Primary motor area

The primary motor area in the posterior frontal lobe (Brodmann area 4) was first described by David Ferrier in 1874, who used electrical stimulation to map the cortex responsible for movement in monkeys.  

Since these initial findings, the description of this area has evolved to include the concept of somatotopy, wherein different parts of the body are represented individually on the primary motor cortex, as observed by Penfield and Boldrey.  

Location

This gyrus is located in the most posterior portion of the frontal lobe, lying immediately anterior to (in front of) the central sulcus, and extending from the apex of the brain down to the sylvian fissure, which delineates the temporal lobe.  

Histology

Large concentration of Giant Betz cells.  

Histologically, neurons in the precentral gyrus have a similar architecture and are denoted as Brodmann’s area 4. The functional organization of the PCG follows a topographical representation of an inverted homunculus (small human), such that the head and face regions are represented at the lowermost portion of the gyrus, the body and limbs extend toward the upper part of the gyrus, and the feet “dangle” over the apex of the brain.

Functions

Damage to the primary motor cortex, supplemental motor, and premotor areas lead to weakness and impaired execution of motor tasks of the contralateral side. The inferolateral areas of the dominant hemisphere are the expressive language area (Broca area, Brodmann areas 44 and 45), to which damage will result in a non-fluent expressive type of aphasia.

The primary motor cortex (M1) plays an important role in the control and execution of voluntary movements. Increasing evidence attributes this function to its connection to a wide range of cortical motor control areas including the premotor area (PMA), the supplementary motor area (SMA) as well as the parietal cortices that are involved in different aspects of a motor task e.g. motor execution, planning and coordination.

Within this network strongest activation during unilateral handmovements is usually found in the M1 motor hand representation contralateral to the executed movement (M1c) as demonstrated in various studies using blood oxygen level dependent (BOLD) functional MRI (fMRI).  

However, even though M1 activation is strongly lateralized to M1c also M1 coactivation ipsilateral to the executed movement (M1i-CoA) is present in healthy volunteers, and is especially thought to be present in patients with lesions in the primary motor cortex of the contralateral hemisphere.

Is the main contributor to generating neural impulses that pass down to the spinal cord and control the execution of movement. However, some of the other motor cortical fields also play a role in this function.
The modulatory role of the primary motor cortex (M1), reflected by an inhibitory effect of M1-stimulation on clinical pain, motivated us to deepen our understanding of M1's role in pain modulation. We used Transcranial Magnetic Stimulation (TMS)-induced virtual lesion (VL) to interrupt with M1 activity during noxious heat pain. We hypothesized that TMS-VL will effect experimental pain ratings. Three VL protocols were applied consisting of single-pulse TMS to transiently interfere with right M1 activity: (1) VLM1- TMS applied to 11 subjects, 20 msec before the individual's first pain-related M1 peak activation, as determined by source analysis (sLORETA), (2) VL-50 (N = 16; TMS applied 50 ms prior to noxious stimulus onset), and (3) VL+150 (N = 16; TMS applied 150 ms after noxious stimulus onset). Each protocol included 3 conditions ('pain-alone', 'TMS-VL', and 'SHAM-VL'), each consisted of 30 noxious heat stimuli. Pain ratings were compared, in each protocol, for TMS-VL vs. SHAM-VL and vs. pain-alone conditions. Repeated measures analysis of variance, corrected for multiple comparisons revealed no significant differences in the pain ratings between the different conditions within each protocol. Therefore, our results from this exploratory study suggest that a single pulse TMS-induced VL that is targeted to M1 failed to interrupt experimental pain processing in the specific three stimulation timing examined here.

Pathology

Lesions of the precentral gyrus may often be deemed unsafe for surgery given a perceived high risk of postoperative neurological complications.

see Precentral gyrus resection

see Precentral gyrus glioma

Shinoura et al., analyzed factors associated with worsened paresis at 1-month follow-up in patients with brain tumors located in the primary motor area (M1) to establish protocols for safe awake craniotomy for M1 lesions. Methods Patients with M1 brain tumors who underwent awake surgery in our hospital (n = 61) were evaluated before, during, and immediately and 1 month after surgery for severity of paresis, tumor location, extent of resection, complications, preoperative motor strength, histology, and operative strategies (surgery stopped or continued after deterioration of motor function). Results Worsened paresis at 1-month follow-up was significantly associated with worsened paresis immediately after surgery and also with operative strategy. Specifically, when motor function deteriorated during awake surgery and did not recover within 5 to 10 minutes, no deterioration was observed at 1-month follow-up in cases where we stopped surgery, whereas 6 of 13 cases showed deteriorated motor function at 1-month follow-up in cases where we continued surgery. Conclusion Stopping tumor resection on deterioration of motor function during awake surgery may help prevent worsened paresis at 1-month follow-up.


