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EEG and Evoked Potentials in Episodic-Dyscontrol Syndrome

Key Words
EEG
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Episodic dyscontrol
Explosive disorder

Abstract
The neurophysiologic correlates of explosive rage and violence are uncertain and controversial. We recorded 17-channel electroencephalograms (EEGs), brainstem auditory-evoked potentials (BAEPs), and long-latency auditory-event-related potentials (AEPs) in 23 patients with impulsive, aggressive and violent behavior satisfying criteria for episodic-dyscontrol syndrome. Most patients also satisfied criteria for intermittent explosive disorder, although some had had conduct disorders in childhood or had previously used psychoactive substances. Sixteen of 23 patients had normal EEGs, while 7 had diffuse or focal slowing not ascribable to drowsiness or the effects of medication. They differed significantly from 20 age-matched patients with headaches, of whom 1 had an abnormal EEG ($\chi^2 = 4.68$, $p < 0.05$), and from 24 depressed patients, all of whose EEGs were normal ($\chi^2 = 4.83$, $p < 0.05$). Patients and normal control subjects did not differ in BAEP latencies. N100 and P160 AEP amplitudes were lower in episodic-dyscontrol patients than in control, but the difference was not significant. These findings suggest that nonspecific cerebral dysfunction and EEG changes may be associated with disordered impulse or behavior control. Episodic dyscontrol may be associated with other evidence of minimal brain dysfunction.

Introduction

The relationship of violent or aggressive behavior to epilepsy, and the incidence and significance of EEG abnormality in such patients have been controversial. The episodic-dyscontrol syndrome [1, 2], in which patients manifest violent or enraged behavior with little or no provocation and sometimes in relation to the ingestion of small amounts of alcohol [3], has been linked with limbic-system dysfunction of which temporal-lobe epilepsy may be one manifestation [4]. The disorder is also linked with past and family psychiatric history, perinatal difficulties, and minimal brain dysfunction [5, 6]. Various estimates of the frequency and specificity of EEG abnormality in violent and aggressive patients have been reported [7]. A smaller number of reports have focused on auditory-evoked potentials in rage or violent attacks [8]. We recorded EEGs, brainstem auditory-evoked potentials (BAEPs), and auditory-event-related potentials (AEPs) in patients with episodic-dyscontrol syndrome and compared them with age-matched patients being evaluated for headaches and depression.

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Subjects and Methods

Twenty-three patients were referred for neurophysiologic studies from the Neurology, Psychiatry, and Veterans' Affairs Outpatient Clinics for episodic rage or violent behavior directed toward others or at inanimate objects, of which they were aware but over which they were unable to exercise control. Seventeen were male and 6 female, and they ranged in age from 9 to 44 years.

Twelve of the 17 males had been involved in physical altercations with or come to the attention of law enforcement agencies while the aggression of 5 of the 6 female patients was verbal in nature or domestic in origin. Seven patients under 21 years of age had been treated for conduct disorder, while 12 of 16 adult patients had previously been given neuroleptics or benzodiazepines for aggressive behavior or had been treated with anticonvulsants for suspected seizures. Five patients used alcohol to excess, another 5 had done so in the past, and 13 patients had had aggressive or violent behavior associated with alcohol use. Fifteen patients had used psychoactive substances in the past, but only 1 was using drugs (infrequent marijuana) at the time of the study. None had identifiable neurologic disorders, and all had normal neurologic examinations, while 10 had had unremarkable imaging studies; 3 patients had histories of febrile convulsions and 8 had suffered past head injury. Two patients had sought treatment of posttraumatic-stress disorder related to military experiences, but had had episodes of aggressive behavior prior to military service. All patients were within the normal range of intelligence, and mentally retarded patients were specifically excluded from the study, but 18 had had learning difficulties or social problems in school, or had required special classes.

