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Electroencephalographic abnormalities in borderline personality disorder

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Abstract

Epilepsy and non-localized brain dysfunction have been invoked, among others, as underlying factors in borderline personality disorder. We have recorded 58 electroencephalograms in 20 borderline patients, first after complete drug washout and then under carbamazepine or placebo double-blind treatment. Taking into account only definite abnormal tracings, we found a 40% incidence of abnormal diffuse slow activity. No patient disclosed focal or epileptiform EEG features. Carbamazepine did not appear to modify the electroencephalogram. © 1998 Elsevier Science Ireland Ltd.

Keywords: Borderline personality disorder; Electroencephalography; Slow activity; Carbamazepine

1. Introduction.

Borderline personality disorder (BPD) is a well-recognized syndrome (American Psychiatric

Association, 1987). Its main clinical features include brief episodes of affective manifestations, brief psychotic episodes, emotional instability, impulsive and unpredictable behavior, frequent self-mutilations and altered interpersonal relations.

BPD has been associated with several psychiatric and organic pathological conditions. Schizophrenia (Gunderson et al., 1975; Gunder-

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son, 1979; Schulz et al., 1989), schizoaffective psychoses (Andrulonis et al., 1982), atypical psychoses (Mitsuda and Fukuda, 1974; Andrulonis et al., 1982) and affective disorders (Akiskal, 1981; Carroll et al., 1981; Pope et al., 1983; Akiskal et al., 1985; Schulz et al., 1989) have been connected with BPD.

Although BPD is the most frequently diagnosed personality disorder (Gunderson and Zanarini, 1987), the physiopathological processes involved in this syndrome remain unclear. Noradrenergic and opiate systems (Van der Kolk, 1987), serotonin (Brown et al., 1982; Coccaro et al., 1989; Hollander et al., 1994) and dopamine (Bonnet and Redford, 1982; Lucas et al., 1987) systems dysfunction have been proposed as components of BPD. Epilepsy-related phenomena have also been invoked since some of the clinical features of BPD resemble the psychological characteristics sometimes observed in epileptic patients with complex partial seizures (CPS) (Cowdry et al., 1980; Fenwick, 1981; Cowdry and Pickar, 1985; Cornelius et al., 1986). Case reports have described CPS in patients previously misdiagnosed as BPD (Snyder and Pitts, 1984; Cowdry and Pickar, 1985; Messner, 1986; Schmid et al., 1989).

Searching for neurophysiologic abnormalities in BPD, Snyder and Pitts (1984), Cowdry and Pickar (1985) and Tanahashi (1988) have found significant abnormalities in the electroencephalographic scalp records of BPD patients. Other studies did not disclose significant abnormalities (Cornelius et al., 1986; Archer et al., 1988; Ogiso et al., 1993). Moreover, positron emission tomography studies have indicated that there is no metabolic evidence for the existence of temporal lobe epilepsy in BPD (De la Fuente et al., 1992, 1994). It has been proposed that the EEG abnormalities in BPD illustrate not the presence of abnormal cortical foci but rather a non-EEG-localizable brain dysfunction (Van Reekum, 1993), as noted by the increased incidence of slow-wave activity and other non-specific EEG changes.

From a pharmacological point of view, one study (Cowdry and Gardner, 1988) suggested that carbamazepine (CBZ), an effective treatment for CPS (Dreifuss, 1983), might improve BPD.

The purpose of the present study was to explore the scalp EEG recordings of 20 unmedicated BPD patients and to examine the evolution of the recordings under CBZ treatment.

2. Methods

2.1. Patients and treatment

Twenty consecutive in-patients (14 women and six men: mean age = 32.4; range, 21–45) fulfilling the DSM-III-R (American Psychiatric Association, 1987) criteria for BPD (mean = 6.8 criteria) and with a score of at least seven (mean = 8.75) on the Gunderson and Kolb diagnostic interview for borderlines (Gunderson et al., 1981) were recruited as part of a more extensive study that also tested the efficacy of carbamazepine on the clinical symptoms of BPD. Other criteria for inclusion were: normal physical and neurological examinations, normal standard biological blood test values and normal electrocardiograms. Present DSM-III-R axis I disturbances, history of epilepsy, encephalitis or head trauma and inability to stop use of alcohol or psychoactive substances during the study were considered exclusion criteria.

The 20 patients underwent a washout period of at least 10 days (15 days for tricyclic antidepressants and monoamine oxidase inhibitor agents). No patient had taken neuroleptics for 2 months prior to the study. The patients were randomized to receive either CBZ ($n = 10$) or placebo (PLC) ($n = 10$) in a double-blind treatment strategy. The washout period was followed by 32 days of CBZ or PLC. We administered the treatment orally. Morning trough plasma levels of CBZ and 10,11-epoxy-CBZ were determined on days 8, 16 and 32 of CBZ therapy. In order to maintain these levels within the therapeutic range required for the management of epileptic or affectively ill patients, a clinician not blind to the drug regimen adjusted the doses of CBZ.

Patient compliance regarding the use of any recreational drug, medication or alcohol during the washout and treatment periods was strictly verified by repeated plasma screenings.

The clinical outcome of the two groups was

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