

general anesthesia for the PSF surgery with an IV infusion of Propofol (40 µg/kg/min) and Sufentanil (0.3 µg/kg/h) and inhalational Sevoflurane to maintain half or less MAC of anesthesia. Neuromonitoring was done throughout the surgery measuring motor evoked potentials (MEP), somatosensory evoked potentials (SSEP) and EMG's.

Following rod placement and pulse lavage of the surgical site, a complete loss of the SSEP's from the right posterior tibial nerve was noted. The SSEP's from bilateral upper extremities, and the left lower extremity in addition to the MEP's from all extremities remained at baseline. We systematically excluded technical error, limb compression, perfusion, anesthetic and surgical causes. Mean arterial pressure (MAP) was raised to 90 mm Hg and surgery was stopped for about 45 minutes to resolve the situation but without success, at which point a senior anesthesiologist was consulted. Taking into consideration the sequence of events and proximity in time from the pulse lavage to complete loss of signal (7 min) we presumed the probable cause of the SSEP loss would be local irritation leading to vasospasm from the pulse lavage. A warm saline irrigation was started and the MAP's were further raised to around 105 mm Hg. Seven minutes after warm saline irrigation and increasing the MAP, signs of reproducible SSEP recovery was noted and it completely recovered to baseline within 30 minutes.

[SNACC-3] Transition to Loss of Consciousness during Anesthesia Induction With Remifentanil and Propofol: EEG Patterns

Leitao Ferreira A*, Nunes C†, Mendes J*, Amorim P‡. *Universidade Do Porto; †Universidade Aberta; ‡Centro Hospitalar Do Porto, Porto, Portugal. The importance of more personalized care is becoming increasingly recognized. Identifying the precise moment of loss of consciousness (LOC) during the induction phase of general anesthesia is important as it will determine the drugs amount required for each individual to maintain adequate levels of anesthesia. The dynamics of EEG signals of volunteers subjected to anesthesia with propofol showed that, at LOC, both alpha (8 to 12 Hz) and low-frequency (0.1 to 1 Hz) power increases significantly.¹ But, no surgical stimulus or drug interactions were present. The aim of our

study is to examine EEG patterns that are associated with LOC in patients submitted to general anesthesia with remifentanil and propofol.

Data of 10 patients were analyzed. Standard anesthesia induction protocol was with a remifentanil effect-site concentration (Ce) target of 2 to 2.5 ng/mL, followed by a propofol infusion of 200 mL/h until LOC. At LOC the propofol's Ce target was set to the value achieved at LOC, until intubation. Data were recorded from the beginning of the remifentanil administration until 10 minutes after LOC. LOC was identified as the lack of eye opening to name calling and a tap on the forehead.

Multitaper spectrograms 2 were computed to observe the dynamics of EEG oscillations in window lengths of 6 seconds with overlap of 0.1 second and a time-bandwidth of 3 to 5 tapers. To quantify the variation of each decomposed wave from EEG spectrogram, the difference between the area under the curve from 1 minute before LOC to LOC and the area under the curve from LOC to 1 minute after LOC, were calculated.

Table 1 presented the results regarding: demographics, remifentanil, and propofol concentrations at LOC, the time to LOC and the magnitude of the waves changes in the transition from conscious to unconscious. Figure 1 is a representative example of one set of data.

At LOC, all EEG spectrograms showed a decrease in gamma-frequency (25 to 40 Hz) power and an increase in alpha (8 to 12 Hz) and delta-frequency (0.1 to 4 Hz) powers.

Our results have not been previously showed in patients undergoing surgery with remifentanil and propofol. Using the spectrograms to quantify the changes at LOC and combining that with behavioral and other physiological variables would be able to create a robust and principled estimate model for tracking the dynamic changes in patients' wakefulness as they enter into the unconsciousness state during induction with general anesthesia.

References:

1. Purdon PL, Pierce ET, Mukamel EA, et al. Electroencephalogram signatures of loss and recovery of consciousness from propofol. *Proc Natl Acad Sci U S A*. 2013;110:E1142–E1151.
2. Babadi B, Brown EN. A review of multitaper spectral analysis. *IEEE Trans Biomed Eng*. 2014;61:1555–1564.

