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Seizure-induced neuroplasticity and cognitive network reorganization in epilepsy

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Epilepsy is a network disorder with properties that inherently generate neuroplasticity. Accordingly, even focal, lesional forms of the disorder initiate neuroplastic responses throughout large regions of cortex, disrupting a wide array of neurocognitive networks. I The author will focus on temporal lobe epilepsy, describing the cognitive reorganization emergent during the disease course, providing evidence from task-based Functional Magnetic Resonance Imaging (fMRI), resting-state functional connectivity, and diffusion tensor imaging for both intra- and inter-hemispheric shifts in cognitive representations. Factors that mediate changes in seizure and cognitive network organization are described, with a discussion of the neuroplastic responses that can emerge following a common treatment for the disease, i.e., anterior temporal lobectomy. I The author will argue that only by understanding and measuring the potential for neuroplasticity will we be able to effectively predict cognitive outcomes in epilepsy, as it is these neuroplastic responses that govern the status of both neurocognitive and epileptogenic networks post-surgery. Multi-modal imaging is discussed as a means of estimating neuroplastic potential, delineating the potential cognitive mechanisms that might be available to serve recovery and good better cognitive outcomes.

Efficacy of cilostazol + nimodipine on cerebral vasospasm following aneurysmal subarachnoid hemorrhage: A prospective, randomized, single-blind trial

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Introduction: Vasospasm has been one of the major complications of aneurysmal SAH that gives poorer prognosis amongst patients suffering from it. Although among cerebrovascular diseases, SAH accounts for only 2-5%, a higher mortality and morbidity rate is accounted for by its complications, i.e. vasospasm.

Objective: This clinical trial aimed to determine the efficacy of oral nimodipine+cilostazol in reducing vasospasm following aneurysmal SAH as compared to using nimodipine alone.

Design: This is an on-going prospective, randomized, single- blind, with intention-to-treat analysis. To have 95% chances, with a significance level of 0.05, of detecting a 50% reduction in an incidence of cerebral vasospasm following aneurysmal SAH, a minimum of 44 patients were required. As of the time being, only 14 patients were recruited.

Patients and randomization: A computer generated ID was drawn to identify to which treatment a patient will be grouped.

Intervention: Group A received nimodipine 60mg q4 x 21days alone, while Group B received nimodipine 60mg q4 x 21days plus cilostazol 100mg q12 x 14days.

Monitoring and end points: Monitoring of the Lindegaard index (LI) from days 4 to 14 post ictus was done by a blinded technician. An LI>3 indicates vasospasm (primary end point).

Preliminary results: This is an on-going study; hence statistical analyses have not been employed due to insufficient number of samples recruited at the present time being. Initial findings revealed lower mean values for LI of patients taking both nimodipine+cilostazol compared to those on nimodipine alone. Also, symptomatic vasospasm occurred more on those taking nimodipine alone.

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