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Stimulation of the Motor Cortex for Chronic Neuropathic Pain: Anatomo- Clinical Correlations

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The aim of this study was to search the relationship between the anatomical location of contactsand the eventual analgesic effect.

Materials and Methods: 22 patients suffering from central and / or peripheral neuropathic pain were implanted withextradural stimulation of the precentral cortex.

Implantation electrodes were performed using intraoperative:

1) Anatomical identification by Neuronavigation with 3D MRI,

2) Somestheticevoqued potentials monitoring,

3) Electricalstimulations to identify the motor responses.

In order to locate postoperatively the electrodes, a 3D-CT was performed and fused with thepreoperative MRI. The clinical analgesic effects of cortical stimulation were collected on aregular basis (VAS reduction > 50%, drugs consumption).

Results: Post implantation analgesic effects were obtained in 19 patients out of 22. The analgesic effectwas companied with reduction of the drugs consumption in 17 patients. The post-operative3 D CT analysis shows a correspondence between the effective contacts localization andthe motor cerebral cortex somatotopy in the patients with post-operative good analgesic effects. No correspondence was found between the contacts localization and the motor cerebral cortexsomatotopy in the 4 patients with no analgesic effects. In three out of these four patients,analgesic effects were obtained after a new surgery allowing a replacement of the electrodeposition over the motor cortex somatotopy corresponding to the painful area.

Conclusion: This study shows the correlation between position of the contact (cathode) overthe precentral cortex and the analgesia obtained when the somatotopy of the stimulated cortexcorrespond to the painful area.

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Presynaptic plasticity: A hiatus in neuroscience, a hotspot for Brain disorders

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Defects in synaptic strength and plasticity underlie many brain diseases, as illustrated by many recent findings from whole exome sequencing, but it remains a challenge to study the functional effect of clinically observed mutations in established models for neurotransmission. These studies typically focused on 'classical' fast neurotransmission that is evoked by actionpotentials (APs). We recentlydiscovered that AP-independent events, historically referred to as 'spontaneous' events, arein fact triggered by local Ca2+ increases that rely on specialized Ca2+ sensor proteins. In contrast to classical fast neurotransmission, genetic ablation of AP-independent neurotransmission produces viable neurologicalphenotypes and is therefore potentially relevant for disease gene characterization.

Using whole-cell patch clamp electrophysiology in mammalian neurons, we refined our current working model by incorporating the contributions of classical and AP-independent neurotransmission. This cellular model was applied to investigate the synaptic effect of clinically observed mutations implicated in movement disorders, epilepsies, cognitive and autism-related disorders. The resultsprovide valuable insight to expand our map of the presynaptic gene network and shed more light on the etiology of several brain disorders. The results may facilitate disease classification based on moleculardiagnostics in future health care.

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