Seventeen-channel EEGs were recorded for 60 min on Nihon Kohden 4217 instruments, with gold cup electrodes affixed with collodion at International 10/20 placements. Referential and anteroposterior and transverse bipolar montages were used, along with hyperventilation and intermittent photic stimulation. BAEPs and AEPs were recorded on Nicolet CA-1000 and Pathfinder I systems, utilizing Cz-A1 and Cz-A2 (BAEP) or Cz-A1+A2 (AEP) derivations. BAEP stimulation was with rarefaction clicks at 75 dB SL, with 40 dB contralateral white-noise masking, while AEPs were elicited by binaural stimulation with 1,000 and 3,000 Hz and tones in an 80:20 ratio; BAEPs utilized a stimulus rate of 11.1/s, with filter bandpass of 150–3,000 Hz and 10-msec analysis time. AEPS were recorded with an interstimulus interval of 1.1 s, filter bandpass of 1–100 Hz, and 1-second analysis time.

Results

Sixteen of 23 patients with episodic dyscontrol had normal EEGs. Seven patients, 3 children and 4 adults, had EEGs outside the normal range for age and state of consciousness: 5 EEGs showed background slowing in the θ range, 1 EEG had bitemporal δ slowing, and right-temporal θ/δ slowing was admixed with sharp waves of epileptiform appearance in 1 record. The 5 patients with diffuse background slowing included 2 using alcohol and the 1 who used psychoactive drugs, and all these patients had had perinatal difficulties and school problems, but were neurologically normal and within the normal range of intelligence. Both patients with focal slowing had been diagnosed as epileptic in the past and had been previously but not currently treated with anticonvulsants, and the individual with right-temporal slowing and epileptiform activity had previously suffered head injury involving this region.

Episodic-dyscontrol patients were compared to 20 age-matched patients with mixed-element tension-vascular headaches referred for EEG after normal neurologic examinations and imaging studies, and to 24-age-matched individuals with depression, receiving EEGs as part or preliminary evaluation for organic causes of mental disorder or in preparation for electroconvulsive therapy. Episodic-dyscontrol patients differed significantly from both groups: only 1 headache patient had an normal EEG, with an excess of θ activity ($\chi^2 = 4.68$, $p < 0.05$), while no depressed patient had an abnormal EEG ($\chi^2 = 4.83$, $p < 0.05$).

All BAEPs and AEPs were within clinical normal limits. No statistical differences were demonstrable between patients and controls in BAEP interpeak latencies. The early AEP components (N100 and P160) were lower in amplitude in episodic-dyscontrol patients than in controls, but this did not reach statistical significance, and no difference was evident in latency or amplitude of the later N200 and P300 components between patients and normal controls.

Discussion

The neurophysiologic correlates of violence and aggression have been extensively studied with equivocal results. Silverman [9] found a 53% incidence of EEG abnormality among prisoners, while Gibbs et al. [10] found no differences from normal in a larger group of inmates. Stafford-Clark and Taylor [11] found EEG abnormality in 73% of murderers, especially with motiveless slayings, but only 25% abnormality among criminals generally, while the abnormalities found by Hill [12] involved slowing but no epileptiform potentials. Levy [13] found a two-fold increase in nonspecific EEG abnormality among recidivists, while Kennard et al. [14] found EEG abnormality, mostly slowing, increased among 'psychopathic' criminals but with no relationship to violence. Small [15] found EEG abnormality in one third of felons but a much stronger relationship (77%) between violent or nonviolent criminality and the presence of seizure history, history of head trauma, or neurologic signs. A signif-

icantly increased incidence of EEG slowing or focal abnormality, not necessarily epileptiform, was found in repetitively assaultive patients by Williams [16]. More recent studies [17] have shown no specific relationship between violence and EEG abnormality.

