Q-EEG and Behavioral Effects of Dilantin Termination in a case of a Post-Bilateral Aneurysm Patient

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In this case study Quanlitative Electroencephalographic (Q-EEG) assessments were made while an audio-visual stimulation (AVS) procedure was used as a treatment for a four year post head injured adult female suffering from herniplegia, aphonia, aphasia, dysphagia, incontinence, compulsive and Inflexible behavior. The AVS procedure consisted of visual and audio stimulation which changed in response to the dominant EEG frequency. The AVS procedure can be described as a "true" biofeedback intervention, as it uses the patient's own brain activity to customize stimulation, which is fed back to the brain through audio-visual stimulation. See Ochs (1994) and Russell, Carter, Bell, & Bush (1995) for procedural details. During AVS feedback, a quantitative analysis of the major EEG bandwidths are determined. It is this Q-EEG across sessions which is the focus of this study. It was noted that major changes in the EEG were detected when Dilantin was withdrawn during the treatment.

Method

Subject

The patient is a 44-year-old Caucasian female who initially suffered from a right frontal intercerebral hematoma, due to thrombosis. The patient was given an angiogram which did not provide evidence for arteriovenous malformation or aneurysm. The hematoma was evacuated and the patient was recovering well for about two days. At this point, she became comatose, and a second CT scan revealed a left deep thalamic hematoma which was not present upon admission to the hospital. The patient had suffered from these problems approximately 3 1/2 years before beginning AVS treatment. Physical therapy and speech therapy had been discontinued because of lack of progress in rehabilitation.

At the beginning of the AVS treatment, the patient was suffering from right sided herniplegia, aphonia, aphasia, dysphagia, incontinence, compulsive and inflexible behavior. The patient was unable to eat or swallow, and was fed through a tube. The patient's medications at the beginning of treatment included Valium 10 mg T.I.D., Jevity Liquid T.I.D., Potassium Chloride 15 cc BID, Dilantin 100 mg BID, Ascendin 25 mg, Pericolace 15 cc, and Tylenol #3 PRN.

Instrumentation

The Q-EEG data was provided by a J & J 1-330 system with a E-201 EEG module, and Biological Operating System software. Electrode placement was a standard 10-20 CZ placement and the right earlobe, with the left earlobe as reference. All sessions lasted 14 minutes.

Procedure

The subject was treated for 68 sessions by one therapists using the AVS procedure. The session occurred twice weekly. The system provided mean magnitude (microvolts squared) and percent time for the bandwidths of Theta (4-7 Hz), SMR (12-15 Hz), Alpha (7-12 Hz), low Beta (15-19 Hz), and high Beta (19-30 Hz). At session 35, Dilantin, which was provided prophylactically since the injury, was withdrawn. Dilantin is an antiepileptic medication (phenytoin) related to the barbiturates in chemical structure (5.5-diphenyl-2.4 imidazildinedione) and is used in the treatment of epilepsy. The primary site of action appears to be the motor cortex where spread of seizure activity is inhibited. Possibly by promoting sodium efflux from neurons phenytoin tends to stabilize the threshold against hyperexcitability caused by excessive stimulation or environmental changes capable of reducing membrane sodium gradient. Q-EEG changes were noted following the withdrawal of this medication.

Results

The effects of Dilantin withdrawal were abrupt and dramatic. In the figure 1 below, the 68 sessions of Q-EEG are plotted for the one year with a vertical mark indicating the termination of DUantin. In some sessions, artifactual data is displayed as missing data. Figure I reveals that percent power of high beta increased dramatically, theta activity was reduced while SMR, alpha, and low beta were essentially unchanged.

