# Tactile information processing associated with the primate hand is altered by motor cortex lesion

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## Introduction

- Tactile information from the fingertips is crucial for motor control underlying manual dexterity (Smith, 2009). The neuronal processes of this sensorimotor integration remain however poorly understood.
- The primary motor cortex (M1) is involved in somatosensory processing in primates. This has been suggested, among others, by sensory inputs from passive hand movements and from tactile stimulations recorded in M1 (Rosén and Asanuma, 1972; Wannier et al., 1991), by somatosensory impairments occurring after lesion of M1 hand representation (Nudo et al., 2000) and by an increase of activity in the primary somatosensory cortex (S1) forelimb area during reversible inactivation of the M1 forelimb area (Sasaki and Gamba, 1984).
- Somatosensory evoked potentials (SSEPs) are a powerful tool to evaluate the plastic changes occurring in the central nervous system (see e.g. Allison et al., 1991).

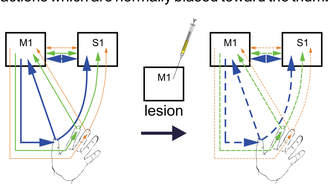
## Goals

1) To investigate how **tactile** information processing from the fingertips is affected by a unilateral lesion of the hand area in **M1** in non-human primates when stimuli are presented repeatedly.

2) To determine the potential validity of cortical activity evoked by tactile stimulation to the fingertips to diagnose e.g. motor cortex damages, instead of the conventional electrical stimulation (more painful) or MRI (more expensive).

## Discussion

- The M1 lesion had a drastic impact on tactile sensory processing from the fingertips. Interestingly, in comparison to other fingertips, sensory processing from the thumb tip was the most affected.
- We do not think that the lesion-induced distortions in volume conduction could explain the post-lesion observed changes in scalp signals as:
  1) both latency and amplitudes were substantially altered after the lesion.
  2) the middle fingertip signals remained largely unaffected by the lesion.
  3) the lesion was not in between the dipole and the head surface.
- The distinct post-lesion evolution of the finger use according to the task supports that the post-lesion tactile signals were not a substitute for the normal tactile signals.
- We hypothesise that the M1 lesion may have damaged the pre-existing strong sensorimotor interactions which are normally biased toward the thumb motor control:




(inspired from Asanuma, 1981)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## Material, Methods and Results

1 adult ♀ ***Macaca fascicularis*** under sevoflurane anaesthesia

+

32-electrode **EEG** recordings of SSEPs (Gindrat et al., 2014)

+

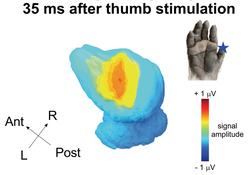
(Image from Heijo Electronics)

Individual **passive tactile stimulation** (supra-threshold 2-ms pulses, jittered around 1 Hz) randomly delivered to the fingertips of right thumb, index finger and middle finger (contralesional hand) with solenoid tappers (Heijo Research Electronics, Beckenham, UK)

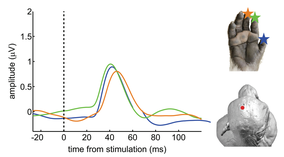
EEG **data analysis** by using EEGLAB and customised MATLAB® scripts (artifact rejection based on threshold (± 20 µV) and on joint probability distribution (3 SD), filters: 5-100 Hz, average reference)

### A) Tactile stimulation evoked potentials

**Voltage topography, 35 ms after thumb stimulation**

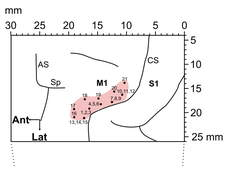



**Brain activation after tactile stimulation**

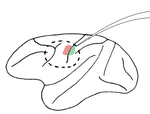



Tactile stimulation to the fingers elicited a focal contralateral brain activity with a high signal-to-noise ratio in anaesthetised macaque monkeys .

Permanent cortical **lesion** of the **finger representation** in left **M1** by microinfusions of ibotenic acid (20 µg/µl, 39.7 µl in total infused in 21 sites), without any post-lesion treatment to promote recovery.

