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EEG and Cerebral Blood Flow Velocity Abnormalities in Chronic Cocaine Users

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Abstract

EEG and cerebral blood flow abnormalities have been documented in chronic cocaine abusers. To identify possible relationships between EEG and blood flow changes and their relationship to the intensity of cocaine use, we recorded the resting eyes-closed EEG and anterior (ACA) and middle (MCA) cerebral artery blood flow velocity during systole (V_S) and diastole (V_D) by transcranial Doppler (TCD) sonography of 99 (76 male, 23 female; mean [SD] age 34.3 [5.2] years, 8.6 [5.5] years of cocaine use, 17.8 [7.7] days of cocaine use in month prior to screening) cocaine users within 5 days of admission to a closed research unit. Forty-two non-drug-using, age-matched control subjects (22 male, 20 female) were tested as outpatients. A 3-minute period of resting EEG was recorded from 16 standard scalp electrodes. Artifact-free EEG was converted to six frequency bands (delta, theta, alpha1, alpha2, beta1 and beta2) using a Fast Fourier Transform. Pulsatility index (PI) was calculated as a measure of small vessel resistance.

Cocaine users had decreased V_D and increased PI in the MCA, with no difference in V_S , and reduced EEG theta, beta1 and beta2 absolute power in posterior brain regions. Recent cocaine use was positively associated with MCA PI ($r = 0.27$, $p < 0.001$) and negatively associated with low frequency EEG power (delta power: $r = -0.25$, $p < 0.002$; theta power: $r = -0.29$, $p < 0.001$). EEG beta1 ($r = -0.211$, $p < 0.05$) and beta2 ($r = -0.176$, $p < 0.05$) power measures were correlated with PI. These observations suggest that EEG and TCD changes reflect related physiological processes during early cocaine abstinence.

Keywords

Cerebral Blood Flow Velocity; Cocaine; Electroencephalography; Pulsatility Index; Transcranial Doppler Sonography

INTRODUCTION

Neurological complications of cocaine use include strokes,^{1–5} seizures,^{6–8} and headache.^{9–11} Cerebrovascular perfusion deficits have been observed in chronic cocaine users with the techniques of single photon emission computed tomography (SPECT) and

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DISCLOSURE AND CONFLICT OF INTEREST

M. L. Copersino, R. I. Herning, W. Better, J.-L. Cadet and D. A. Gorelick have no conflicts of interest in relation to this article.

magnetic resonance imaging (MRI),^{12–18} primarily in frontal, central and temporal cortices. These perfusion deficits have been found after 6 months of monitored abstinence from cocaine.¹⁸ We have previously reported cerebrovascular deficits detected by transcranial Doppler (TCD) sonography over 4 weeks of monitored cocaine abstinence, including lower systolic and diastolic blood flow velocities and increased pulsatility index in the anterior and middle cerebral arteries.¹⁹

Prolonged use of cocaine is also associated with EEG changes,^{20–31} including decreases in delta and theta and/or increases in alpha and beta frequency band power. These EEG changes have not been clearly associated with specific neurological or cognitive deficits. Alper²⁹ suggested that the decrease in EEG delta activity observed in cocaine abusers is associated with cognitive deficits, while increases in EEG delta activity have been traditionally associated with impaired cognitive function in other disorders.^{32,33}

The increased EEG alpha activity observed in cocaine users^{20,24} might have resulted from cocaine users with co-morbid depression, since those studies included depressed cocaine abusers. Alper and associates²⁰ tested seven depressed cocaine users and found increased absolute and relative alpha power. Roemer and associates²⁴ attributed the increase in relative alpha power to the decrease in power in other bands. Increases in EEG beta observed in cocaine abusers might be linked to increased risk alcohol dependence, conduct disorder and a family history of alcoholism.^{22,34}

The relationship between the cerebrovascular perfusion deficits and the resting EEG changes both observed early in cocaine abstinence remains unclear. The goals of the present study were to (1) examine the relationship between EEG and cerebrovascular changes in chronic cocaine users, and (2) examine the relationship between these changes and history of cocaine use.

