The Effect of Dexmedetomidine on Intracranial Electroencephalographic Activity

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Introduction

Dexmedetomidine is a sedative that acts as an agonist at a2-adrenergic receptors. Several authors have reported that sedation with dexmedetomidine can cause a modest increase in power in the delta (0-4 Hz), theta (4-8 Hz), and alpha (8-16 Hz) frequency ranges. These studies examined the effects of dexmedetomidine using electroencephalography (EEG) in adults and children. In order to further examine the effects of dexmedetomidine, we conducted spectral analysis on the effect of dexmedetomidine on intracranial electroencephalographic (iEEG) activity.

Methods

We acquired continuous iEEG recordings for five patients (age >18 years) with medically refractory epilepsy undergoing intracranial electrode explantation. Data were continuously acquired at 5 kHz (0.05-1 kHz band pass filter). Following five minutes of baseline recording, intra-venous infusion of dexmedetomidine was initiated until the calculated loading dose was achieved. Power spectra were computed using the Fast Fourier Transform on 4s non-overlapping windows. Baseline power spectra were compared to spectra obtained during dexmedetomidine over the final 1/3 of dose loading time, with an interval size equal to the baseline interval. The Mann-Whitney U statistical test and false discovery rate correction were used to establish significance.

Results

Power spectra were computed from 209 electrodes across five patients. Comparing the group dexmedetomidine interval to the group baseline interval, qualitatively increases in power from 0-10 Hz could be appreciated. Specifically, dexmedetomidine exhibited increase in power in the delta ($p=0.003$), theta ($p=0.02$), and alpha ($p<0.001$) frequency ranges when compared to baseline iEEG recording (Figure 1).

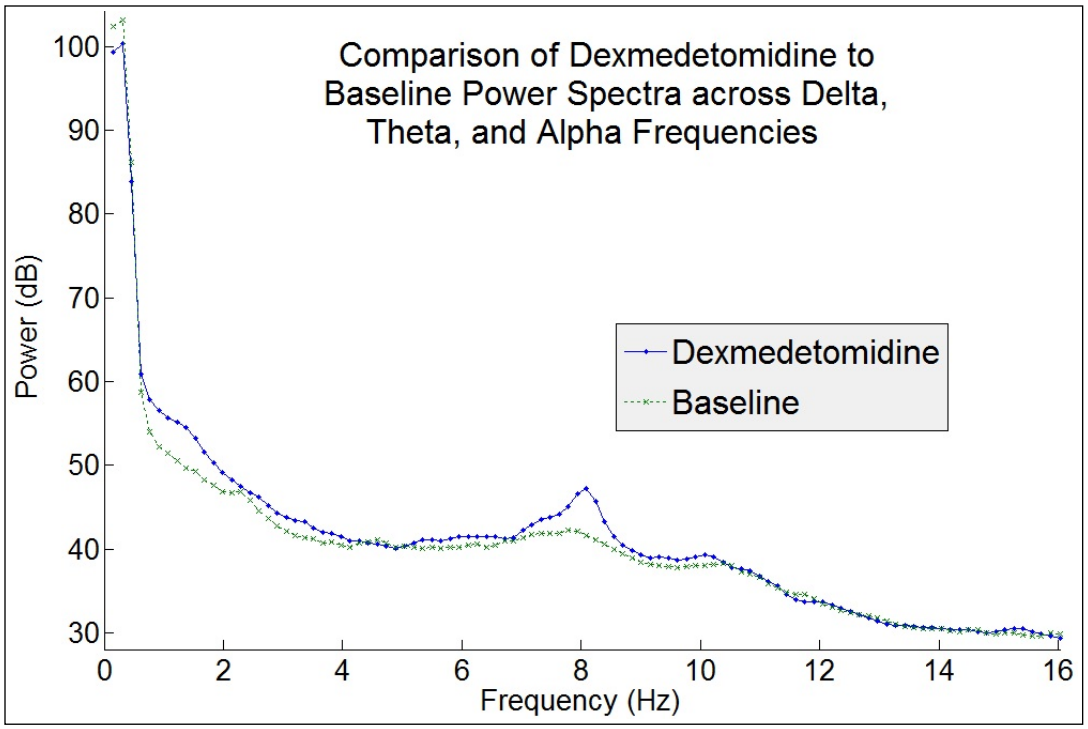

Figure 1: A comparison of power spectra between the baseline time interval (iEEG recording before the administration of dexmedetomidine) and the dexmedetomidine interval (final third of dose loading time). The delta (1-4 Hz), theta (4-8 Hz), and alpha (8-16 Hz) frequency ranges were examined.

Conclusions

From iEEG recordings dexmedetomidine increases power in the delta, theta, and alpha frequency ranges, which may underlie its clinical effects. Given its distinct clinical effects as compared to propofol we propose future examinations to compare spectral changes associated with dexmedetomidine to propofol anesthesia. Furthermore we plan to compare the spectral changes these pharmacological agents have on epileptogenic vs. non-epileptogenic cortex.