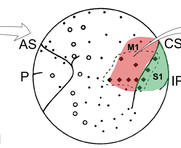


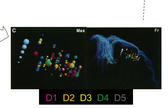

The precise targeting of the M1 finger representation was based on results from previous studies having mapped the motor cortex of macaque monkeys by using intracortical microstimulation (ICMS) (e.g. Sessle and Wiesendanger, 1982; Wyss et al., 2013):



Finger representation in M1 revealed by ICMS

(modified from Sessle and Wiesendanger, 1982)

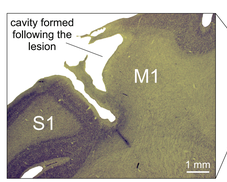



distributed overlapping mosaic representation of the fingers in M1

(Schieber and Hibbard, 1993)

### B) Lesion of the M1 finger representation




Nissl-stained section 39

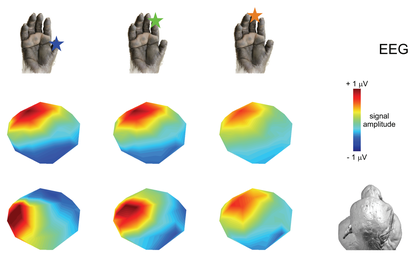
contour of lesion in grey matter
border grey matter/white matter

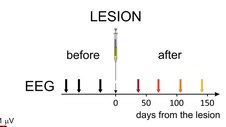
Projection of the grey matter lesioned area on lateral view of the brain, based on consecutive coronal Nissl-stained sections.

Histology confirmed that the lesion targeted the finger representation in M1 and spared S1.

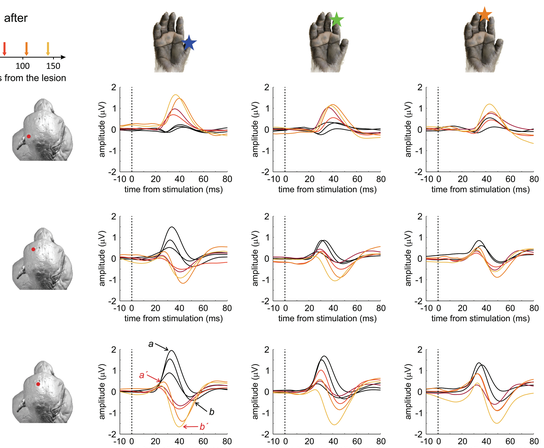
### C) Pre- and post-lesion brain activity

**1) Voltage topography, 33 ms after stimulation**




**2) Waveforms at 3 electrodes of interest**

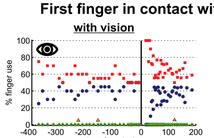


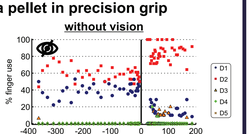

- The M1 lesion induced major changes in brain activity after tactile stimulation applied to the contralesional fingers, mainly to the thumb (**C 1, 2**).
- The lesion had a drastic impact on the amplitude and latency of both early and later cortical sensory potentials (**C 2**: compare *a* vs *a´* and *b* vs *b´*).
- The alteration in cortical sensory processing by the lesion was further enhanced over time (**C 2**). Although the functional consequence of these sensory changes for motor behaviour is not clear, post-lesion behavioural adaptation of the finger use occurred over time: the use of the thumb was partially recovered in the Brinkman box task with vision whereas it further deteriorated when the task was performed without vision (**D**).

### D) Fine manual dexterity (contralesional hand)

**Brinkman box task**

**First finger in contact with a pellet in precision grip**




## Highlights

1) The M1 lesion induced profound modifications in tactile sensory processing, especially from the thumb, which cannot be explained by volume conduction artifacts resulting from the lesion.

2) We propose that the development of behaviour after M1 injury involved the recruitment of new sensory and motor processes.

3) Brain activity evoked by finger tactile stimulation is relevant to detect the impact of an M1 injury.

4) M1 is important for tactile somatosensory processing from the fingers in primates.