities in the use of acute ischemic stroke revascularization treatments by socioeconomic status despite access to universal health care.

GR.4

Neurophysiological and clinical effects of low-intensity transcranial ultrasound of the motor cortex in Parkinson's disease

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Background: Low-intensity transcranial ultrasound (TUS) is a non-invasive neuromodulation technique, which in theta burst mode (tbTUS) can increase cortical excitability. Parkinson's disease (PD) has altered cortical excitability of motor cortex (M1). We evaluated the neurophysiological and clinical effects of M1 tbTUS in PD patients. Methods: Sixteen PD patients (4F, 59.5±9.7 years) in ON and OFF dopaminergic medication states, and 15 controls (5F, 61.9 ±8.7 years) were evaluated. tbTUS was applied for 80 seconds at M1 with 20W/cm^2. Motor evoked potential (MEP) was recorded at baseline, at 5-minutes (T5), T30, and T60 after tbTUS. Motor (m) UPDRS was evaluated in PD at baseline and T60. Results: A linear mixed model on MEP amplitudes comparing PD-ON, PD-OFF and controls showed significant effect of time (F=4.83, p=0.003). Post-hoc analysis showed significant difference between baseline and T30 timepoints (p=0.0003). The MEP increase at T30 was higher in controls (66%), followed by PD-ON (41%) and PD-OFF (21%). PD-ON showed reduced mUPDRS at T60 when compared to PD-OFF, with significant effect of time (F=6.14, p=0.017) and group (F=5.39, p=0.025). Conclusions: tbTUS induced motor cortical plasticity is reduced in PD-OFF, that is partially restored by dopaminergic medications.Repeated sessions of tbTUS can be further investigated as a novel non-invasive treatment for PD.

GR.5

Incidence of orbital infarction syndrome following endovascular thrombectomy

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Background: Orbital infarction syndrome (OIS) is a rare entity defined as acute ischemia of intraorbital structures. Three case reports of OIS post-endovascular thrombectomy (EVT) have recently been published, two demonstrating absent choroid blush (CB) on digital subtraction angiogram (DSA). Our goals are to determine the true incidence of OIS post-EVT and to identify imaging findings (e.g. CB) that may alert neurologists to potential cases. Methods: A retrospective cohort study including all EVT patients from Health Sciences Center (HSC), Winnipeg in 2019-20 was performed. Patient charts were reviewed to determine the incidence of OIS. Pre- and post-EVT DSA images were reviewed, and the sensitivity and specificity of absent CB for OIS was calculated. Results: Out of 248 patients, 13 were excluded for incomplete charts, and 4 cases (1.7%) of OIS were discovered. During sensitivity/specificity analysis of absent CB for OIS, 51 patients were excluded for inadequate imaging. There were 4 true positives, 0 false-negatives, 113 true-negatives, and 67 false-positives; resulting in a sensitivity of 100% and worst-case scenario specificity of 63% (assuming all 51 indeterminate cases were false positives). Conclusions: OIS is rare post-EVT with an incidence of 1.7%. Absent CB is very sensitive for diagnosing OIS with lower specificity.

GR.6

Harnessing the endogenous regenerative potential of the injured spinal cord

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Background: The adult spinal cord contains a population of ependymal-derived neural stem/progenitor cells (epNSPCs) with the potential to enhance endogenous regeneration. However, little is known about the mechanisms that regulate the activation of these cells after injury. Recently, we discovered that glutamate excitotoxicity, a hallmark in the pathophysiology of acute SCI, promotes epNSPC proliferation/survival. Here, we characterize the downstream signaling pathways involved in this response and target this mechanism *in vivo* to enhance the endogenous regenerative capacity of these cells. Methods: epNSPCs were isolated from the central canal region of the adult spinal cord. *In vitro* pathway analysis was conducted using immunohistochemistry, RNAseq and Western Blot. *In vivo*, rats underwent SCI and at 1-week post-injury were randomized to receive CX546 (positive AMPAR modulator), or vehicle-control. Animals underwent behavioural testing and spinal cords were extracted for analysis. Results: Glutamate excitotoxicity leads to calcium influx in epNSPCs via AMPARs and together with Notch signaling drives proliferation and astrocytic differentiation. Positive modulation of AMPARs subacutely after SCI enhances epNSPC proliferation, astrogliogenesis, neurotrophin production, neuronal survival and functional recovery. Conclusions: We uncover an important mechanism by which AMPARs regulate the growth/phenotype of epNSPCs which can be targeted therapeutically to harness the regenerative potential of the injured spinal cord.