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

Aff AFIF and Patrick MERTENS

Department of Neurosurgery, Pain center, Pierre WERTHEIMER Hospital, Hospices Civils de LYON, France

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

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

Implantation electrodes were performed using intraoperative:

- 1) Anatomical identification by Neuronavigation with 3D MRI,
- 2) Somesthetic evoked potentials monitoring,
- 3) Electrical stimulations to identify the motor responses.

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

Results: Post implantation analgesic effects were obtained in 19 patients out of 22. The analgesic effect was accompanied with reduction of the drugs consumption in 17 patients. The post-operative 3D CT analysis shows a correspondence between the effective contacts localization and the 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 cortex somatotopy 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) over the precentral cortex and the analgesia obtained when the somatotopy of the stimulated cortex corresponds to the painful area.

afif_acc@hotmail.com

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

Alexander J.A. Groffen

Dept. of Functional Genomics and Clinical Genetics, VU University and VU Medical Center, De Boelelaan 1085, 1081HV Amsterdam, The Netherlands

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 action potentials (APs). We recently discovered that AP-independent events, historically referred to as 'spontaneous' events, are in 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 neurological phenotypes 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 results provide 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 molecular diagnostics in future health care.

a.j.a.groffen@vu.nl