METHODS

Subjects

Participants were 99 adult (76 male, 23 female; mean [SD] age 34.3 [5.2] years), chronic cocaine users and 42 adult (22 male, 20 female) non-drug using, age-matched controls recruited from the community. The study was approved by the Institutional Review Board of the National Institute on Drug Abuse (NIDA) Intramural Research Program (IRP). Subjects gave written informed consent and were paid for their research participation. All subjects were physically healthy, based on medical history, physical examination, clinical laboratory tests, and 12-lead ECG. Psychiatric and substance use histories were assessed with the Diagnostic Interview Schedule,^{35,36} Addiction Severity Index (ASI),³⁷ and urine drug testing.

Both groups were primarily African-American. The cocaine users were slightly older, less educated, and more likely male than the controls (Table 1). Cocaine users averaged 8.6 [5.0] years of cocaine use and 17.8 [7.7] days of use in the month prior to screening (Table 2). They were not currently dependent on any substances other than nicotine (and cocaine). Cocaine users were more likely than controls to smoke cigarettes (90.1% vs. 31.0%, $\chi^2 = 56.8$, $df = 1$, $p < 0.001$), but did differ in prevalence of alcohol use (88.8 % vs. 76.2%, $\chi^2 = 74.5$, $df = 1$, $p > 0.05$). Only subjects in the cocaine group used marijuana (80.0%) or heroin (35.4%). Neither group had non-substance-related Axis I psychiatric disorders (DSM-III-R criteria^{35,36}).

Study Design

Cocaine users were studied within 8 days (mean = 1.9, SD= 2.3 days) of admission to a closed research ward. Abstinence on the ward was monitored by searches of personal belongings and mail, absence of visitors or passes, and random urine drug screening. At the study session, TCD was done first, followed immediately by resting EEG. Subjects were not allowed to smoke for at least 20 minutes before testing. Control subjects were tested using the same measures and procedures, except they were tested as outpatients.

EEG Procedures

EEG was recorded for 3 minutes while seated with eyes closed from standard 16 scalp leads (Fp₁, F₃, C₃, P₃, O₁, F₇, T₃, T₅, Fp₂, F₄, C₄, P₄, O₂, F₈, T₄, and T₆). Ear tips served as the reference. The EEG was amplified with Grass (Model 7P511) amplifiers and processed with a 1 to 50 Hz half-amplitude band pass and notch filter at 60 Hz. The EEG was sampled at the rate of 104 samples per second per channel. Artifacts in the EEG were removed by computer assisted visual inspection by an operator blind to the test day and subject. Artifact-free EEG was converted to 6 frequency bands using a Fast Fourier Transformation³⁸: delta (1 to 4 Hz), theta (4 to 8 Hz), alpha (8 to 12 Hz), and beta1 and beta2 power (12 to 40 Hz). Data were analyzed in terms of absolute power in each frequency band.

TCD Procedures

Blood flow velocity was determined using a temporal window (zygomatic arch) for four arteries, right and left middle (MCA) and right and left anterior (ACA) cerebral arteries, using pulsed transcranial Doppler sonography (Model TC2000, Nicolet, Madison, WI). TCD uses a hand-held ultrasound transducer placed against the skull to locate the cerebral artery of interest, based on skull location, angle, and depth setting. A shift in frequency in the reflected sound indicates the blood flow velocity in that artery. Blood flow velocity (cm/s) during cardiac systole (Vs) and diastole (Vd), mean velocity (Vm), and pulsatility index (PI = (Vs–Vd)/Vm) were obtained for each artery. PI is considered to reflect downstream small vessel resistance.^{39,40} Resting heart rate and blood pressure were measured at the time of TCD recording. Because no laterality differences were observed (data not shown), TCD data were analyzed as mean of the left and right side.

