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Intraoperative subdural low-noise EEG recording of the high frequency oscillation in the somatosensory evoked potential

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HIGHLIGHTS

- Low-noise EEG improves signal-to-noise ratio for evoked high-frequency oscillations (HFO) in subdural recording.
- Somatosensory evoked potential amplitude decreases with increasing stimulation rate under propofol.
- Somatosensory evoked potential amplitude were indistinguishable between propofol and sevoflurane anesthesia.

ABSTRACT

Objective: The detectability of high frequency oscillations (HFO, >200 Hz) in the intraoperative ECoG is restricted by their low signal-to-noise ratio (SNR). Using the somatosensory evoked HFO, we quantify how HFO detectability can benefit from a custom-made low-noise amplifier (LNA).
Methods: In 9 patients undergoing tumor surgery in the central region, subdural strip electrodes were placed for intraoperative neurophysiological monitoring. We recorded the somatosensory evoked potential (SEP) simultaneously by custom-made LNA and by a commercial device (CD). We varied the stimulation rate between 1.3 and 12.7 Hz to tune the SNR of the N20 component and the evoked HFO and quantified HFO detectability at the single trial level. In three patients we compared Propofol® and Sevoflurane® anesthesia.
Results: In the average, amplitude decreased in both in N20 and evoked HFO amplitude with increasing stimulation rate ($p < 0.05$). We detected a higher percentage of single trial evoked HFO with the LNA ($p < 0.001$) for recordings with low impedance (<5 kΩ). Average amplitudes were indistinguishable between anesthesia compounds.
Conclusion: Low-noise amplification improves the detection of the evoked HFO in recordings with subdural electrodes with low impedance.
Significance: Low-noise EEG might critically improve the detectability of interictal spontaneous HFO in subdural and possibly in scalp recordings.
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1. Introduction

Pathological high frequency oscillations (pHFOs, 80–500 Hz) recorded in the pre and intra-operative ECoG of epilepsy patients have been proposed as a biomarker for the identification of the epileptogenic zone (Jacobs et al., 2012). Particularly for epilepsy surgery, it has been demonstrated that residual fast ripples (FR, >250 Hz) are a reliable predictor of seizure outcome (Fedele et al., 2017; van 't Klooster et al., 2015; Wu et al., 2010). While faster oscillations have higher clinical relevance, they suffer from a poor signal-to-noise ratio (SNR). Therefore, an improvement in their detectability is highly desirable.

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An example of a physiological evoked HFO is represented by somatosensory evoked potentials (SEP) elicited by median nerve stimulation, which is standardly used in intraoperative neurophysiological monitoring. The low-frequency cortical response (N20), generated from area 3b in the primary somatosensory area (Ozaki and Hashimoto, 2011), concurs with a high frequency oscillation (HFO, >400 Hz) with an amplitude of few hundreds of nV peak-to-peak (pp), first recorded in the scalp EEG and MEG after massive averaging (Cracco and Cracco, 1976; Curio et al., 1994). The evoked HFO has been reported also in the subdural electrocorticogram (ECoG) in awake patients (Kojima et al., 2001; Maegaki et al., 2000) and anaesthetized patients (Burnos et al., 2016; Urasaki et al., 2006). The evoked HFO is a special case of neural population spiking which can be recorded at the macroscopic level. While the properties of evoked HFO could be studied in the averaged evoked response (Ozaki and Hashimoto, 2011), its poor signal-to-noise ratio (SNR) prevented access to the signal at the single trial level.

The spectral power of the EEG signal stems from biological sources and noise from the recording setup, the latter being negligible until around 100 Hz (Scheer et al., 2006). While for frequencies <100 Hz the EEG spectrum mainly reflects physiological activity, for frequencies >100 Hz the contribution of the recording system becomes increasingly dominating, critically affecting the SNR. The introduction of custom-made low-noise EEG amplifiers has opened the way to investigate the variability of the evoked HFO across single trials (Waterstraat et al., 2016, 2012). The combination of optimized hardware and tailored de-noising techniques maximally improve the SNR (Waterstraat et al., 2015a,b).

