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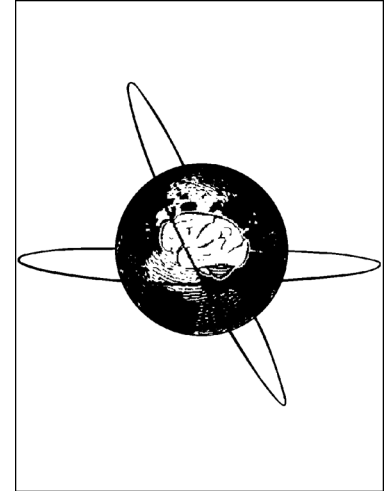
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Davide Giampiccolo, Luigi Cattaneo, Federica Basaldella, Giuseppe Kenneth Ricciardi, Francesco Sala

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Intraoperative Neuromonitoring predicts motor recovery in a long-term hemiplegic patient with a Rolandic metastasis

Davide **Giampiccolo**^a

Luigi **Cattaneo**^b,

Federica **Basaldella**^c

Giuseppe Kenneth **Ricciardi**^d

Francesco **Sala**^a

^aDepartment of Neuroscience, Biomedicine and Movement Sciences, Section of Neurosurgery, University of Verona, Verona, Italy

^bInstitute of Psychology, CIMEC - Center for Mind/Brain Sciences, Trento, Italy

^cDivision of Neurology, Verona City Hospital, Verona, Italy

^dDepartment of Neuroradiology, Verona City Hospital, Verona, Italy

Corresponding author:

Francesco Sala

Dept. of Neurosciences, Biomedicine and Movement Sciences, Section of Neurosurgery,
University of Verona, 37124 Verona, Italy

E-mail: francesco.sala@univr.it

In brain surgery of motor areas, a combination of motor evoked potential (MEP) monitoring and subcortical mapping is used to avoid neurological deficits. Continuous MEP stimulation from the Rolandic cortex monitors the integrity of corticospinal tract (CST), while mapping identifies it as the resection continues into white matter (Bello et al. 2014). Currently, there is little debate as to whether patients with preserved or mildly impaired motor function should undergo intraoperative neuromonitoring (IONM). However, indication for this in patients who are hemiplegic at admission is controversial, as no recovery would be expected. Here we describe a patient who, despite suffering from long-term hemiplegia, underwent IONM. Intraoperative MEPs were identified and predicted near total motor recovery.

A 57-year old right-handed woman with a history of high grade serous ovarian carcinoma (FIGO grade IVB) presented to the emergency room with sudden severe right hemiparesis (MRC scale 2), which rapidly deteriorated into hemiplegia (MRC scale 0) in two days. Right-sided hyperreflexia and positive Babinski's sign were noted. T1-weighted MRI with Gadolinium disclosed two left intra-Rolandic lesions (Fig. 1a). Preoperative tractography showed considerable CST displacement (Fig. 1b) from the central sulcus, although this was unreliable due to extensive perilesional edema. This latter and lesion volume contraindicated radiosurgery. Therefore, when considering volume, bifocality and proximity of the two lesions, the patient underwent asleep surgery with IONM ten days after admission. Once the dura was opened, train-of-five stimulation (monophasic, pulse width: 0.5 ms, interstimulus interval (ISI) 2 ms, 1 Hz repetition rate) with a monopolar probe evoked MEPs from the right abductor pollicis brevis (APB) and extensor digitorum communis (EXT) at 15 mA during cortical mapping (Fig. 1c), anterior to the CST shown with tractography (Fig. 1d). The central sulcus was opened to exclude CST activation due to current spread, the gyri were separated and MEPs were confirmed anterior to the central sulcus only. A strip electrode for continuous MEP monitoring was placed on the primary motor cortex (M1) hotspot, showing stable MEPs at 23

mA for ABP, EXT and biceps brachii, following stimulation of adjacent electrodes (Fig. 1e). Two small corticectomies were performed to remove the lesions and subcortical stimulation in the resection cavity demonstrated MEPs in the APB with an amplitude of 15 mA (Fig. 1f; 1g). MEPs remained unchanged throughout the procedure.

Early postoperatively, the patient remained hemiplegic (MRC scale 0), and the MRI showed complete excision of the lesions, which remained confined in the precentral gyrus (Fig. 1h). After one month the patient reported voluntary movement of the formerly paralyzed contralateral hand, and after two months, movement of the contralateral leg during sleep. At three months, the patient walked with spastic gait when supported, with mild paresis in right upper and lower extremities (MRC scale 4) and right foot dorsiflexion (MRC scale 3). Plantar response was flexor and tendon reflexes were symmetrical. After three months, tractography showed that the CST had been spared (Fig. 1i). After one year, complete motor recovery for both right upper and lower extremities was reported (MRC scale 5), except from a mild foot dorsiflexion deficit (MRC scale 4). Nerve conduction studies were normal in the lower limbs and navigated transcranial magnetic stimulation (TMS) demonstrated MEPs to stimulation posterior to the surgical resection for both hand and foot areas (Fig. 1j).