Statistical Analysis

Comparisons of subject characteristics between the cocaine and control groups were done by t-test for quantitative variables and by χ^2 test for categorical variables. The effect of cocaine use on EEG power was evaluated with 2-way, group (cocaine vs. controls) by electrode ANOVA applied separately to each frequency band. The effect of cocaine on each TCD measure was evaluated by one-way, between groups (control versus cocaine) ANOVA. Deviations from the assumptions of the repeated measures model were evaluated using the Mauchly's test. When assumptions of the model were violated, the Greenhouse-Geisser corrected p value (P_{G-G}) is given. When assumptions of the model were not violated, the unadjusted p-value (p) is presented. Partial correlation coefficients (controlling for age) were calculated between (1) PI and mean EEG absolute power for each frequency band, (2) cocaine use measures and PI and (3) cocaine use measures and mean EEG absolute power for each frequency band. The two-tailed alpha level was set at $p = 0.05$. Statistical testing was performed with SPSS, Version 13 (SPSS, Inc., Chicago, IL).

RESULTS

Cocaine users had decreased theta, beta1, and beta2 absolute power in posterior scalp regions, with a trend towards decreased delta power in the left parietal region ($p = 0.08$) as

compared to the control subjects (see Figure 1). Recent cocaine use was negatively correlated with EEG power, i.e., the greater the amount of recent cocaine use, the less power in the theta ($r = -0.25$, $df = 137$, $p < 0.001$) and beta1 ($r = -0.19$, $df = 137$, $p < 0.02$) frequency bands.

Cocaine users had decreased diastolic blood flow velocity and increased PI in the MCA compared to controls for both the ACA and the MCA (see Table 3). There was no difference in systolic blood flow velocity between cocaine users and controls. Recent cocaine use was positively-correlated ($r = 0.27$, $df = 137$, $p < 0.001$) with PI, i.e., the greater the amount of recent cocaine use, the greater the PI. There was no significant correlation between either systolic or diastolic blood flow velocity and cocaine use.

There were no significant correlations between velocity measures and absolute power in any of the 6 EEG frequency bands (Table 4). PI was inversely correlated with beta1 and beta2 power (Table 4).

DISCUSSION

The present study found that, in comparison to controls, recently abstinent cocaine users showed decreases in absolute EEG power in theta, beta1, and beta2 frequency bands in posterior brain regions. The theta findings are similar to other published studies.^{20,24,25,27} The finding of decreased beta power appears inconsistent with prior findings from this laboratory^{28,30} and others showing increased beta power among cocaine users.²⁶ This may be due to different analytic techniques or to differences in subject gender, alcohol use, or prevalence of anti-social personality disorder or family history of alcoholism, all of which have been associated with changes in beta power. Both alcohol dependence and a family history of alcoholism have been associated with increased beta power.³⁴ Women have less increased beta power than do men in association with cocaine³⁰ or alcohol use.³⁴

Diastolic blood flow velocity measured by TCD was significantly greater in the anterior and middle cerebral arteries of controls compared with chronic cocaine abusers, while PI, a measure of small vessel resistance,^{39,40} was significantly elevated in cocaine users compared with controls. This is consistent with our earlier findings in a smaller sample of cocaine users.¹⁹ Cerebral perfusion deficits in abstinent cocaine users have been observed with other methodologies, such as SPECT and MRI.^{12–18} Recent cocaine use was positively-correlated with PI, i.e., the greater the amount of recent cocaine use, the greater the PI. There was no significant correlation between cocaine use and either systolic or diastolic blood flow velocity. Recent cocaine use was negatively correlated with EEG power, i.e., the greater the amount of recent cocaine use, the less power in the theta, and beta bands. There were no significant correlations between blood flow velocity measures and any of the six EEG frequency bands. The pulsatility index was inversely correlated beta1 and beta2 power, i.e., the greater the PI or resistance in small cerebral blood vessels, the lower the power in the beta EEG bands.

In previous EEG studies of cocaine abusers by this laboratory²⁸ and others,²⁶ relative EEG beta power was increased in cocaine users as compared to control subjects. Costa and Bauer³¹ were the only group of researchers to observe an increase in absolute EEG beta power in cocaine abusers compared to control subjects. The present study found a decrease in both beta1 and beta2 power in cocaine users, which was correlated with an increase in cerebral small vessel resistance as measured by TCD sonography. In studies of ischemic stroke, increases in delta and theta EEG activity have been associated with deficits in cerebral perfusion, while decreases in beta activity have been associated with subtle decreases in brain perfusion.⁴¹

The present study has several strengths and was performed on a large sample of well characterized control subjects and cocaine users. Both EEG and cerebral perfusion were measured at the same time in the same subjects, allowing for direct comparison of the two measures.