Acknowledgments: FCT:SFRH/BD/98915/2013;UID/SEM/50022/2013.

Table 1: Patient demographics and drug concentrations at LOC, time to LOC, gamma, delta and alpha frequency changes at LOC, for all patients.

# Patient	1	2	3	4	5	6	7	8	9	10
Gender (M/F)	M	F	M	F	M	M	F	F	M	F
Age (yrs.)	76	58	60	58	59	45	38	72	36	69
Weight (Kg)	80	80	53	59	50	67	60	74	120	62
Height (cm)	166	180	178	157	175	180	166	164	200	154
Ce Remifentanil at LOC (ng/mL)	2.00	2.00	2.50	2.50	2.49	2.00	2.39	2.49	2.10	2.36
Ce Propofol at LOC (µg/mL)	4.11	5.11	3.82	3.64	6.07	6.17	5.26	3.94	5.41	2.91
Time from starting Propofol to LOC (min)	6.92	3.52	2.26	2.28	3.65	4.15	3.55	2.45	4.12	1.88
Gamma waves decreasing (%)*	86.86	79.22	96.25	20.64	93.25	52.73	56.81	65.04	11.97	34.58
Delta waves increasing (%)*	41.61	24.42	17.38	50.03	34.36	14.96	45.39	4.97	7.46	15.57
Alpha waves increasing (%)*	51.63	18.56	40.53	62.31	25.46	36.93	64.36	20.25	63.79	53.48

