Brain damage affects all three levels of structural and functional organization: Cellular and molecular level, circuitries level and dynamic network level and launches an endogenous continuous brain defense response which consists both neuroprotection (the immediate response) and neurorecovery (a later response). Endogenous neuromodulation represents at the cellular and molecular level the optimization of common biological processes that could potentially generate cell death or promote neurodegeneration. At the circuitries and dynamic network levels, it represents the tendency in rebalancing of functional connectivity in resting-state networks. In the years, there has been a substantial effort in understanding the brain functioning and how to enhance endogenous neuromodulation and neurorehabilitation in general, by using a large spectrum of neurotechnologies such as imaging techniques (functional magnetic resonance imaging, ligand-based positron emission tomography, diffusion-tensor imaging), quantitative electroencephalogram, magnetoencephalography, eye tracking, optogenetics, transcranial magnetic stimulation, transcranial direct current stimulation, deep brain simulation, computational neuroscience and brain-computer interfaces. The combination between these technologies provide valuable information about the structure-function relationship underlying resting-state networks, about the dynamic cross-talk between networks and about the abnormalities in the functional connectivity in different pathologies. Neurorecovery can be enhanced by pharmacological intervention, physical activity, electromagnetic stimulation, psychological support, environmental stimulation or any demonstrated combinations of these factors capable of improving the patient's condition after brain and spinal cord injuries. From the pharmacological perspective, it is clear that the focusing on molecules that are capable of mimicking the function of endogenous molecules with multimodal and pleiotropic neuroprotective effects is the best approach in neurorecovery, especially when they are associated with intensive physical training. Biological agents (e.g., neurotrophic factors and related molecules) with modulating and multimodal effects are better pharmacological agents for brain and spinal cord protection and recovery, because they usually have also pleiotropic neuroprotective effect. That is why they are capable of pharmacologically bridging acute neuroprotective processes with the long-term recovery processes. There are many animal and human studies trying to elucidate the cellular and molecular mechanisms of plasticity of the nervous system. A better understanding of the mechanisms underlying the neuroplasticity will reflect in a more efficient and comprehensive treatment. This presentation will focus on the therapeutic effects of multimodal drugs on neurorecovery after brain lesion.

Alprazolam potentiates analgesic effect of Ibuprofen in postendodontic pain

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Postendodontic pain (PEP) has always been a major problem for patients and dentists and NSAIDs (Nonsteroidal anti-inflammatory drugs) are being used to relieve PEP and it is supposed that some benzodiazepines may potentiate the analgesic effects of NSAIDs. This study was conducted to evaluate the effect of alprazolam on the analgesic effect of Ibuprofen in PEP treatment. This randomized double-blind clinical trial was conducted on 45 patients aged 20-45 years who were subjected to root canal treatment. A written informed consent was obtained from each patient. The subjects were randomly divided into three groups; placebo, Ibuprofen (400 mg) and alprazolam (0.5 mg)+Ibuprofen (400 mg). The intensity of pain was recorded using visual analog scale (VAS) at 4, 6, 12, 24, 48 and 72 hours after drug administration. Of the participants, twenty six (57.8%) were males and 19 patients (42.2%) were females. Four hours after starting the treatment, the VAS scores in the placebo and Ibuprofen-treated groups were significantly higher than Ibuprofen and alprazolam+Ibuprofen groups (p<0.0001). The VAS scores in alprazolam+Ibuprofen group (2.33±1.05) were significantly lower at 6 hours after treatment when compared to the other groups. The average pain score in female patients who received alprazolam+Ibuprofen was significantly lower than males at 12 hours (1.3±0.6 vs 2.14±0.9, P=0.002) and 24 hours after treatment (0.88±0.6 vs 1.86±0.9, P=0.003). It could be concluded that alprazolam may enhance the analgesic efficacy of Ibuprofen in postendodontic pain.