This study has several limitations. The two groups were not age or sex matched, although age was statistically controlled by using an analysis of covariance or partial correlation analyses. While one would not expect to see gender differences in the EEGs of healthy adults, resting EEG is relatively stable throughout healthy adult life and TCD changes start above age 50, or in post-menopausal women, age was used as a statistical control throughout the present study.

In conclusion, these TCD findings are consistent with those from SPECT and fMRI showing abnormal brain perfusion in cocaine users. EEG findings are also consistent with those observed in abstinent cocaine users in other studies. Our study shows a relationship between the cerebral perfusion and EEG in abstinent cocaine abusers suggesting the probability of using these parameters as potential markers of treatment response.

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Figure 1.

Visual representation of log transformed absolute EEG power in each of the six frequency bands for the control group (1st column) and cocaine-using group (2nd column). The 3rd column shows the statistical significance of the difference between the two groups (see text for description of statistical methods). Vertical bar at lower right shows color code for EEG power (left side) and p value (with Bonferroni correction) of group difference (right side). In comparison to controls, cocaine users showed decreased power in posterior regions in theta, beta1, and beta2 frequency bands.

Table 1

Subject demographic characteristics

	Cocaine (n = 99)		Control (n = 42)	
	Mean	SD	Mean	SD
Age [*] (yrs)	34.3	5.2	28.2	8.1
Education ^{**} (yrs)	12.0	1.6	12.9	1.6
Female [*] (%)	23.3	—	47.6	—
African-American (%)	84.8	—	71.4	—

Group differences:

^{*}
p<0.001,^{**}
p<0.01

Table 2

Subjects' substance use history

	Cocaine (n = 99)		Control (n = 42)	
	Mean	SD	Mean	SD
Alcohol				
Current use (days/month) [#]	8.5	6.6	1.4	4.8
Lifetime use (years)	14.4	7.0	3.4	6.1
Marijuana				
Current use (days/month) [#]	2.5	5.2	—	—
Lifetime use (years)	10.0	8.5	—	—
Cocaine				
Current use (days/month) [#]	17.8	7.7	—	—
Lifetime use (years)	8.6	5.0	—	—
Cigarettes per day [*]	17.0	10.4	15.0	10.0
Years smoking [*]	17.5	6.8	11.5	6.9
Heroin				
Current use (days/month)	0.9	1.5	—	—
Lifetime use (years)	4.8	1.0	—	—

^{*} n = 90 (90.1%) for cocaine group, n = 13 (31.0%) for controls

[#] In month prior to study screening

Table 3

Effect of cocaine use on transcranial doppler measures

	Control (n = 42)		Cocaine Users (n = 99)		F	P
	Mean	SD	Mean	SD		
Middle Cerebral Artery						
Diastolic Velocity (cm/s)	42.3	9.5	37.3	6.9	8.8	0.003
Systolic Velocity (cm/s)	97.8	20.7	91.3	18.5	1.1	0.29
Mean Velocity (cm/s)	60.8	12.8	55.3	10.2	4.0	0.05
Pulsatility Index (PI)	0.89	0.12	0.96	0.11	17.5	0.001
Anterior Cerebral Artery						
Diastolic Velocity (cm/s)	39.8	7.8	34.0	8.5	16.0	0.001
Systolic Velocity (cm/s)	92.8	19.4	81.4	14.7	7.4	0.07
Mean Velocity (cm/s)	56.7	11.0	49.5	9.3	10.9	0.001
Pulsatility Index (PI)	0.90	0.16	0.93	0.12	3.8	0.05

Table 4

Correlation between transcranial doppler measures and EEG power

EEG Frequency Band	Diastolic Velocity	Systolic Velocity	Mean Velocity	Pulsatility Index
Delta	.071	.056	.093	-.118
Theta	-.001	-.008	-.001	-.103
Alpha1	.005	-.005	.005	-.135
Alpha2	.029	.016	.048	-.116
Beta1	.100	.030	.088	-.211 *
Beta2	.090	.038	.098	-.176 *

*
p < 0.05,

df =139, controlled for age.