This is, to our knowledge, the first reported case of delayed post-operative recovery of voluntary movement after complete hemiplegia. Duffau et al. (2001) described a similar case of a 45-year old right-handed man with a left postcentral bleeding embryonal carcinoma, presenting with right acute hemiplegia. Upper limb movements were elicited throughout surgery, at 16 mA at the beginning, with a decrease in amplitude needed for movement (10 mA) at the end of the surgery. In contrast to our case, the patient recovered voluntary movement in the upper and lower limbs almost immediately (3-12 hours) after surgery (Duffau 2001). Accordingly, the pathophysiology of this preoperative motor deficit remains elusive. It is well known that

supplementary motor area (SMA) syndrome presents with motor akinesia in spite of preserved intraoperative MEPs. However, the pre- and post-operative MRI confirmed the lesion did not involve the SMA. We cannot exclude that preoperative edema extending to SMA may have been responsible for the perioperative motor deficit, although the patient's hemiplegia did not improve despite receiving a high dose of dexamethasone (12 mg/day i.v.) before and after surgery. The anterior portion of the precentral gyrus was damaged. Increasing evidence has shown that the anterior portion of the precentral gyrus is involved in motor modulation with the posterior portion in proximity of the central sulcus carrying corticomotoneuronal fibers involved in motor execution (Viganò et al. 2018; Rech et al. 2019), which could explain why MEPs were preserved despite no perioperative voluntary movement. However, irrespective of the pathophysiology, we assert that even in cases of severe motor deficit the CST may still be preserved. Thus, there is a rationale to use IONM also in these patients. Unfortunately, we did not have the chance of using preoperative navigated TMS (nTMS). However, we speculate that a combination of IONM with preoperative nTMS motor mapping may be an appropriate way to screen for hemiplegic patients that should undergo IONM, since it may be used to distinguish hemiplegia from hemiakinesia, i.e. disruption of access of voluntary motor command to M1 (Seidel et al. 2018).

To conclude, we show that it may still be possible to reverse preoperative hemiplegia/hemiakinesia if MEPs are elicited intraoperatively and preserved, even when the lesion is in the precentral gyrus. This suggests that clinical evaluation alone may be insufficient to distinguish hemiplegia from other disorders of motor control that may subtend absence of voluntary movement, both short- and long-term. Therefore, we propose that IONM should be considered in "hemiplegic" patients, as intraoperative discovery of MEPs and their preservation may enable patients to return to functional levels of motor ability.

Conflict of interest

None.

Data

Anonymized preoperative and postoperative MRIs and videos of the neurological examination that support the findings of this letter are available on the OSF framework (<https://osf.io/kfyx8/quickfiles>).

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Figure

Figure 1. a) Contrast-enhanced, T1-weighted MRI showing two left Rolandic lesions with conspicuous perilesional edema. b) preoperative HARDI tractography of the corticospinal tract (CST, in yellow). The two lesions (in red) were located between and anterior to the CST, that appeared to be displaced posteriorly. c) Cortical stimulation on showing MEPs at 15 mA for the ABP and EXT. d) Intraoperative site for cortical motor evoked potentials (MEPs) as disclosed by the neuronavigation (green dot). e) MEP monitoring throughout surgical resection showing preserved MEPs at 23 mA for the upper limb. Note that MEP for the lower limb were not monitored due to strip placement. f) Intraoperative picture showing sites for cortical (green dot) and subcortical stimulations (yellow asterisk). g) Subcortical stimulation showing preserved MEPs at 15 mA for the ABP. h) After three months MRI revealed complete resection of the lesions in the precentral gyrus. i) The postoperative HARDI-optimized tractography at three month showed cortico-subcortical integrity for the corticospinal tract (yellow). j) After one year navigated transcranial magnetic stimulation showed bilateral MEP persistence for upper (APB; FDI) and lower limb muscles (TA; AH). Grey dots: no response. Red dots: motor responses between 50 to 500 μ V. Yellow dots: motor responses between 500 to 1000 μ V. White dots: motor responses over 1000 μ V.

OOM = *orbicularis oris*; Bic= *Biceps brachii*; EXT= *extensor digitorum communis*; APB= *abductor pollicis brevis*; FDI= *first dorsal interosseous*; Qua= *Quadriceps femoris*; TA= *tibialis anterior*; AH= *abductor hallucis*