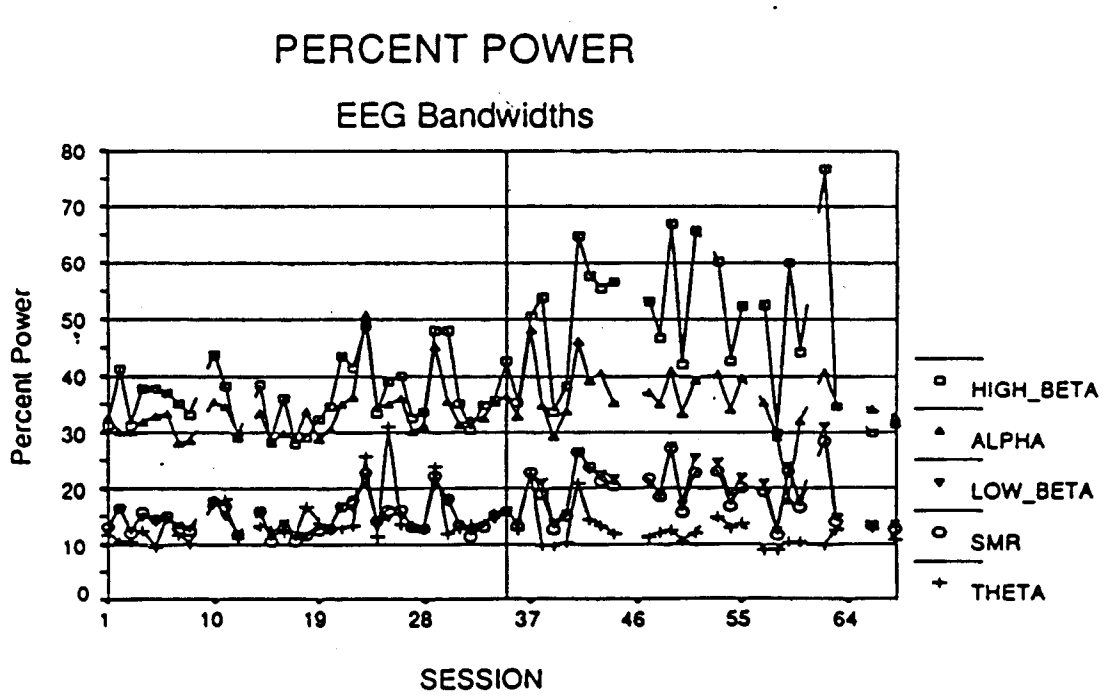

Figure 1. Percent Power of All Bandwidths

Figure 2 depicts the mean amplitude of the bandwidths pre and post Dilantin withdrawal. The major change was in the increase in amplitude of High Beta. Following Lubar's model of increased attentiveness (Lubar, 199 1), such a change in theta/beta ratio would lead to an interpretation of increased cortical arousal upon withdrawal of the medication. In this case there was also behavioral evidence of the change in that the patient became more assertive, activity level increased along with displays of communication of affect, and attention (read a book). Increases in fine motor control (stirring coffee, improved signature) and swallowing of pureed food and liquids were also noted. A possible confound in the EEG changes was the ongoing biofeedback treatment using the audio-visual entrainment at the time the Dilantin was used, when it was withdrawn, and during the subsequent period.

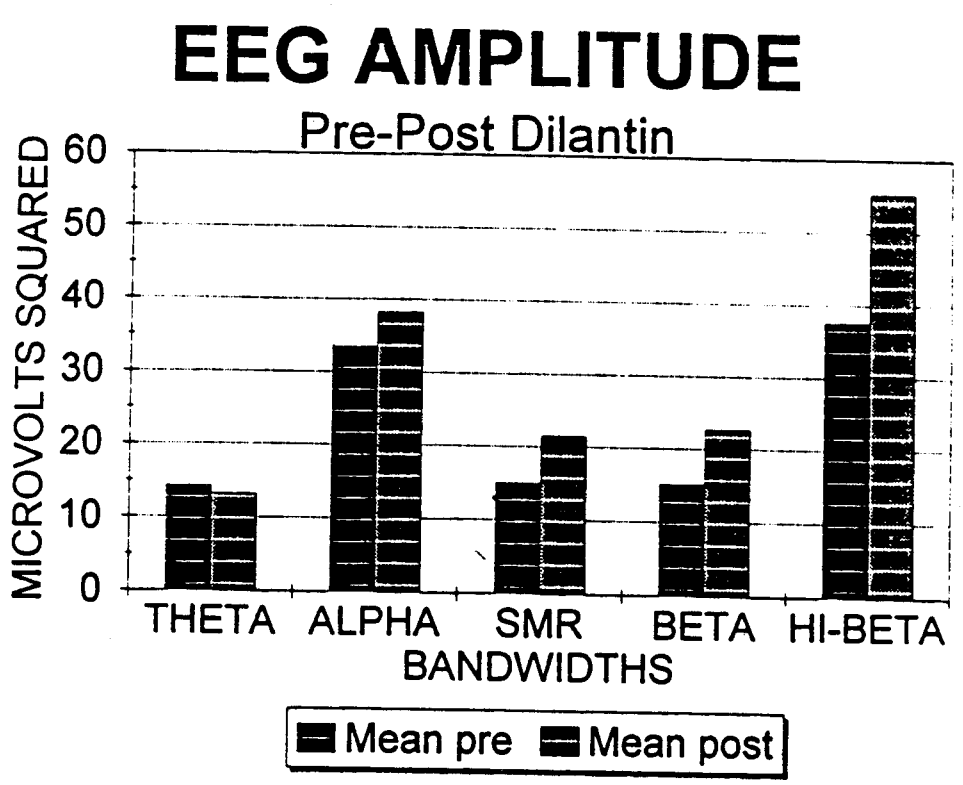

Figure 2. Pre-Post amplitudes for All Bandwidths

Summary

In summary, the Q-EEG data provide a physiological basis for the reported effect of Dilantin working as a central nervous system depressant. The data, in this case study, demonstrates dramatic changes in brain wave activity when Dilantin is withdrawn. The changes in brain waves patterns are consistent with those EEG patterns associated with shifts from low cortical arousal to high cortical arousal.

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1. Dilantin
2. Q-EEG
3. AVS