In this study, we identify the optimal conditions for the detection of the N20 and the evoked HFO in the ECoG. Modulating the amplitude of the evoked HFO through the stimulation rate (Klostermann et al., 1999; Urasaki et al., 2002) lets us vary the SNR in a physiological way. We compare the detectability of the evoked HFO between a commercial device (CD) and a custom-made low-noise amplifier (LNA). Using the well-defined biophysical framework of the evoked HFO, we can quantify the advantage of the LNA for HFO detection under critical SNR conditions.

2. Methods

2.1. Patients

We included nine patients (four men, median age 60y, range 35–75y, Table 1), who underwent tumor surgery at the Neurosurgery Department of the University Hospital in Zurich, and where subdural electrode strips were placed to locate the central sulcus by the phase reversal of the SEP. Collection of personal patient data and retrospective scientific workup was approved by the institutional ethics review board (Kantonale Ethikkommission KEK-ZH-Nr. 2012-0212) and collection of patients' written informed consent was waived.

2.2. Anesthesia management

Following our standard protocol for neurosurgical interventions, anesthesia was induced with intravenous application of Propofol (1.5–2 mg/kg) and Fentanyl (2–3 μg/kg). The intratracheal intubation was facilitated by Atracurium (0.5 mg/kg). Anesthesia was maintained with Propofol (5–10 mg/kg/h) and Remifentanil (0.1–2 μg/kg/min). Atracurium was omitted after intubation because of its interference with electrophysiological monitoring and mapping of motor function. In three patients the Propofol was ceased after one hour and anesthesia was sustained with sevoflurane (MAC < 0.5).

2.3. Stimulation

We stimulated the median nerve at the patient's wrist contralateral to the recording side with square-wave pulses (20 mA, 200 μs, OSIRIS NeuroStimulator, Inomed Medizintechnik GmbH, Germany, www.inomed.com). The stimulation rate ranged between 1.3 and 12. 7 Hz (Table 1).

2.4. Recording setup

Subdural strip electrodes (6 contacts, contact diameter 6 mm with a 5 mm exposure, spacing between contact centers 10 mm, Ad-Tech Medical) were placed after craniotomy in order to localize the central sulcus. Depending on surgical restrictions, the strip was placed directly on the sensory cortex, motor cortex, or both. After the localization, the strip was left in place for intraoperative monitoring of the SEP during tumor resection. The strip location was documented by the surgeon. A needle was placed in the dura (impedance ~1 kΩ) as electrical reference, in order to minimize the electrical interference from the environment. A subdermal needle served as patient ground.

All patients were recorded with a CD ($N = 44$ recordings, Table 1), featuring an input noise level of ~21 nV/$\sqrt{}$Hz, which was the ISIS® IONM (Intraoperative neural monitoring, 16 bit analog-to-digital conversion (ADC), Inomed Medizintechnik GmbH, Emmendingen, Germany; sampling rate 10 kHz, 30–2500 Hz bandpass, input range ±5 mV, LSB 0.153 μV).

In six patients ($N = 16$ recordings, Table 1) we added simultaneous recording with the LNA. We connected the active lead, reference lead and ground lead of the electrodes in parallel to the active lead, reference lead and ground lead of the CD and LNA. The custom-built LNA featured an input noise level of 2.3 nV/$\sqrt{}$Hz, (24 bit ADC, 10 kHz sampling rate, 0.1–3000 Hz bandpass, input range ±5 V combined with gain 100, LSB = 6 nV). The LNA is

Table 1
Patient characteristics. x: recording session with CD ($N = 35$ for propofol, $N = 9$ for sevoflurane); x*: recording session with CD and simultaneous LNA ($N = 16$).

Patient	Age, sex	Histology	SEP stimulation rate [Hz]									
			Under propofol					Under sevoflurane				
			1.3	3.7	5.7	8.7	12.7	1.3	3.7	5.7	8.7	12.7
1	67, f	Low Grade Glioma	x*		x*	x	x					
2	37, f	Meningeoma			x		x*					
3	75, f	Metastasis	x		x							
4	68, m	Glioma	x	x	x	x*	x*			x		x
5	75, m	High Grade Glioma	x*		x*	x*	x			x		x
6	35, f	High Grade Glioma	x*	x*	x*	x*	x*	x	x	x	x	x
7	59, m	Metastasis	x	x	x	x	x					
8	36, m	Low Grade Glioma	x	x	x	x	x					
9	60, f	Metastasis			x*	x*	x*					

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