*Waves behavior in the transition between conscious and unconscious state

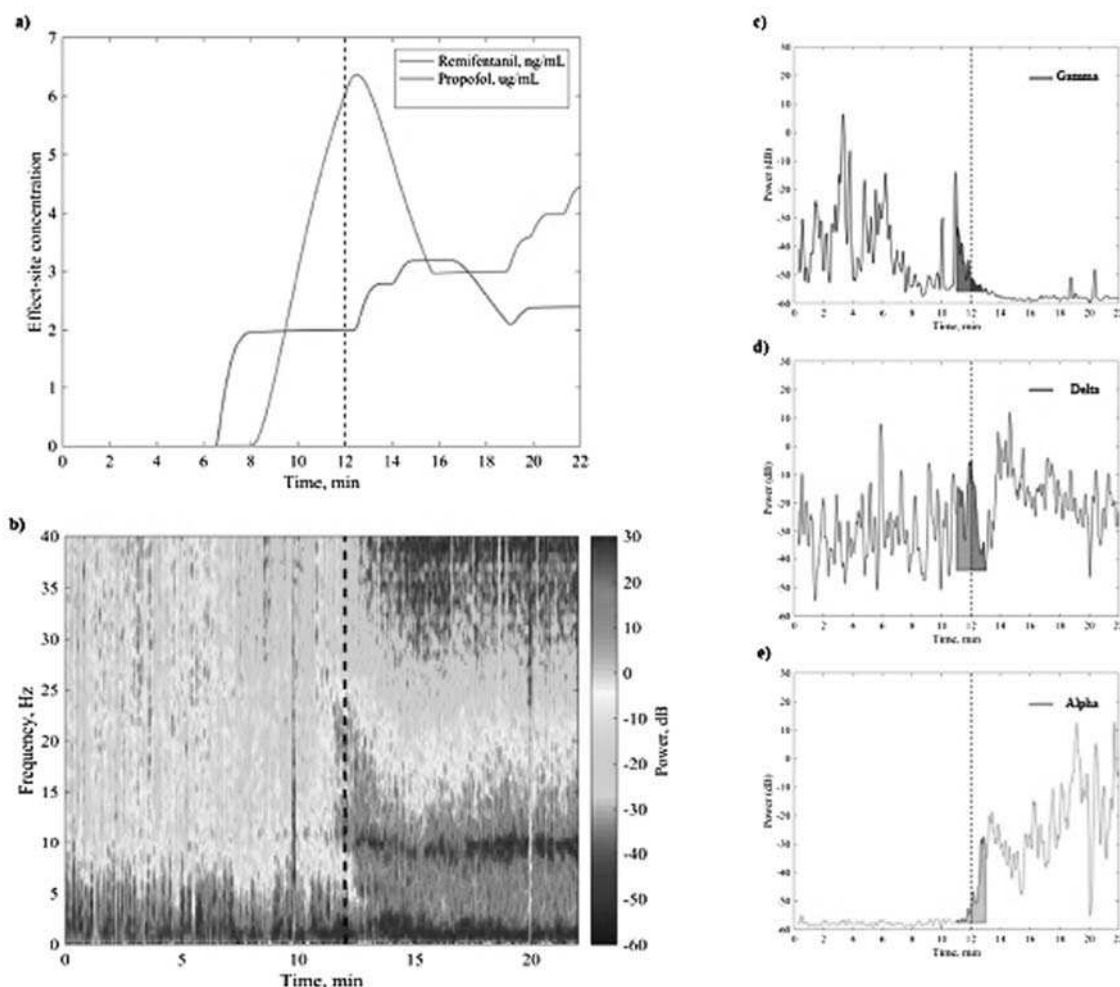


Figure 1: Dynamics from a representative patient during induction of general anesthesia with remifentanyl (Ce target of 2ng/mL) and propofol: a) Effect-site drugs concentration; b) EEG spectrogram; c) Power in gamma frequency (25–40Hz); d) Power in delta frequency (0.1–4Hz); e) Power in alpha frequency (8–12Hz). All figures are aligned with respect to LOC (dashed vertical line).

[SNACC-4] Loss of Motor Evoked Potentials Before Posterior Cervical Fusion and Return With Subtle Head Manipulation

Kramer D, Ferastraoaru V, Zhang L, Legatt A. *Montefiore Medical Center, Bronx, NY.*

A 77-year-old woman with cervical spondylosis and previous ACDF at C3-C5 presents with severe right arm and hand weakness and bilateral leg weakness.

GA was induced and intubation was easily achieved with #3 Glidecope, keeping the head in neutral position. The patient was maintained initially on desflurane and converted to TIVA. Mean arterial pressure was maintained at 80 mm Hg. SEPs, motor evoked potential (MEPs), TOF, and EMG were utilized.

SEPs were recorded following stimulation of either posterior tibial nerve and of the right ulnar nerve; and remained stabler during the operation. SEPs to left ulnar nerve stimulation were intermittently identifiable and thus were not adequate for intraoperative monitoring.

MEPs were initially present in the left deltoid, biceps and EDC muscles and small MEPs were present in the right deltoid and EDC muscles. There were no monitorable MEPs in the right biceps muscle or in either APB muscle. Twenty minutes after induction, with the patient supine, end tidal desflurane at 0%, mean arterial pressure was 80 mm Hg, and TOF 4/4 twitches, the MEPs that had been present in arm muscles disappeared bilaterally and were

reproducibly absent (Fig. 1). There was no identifiable technical cause of the change; no anesthetic boluses, inhalational agents, or additional paralytic drugs had been given.

The anesthesiologist gently lifted the patient’s occiput resulting in 1/2 inch of elevation. Immediate return of the MEPs (Fig. 1) with the recovery to amplitudes that was comparable to baseline.

A wake-up test was performed. TIVA was reinduced. MEPs were still present on the left and small MEPs were present in the right deltoid and EDC muscles, and did not change with prone positioning. The patient underwent decompression at C3-C6 and fusion at C2-T2. MEPs that were monitorable at baseline levels with no significant changes. At the end of procedure, the patient was extubated. Her arm and hand strength was markedly improved from her preoperative examination.

Discussion: In a patient with severe cord compression, small alterations in the positioning of the head and neck can cause compromise. MEP loss in our patient’s arms was most likely due to small alterations in her neck position. This is confirmed by reappearance of the MEPs following small readjustment of the head.

If MEPs are suddenly lost in a patient with spinal compression, gentle adjustment of the patient’s head may be performed to relieve increased cord compression.³ MEP monitoring should be initiated early and recorded frequently. This case illustrates that cooperative interaction