The relationship between violence, aggression, and evidence of epilepsy has also been problematic. Knox [18] reported only 1 of 43 patients with peri-ictal aggression, and this was precipitated by stimulation, particularly touching. Sixteen of 666 complex partial seizure patients reported by Currie et al. [19] had attacks of rage or violence in association with epilepsy, while Gunn and Fenton [20] described 4 prisoners with seizures before violent offenses, 5 with seizures after crime, and 4 with violent attacks of equivocal automaticity. No directed violence was found by Rodin [21] in 42 patients with ictal and 15 with postictal automatisms of potentially violent nature. King and Ajmone Marsan [22] described patients with vigorous and violent-appearing automatisms during or after seizures, but no demonstrabel attacks. The general consensus has since been that directed violence or aggression is an exceedingly infrequent occurrence in patients with definite epilepsy, and is probably interictal and multifactorial.

The initial reports of episodic-dyscontrol syndrome [1, 2] included epileptic patients, and suggested the frequent occurrence of scalp- or depth-recorded EEG abnormalities, as often epileptiform as abnormally slow. These EEG abnormalities and the paroxysmal nature of verbal or physical violence suggested a relationship to seizure disorders, but the precipipation of aggressive behavior by alcohol that is often seen in the disorder [3] is not specific for epilepsy, being seen in some seizure patients and not others; in addition, pathologic intoxication is more often seen with encephalopathic disorders than with epilepsy [5]. Abnormal EEGs in episodic-dyscontrol syndrome have been correlated with past or family psychiatric history [4], and violent individuals have been shown to have histories of childhood behavior disorder and hyperactivity, familial predisposition to alcoholism, and legal difficulties which may in part be economically or situationally determined, more often than abnormal EEGs [5]. Recent studies [6] have shown a high incidence of minimal brain dysfunction and previous history or clinical evidence of encephalopathy, with no specific EEG findings. Our patients had no discernible neurologic or psychiatric disorders in initial outpatient evaluation, and many of them had had unremarkable brain imaging, but histories of borderline, school and vocational performance, past conduct problems, previous head injury, and treatment in the past with various psychotropic or antiepileptic agents may point to the same predisposing factors. Alcohol and drug use prior to evaluation may also have contributed to our patients' clinical features, but were unlikely except in 2 instances to have contributed to their EEG abnormality. It is probable that the degree of predisposing situational factors, substance use or abuse, and minimal brain dysfunction were underestimated, as most were initially evaluated and subsequently followed elsewhere.

Few studies have focused on evoked or event-related potentials in violent or aggressive patients, and, to our knowledge, episodic-dyscontrol syndrome has not been studied with these previously. BAEPs were shown to be relatively prolonged compared to controls in antisocial-personality disorder [23], and we [24] found differences from controls in a largely retarded population with rage attacks and self-injurious behavior. BAEP latencies have been correlated with psychometric measures of personality disturbance [25]. AEPs have been studied in various manifestations of psychopathy, and amplitude and latency differences have led to suggestions that criminal and psychopathic individuals may differ from normals in some aspects of attention [26]. We found AEP latency prolongation in violent as compared to nonviolent prisoners, in particular AEP attenuation over the nondominant hemisphere in violent offenders [8], although the history of violence was limited and available psychiatric information scant in a population of chronically incarcerated individuals. These findings suggest that some types of violence and aggression may reflect dysfunction of subcortical attention or behavior-modulating systems. The reduction in amplitude of early AEP components in episodic-dyscontrol patients, even though not statistically significant, could accord with the earlier findings. Episodic dyscontrol is likely to represent multifactorial cerebral disturbance, however, and the diagnostic utility of event-related potentials is uncertain.

The findings do suggest a screening role for EEG in the evaluation of violent or aggressive individuals. Although nonspecific, EEG abnormality may be associated with similarly nonspecific cerebral dysfunction which could result in disordered impulse and behavior control for which pharmacologic treatment may be appropriate. Clinical evidence of episodic dyscontrol may be associated with other signs of minimal brain dysfunction, which perinatal, developmental and educational history, and psychometric assessment may also help to reveal.

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