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TITLE: PROPOFOL DOES NOT CAUSE EPILEPTIFORM ELECTROENCEPHALOGRAPHIC ACTIVITY

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Propofol has been reported to both produce (1) and suppress (2) epileptic activity. This institutionally approved study was designed to document, through continuous 16-channel electroencephalographic (EEG) monitoring, the incidence of epileptiform activity and to compare EEG patterns produced by propofol with those produced by isoflurane.

Informed consent was obtained from 60 patients scheduled for lumbar surgery. Gold cup EEG electrodes were applied to the patients' scalps with collodion. Sixteen channel parasagittal referential EEG was recorded on paper and video tape from before induction of anesthesia until the patient was awake at the end of surgery. All patients received bolus sufentanil, up to 0.5 mcg/kg, prior to intubation; vecuronium, 0.1 mg/kg, to facilitate intubation; and a low-dose sufentanil infusion (S) for maintenance with no additional muscle relaxant. Patients were randomly assigned to 1 of 3 anesthetic regimens: induction with propofol and maintanence with nitrous oxide, oxygen, and a variable rate infusion of propofol ($P/N_2O/S$; n = 18); induction with propofol and maintenance with a variable rate propofol infusion (P/S; n = 21); or induction with thiopental and maintenance with isoflurane, N_2O, oxygen ($I/N_2O/S$; n = 21). Investigators blinded to the anesthetic techniques divided EEG records into 10-s epochs and counted epileptiform activity.

The groups did not differ by severity of operation, duration of anesthesia, age, weight, sex, or ASA class. Patients receiving propofol demonstrated a high-amplitude delta rhythm with superimposed waxing and waning 8- to 11-Hz activity seen primarily in the frontal and central, but also in the parietal, distributions. The delta frequency tended to be slower and the alpha frequency faster with propofol than when isoflurane. Addition of N_2O to propofol decreased delta activity and made alpha frequency more continuous. Contrary to other reports (3), very few periods of burst suppression were noted in adequately anesthetized, unparalyzed patients receiving propofol. Numerous transient waveforms produced by fusion of alpha and delta or theta frequency patterns had some spike and slow-wave characteristics but did not represent true epileptic activity. With $P/N_2O/S$, 21,909 10-s epochs were analyzed; with P/S, 34,288; and with $I/N_2O/S$, 34,217. No epileptic EEG activity was seen in any patient.

Propofol does not cause epileptic activity in patients without history of seizure disorder. Burst suppression EEG pattern, as with other anesthetics, probably indicates excessive depth of anesthesia.

References

1. Lancet 2:1518, 1987.
2. ANESTHESIOLOGY 70:412-417, 1989.
3. ANESTHESIOLOGY 73:A202, 1990.

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Title: EEG CHARACTERISTICS OF EMERGENCE FROM PROPOFOL/SUFENTANIL TOTAL INTRAVENOUS ANESTHESIA

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Prediction of awareness or imminent emergence from anesthesia using univariate EEG descriptors has not been very successful to date. This study investigated the EEG changes in the traditional power spectral bands and median frequency (MF) during emergence from propofol/sufentanil total intravenous anesthesia (TIVA) in patients undergoing various surgical procedures of moderate duration.

Methods. Ten patients, ASA physical status 1, aged 19-47 yr, undergoing general surgical procedures of 60-120 min duration gave informed consent to participate in the study, which was approved by the local Human Subjects Committee. Anesthesia was induced with propofol 2 mg/kg and maintained with a continuous infusion of propofol 6 mg/kg/hr (10 mg/kg/hr for the initial 10 min, 8 mg/kg/hr for the next 10 min). Analgesia was achieved with a loading dose of sufentanil 1 μg/kg, and additional sufentanil bolus (10 μg) as indicated by clinical signs of inadequate analgesia. ECG, SpO_2, heart rate and blood pressure were recorded. Two channels of EEG (F_3-A_1,F_4-A_2) were recorded on a digital taperecorder and analyzed off-line using a Nicolet Pathfinder II with fast Fourier frequency analysis software. Power in the delta (1-3 Hz), theta (4-7 Hz), alpha (8-12 Hz) and beta (13-30 Hz) bands and MF were analyzed. After completion of the surgical procedure, the propofol infusion was stopped and the patient allowed to emerge from anesthesia. Care was taken not to stimulate the patient in any way (suction, moving, loud noise). At one min intervals the patient was asked to open the eyes. EEG data derived from four one minute intervals (15 four sec epochs, excluding artifacts) were compared: **A** - 1 min before stopping the propofol infusion, **B** - 5 min after stopping the propofol infusion, **C** - 3 min before eye opening, **D** - one min before eye opening. EEG power was log-transformed before statistical analysis. Data were analyzed by repeated measures ANOVA and are presented as mean ± SD or 95% confidence limits (absolute power). Differences between means were compared using t-tests with corrections for multiple comparisons.

Results. The EEG during propofol/sufentanil TIVA was characterized by a bimodal distribution of power with predominance of activity in the delta ((41.9 ± 16.2% of total power) and alpha (35.6 ± 13.8%) bands. Average time between stopping the propofol infusion and eye opening was 11.9 ± 6.4 min. The most consistent EEG changes during emergence were pronounced decreases in power in the delta and alpha band ($P<0.01$). The difference between power in the alpha band at 3 and 1 min before eye opening was significant ($P<0.05$). Beta power increased 5 min after discontinuation of the propofol infusion, but thereafter decreased. The change in median frequency (+1.8 Hz) was not statistically significant.

Table: EEG power (μV^2) and median frequency (Hz).

	propofol stop		eyes open	
	0 min	+5 min	-3 min	-1 min
Delta Pwr	85*(38-190)*	49*(25-95)**	35*(17-74)**	25*(12-55)**
Theta Pwr	33*(18-63)*	28*(15-54)*	18*(9-34)*	14*(8-27)*
Alpha Pwr	71*(37-136)*	65*(34-126)**	28*(11-72)**	18*(8-40)**
Beta Pwr	12*(4-32)*	17*(5-52)**	9*(3-31)*	13*(4-42)*
MF (Hz)	5.2*(3.8-7.1)*	6.8*(5.5-8.6)*	6.0*(4.8-7.6)*	7.0*(5.6-8.7)*

Data are mean (95% confidence limits); *P < 0.05 versus propofol stop.

Discussion. The results indicate that emergence from TIVA with the propofol/sufentanil regimen employed in this study is accompanied by pronounced decreases of power in the delta and alpha bands to less than 30% of the values during the propofol infusion, and a transient increase in beta power. These changes in alpha and delta power are of sufficient magnitude that they can be visually detected on graphic trends of power versus time, and, accordingly, they may be valuable for detecting insufficient plasma levels of propofol and impending emergence.