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Similar or Disparate Brain Patterns? The Intra-Personal EEG Variability of Three Women With Multiple Personality Disorder

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Key Words

Dissociative Identity Disorder
Interpersonal EEG Variability
Intra-personal EEG Variability
Multiple Personality Disorder
Quantitative EEG

ABSTRACT

Quantitative EEG was used to assess the intra-personal variability of brain electrical activity for 3 women diagnosed with Multiple Personality Disorder (MPD). Two separate control groups (within-subject and between-subject) were used to test the hypothesis that the intra-personal EEG variability between 2 alters would be less than the interpersonal EEG variability between 2 controls, and similar to the intra-personal EEG variability of a single personality. This hypothesis was partially supported. In general, the 2 EEG records of a MPD subject (alter 1 vs. alter 2) were more different from one another than the 2 EEG records of a single control, but less different from one another than the EEG records of 2 separate controls. Most of the EEG variability between alters involved beta activity in the frontal and temporal lobes.

INTRODUCTION

Dissociative Identity Disorder (DID),¹ formerly called Multiple Personality Disorder (MPD),² is characterized by "the presence of two or more distinct identities or personality states (each with its own relatively enduring pattern of perceiving, relating to, and thinking about the environment and self)... [that] recurrently take control of the person's behavior" (p. 529).¹ Patients with DID typically switch between three and nine different personalities, often referred to as "alters." Frequently, there is a dominant or core personality (i.e., "host") and several subordinate alters.^{1,3} Generally, each alter has a well-defined role (e.g., protector) and limited access to certain autobiographical memories.^{1,3,5} Most cases of DID (over 90% in some reports) involve female patients with a self-reported history of severe childhood abuse.^{1,3,5,8}

Some clinicians believe that DID is extremely rare, whereas other clinicians believe it is vastly under diagnosed.^{1,3,5,8} Recent surveys in the United States and Canada suggest that many North American psychiatrists

are highly skeptical of DID. Pope et al.⁹ acquired survey data from 301 board certified American psychiatrists and reported that 57% of these psychiatrists concurred that DID should either be excluded from the Diagnostic and Statistical Manual of Mental Disorders (DSM), or included solely as a proposed diagnosis or "iatrogenic" condition (i.e., a condition that is caused by the clinician's diagnosis or treatment). Furthermore, 71% of these psychiatrists reported that DID is supported by partial to no scientific evidence. A similar survey of Canadian psychiatrists found even higher rates of professional skepticism toward DID.¹⁰

One approach to the investigation of dissociative phenomena is to utilize objective measures of brain activity. Some researchers believe that dissociative states are regulated by the prefrontal cortices and temporal lobes.¹¹⁻¹³ The prefrontal cortices seem to be actively involved in the organization of behavior, the regulation of emotion, and the inhibition of impulsive urges. The prefrontal cortices have strong connections with limbic structures in the temporal lobes.¹⁴ Damage to the prefrontal cortices can lead to severe changes in personality, whereas damage to the temporal lobes can cause dissociative symptoms such as déjà vu and amnesia.^{4,11,14,15}

The stability and specificity of brain electrical activity make EEG an excellent modality to use in the assessment of DID. Extensive data suggest that the healthy adult undergoes few significant long-term changes in EEG between the ages of 20 and 60.¹⁶ Additional data suggest that the "between-subject" or interpersonal EEG variance is greater than the "within-subject" or intra-personal variance of EEG when identical recording conditions are

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Table 1

Subject	Session 1	Session 2
Control 1	Baseline	Baseline
Control 2	Baseline	Baseline
Control 3	Baseline	
Control 4	Baseline	
MPD 1	Alter 1 (5-year-old)	Alter 2 (32-year-old)
MPD 2	Alter 1 (unknown)	Alter 2 (unknown)
MPD 3	Alter 1 (male taxi driver)	Alter 2 (host personality)

Note: Session 1 and Session 2 were between 2 and 10 days apart. Session 2 data were not acquired for Control 3 and Control 4.

used.^{17,18} Together, these data imply that the human EEG is a fairly stationary process that is heavily influenced by individual differences.

A consistent finding in the literature is that patients with MPD or DID demonstrate variable EEGs when their multiple alters are examined.^{19,22} Hughes, Kuhlman, Fichtner, and Gruenfeld²⁰ (p.208) even stated that "a rank ordering of the differences in the [EEGs] of the alternate personalities from [a single MPD subject] were similar to the rank ordering of the differences in personality characteristics, as judged by the psychiatrist dealing with this patient." Critics of these findings contend that alter-related differences in EEG are attributable to EEG artifact or changes in mood and alertness rather than changes in personality.²³⁻²⁵ Furthermore, some data suggest that the intra-personal variability of EEG is just as high for healthy controls as it is for patients diagnosed with MPD.²³ In fact, Coons, Milstein, and Marley²³ reported that a healthy control who simulated different personalities during EEG was able to alter the relative amplitude of five frequency bands more significantly than two MPD patients who reportedly switched personalities during EEG.

The purpose of this study was to examine the intra-personal EEG variability for 3 patients diagnosed with MPD. Because EEG varies more between people than it does in the same person over time, it was hypothesized that the intra-personal EEG variability between 2 alters would be less than the interpersonal EEG variability between 2 controls, and similar to the intra-personal EEG variability of a single personality.

METHOD

Participants

The patient group included 3 females (mean age = 33.3; SD = 1.2) who were diagnosed with MPD according to the DSM-III-R.² All of these women were white and right-handed. Each one was reported to have at least five alternate personalities and a history of self-mutilation and severe

childhood abuse. These subjects are referred to as MPD 1, MPD 2, and MPD 3 throughout the rest of this paper.

The control group included 4 females (mean age = 26; SD = 8.7) who denied a history of mental illness or substance abuse. All of these women were white and right-handed. These subjects are referred to as Control 1, Control 2, Control 3, and Control 4 throughout the rest of this paper.

Procedure

Nineteen electrodes (FZ, FP1, FP2, F3, F4, F7, F8, CZ, C3, C4, T3, T4, T5, T6, PZ, P3, P4, O1, O2) were placed on the participant's scalp following the International 10/20 System and referenced to linked ears. EEG was recorded and quantified with the Neuroscan acquisition system.²⁶ The sampling rate of the data was 128 Hz with filter settings at 0.01 Hz and 70 Hz. Following EEG acquisition, a low band pass filter was used to attenuate the frequencies above 30 Hz.

EEG was recorded while the subjects sat in a reclining chair with their eyes closed. A video camera system was used to monitor all subjects' behaviors. During the recording of EEG, the subjects received an intravenous injection ([^{99m}Tc]-HMPAO) that was later used to administer a brain SPECT scan. The brain SPECT data are not reported in this paper.

Between 2 and 10 days later, the EEG and brain SPECT procedures were repeated on Control 1, Control 2, and all 3 MPD subjects. These 2 sets of data are referred to as session 1 and session 2 throughout the rest of this paper. Both sessions reflected baseline EEG measurements for Control 1 and Control 2. For the MPD subjects, each session captured the EEG of a separate alter. MPD 1 was a 5-year-old girl during session 1 and a 32-year-old female during session 2. MPD 3 was a male taxi driver during session 1 and the host personality, that is a 34-year-old female, during session 2. No descriptors were available for the two personalities of MPD 2. In each case, a therapist who had experience working with the patient helped draw out the identified alter. The design of the study is depicted in Table 1.

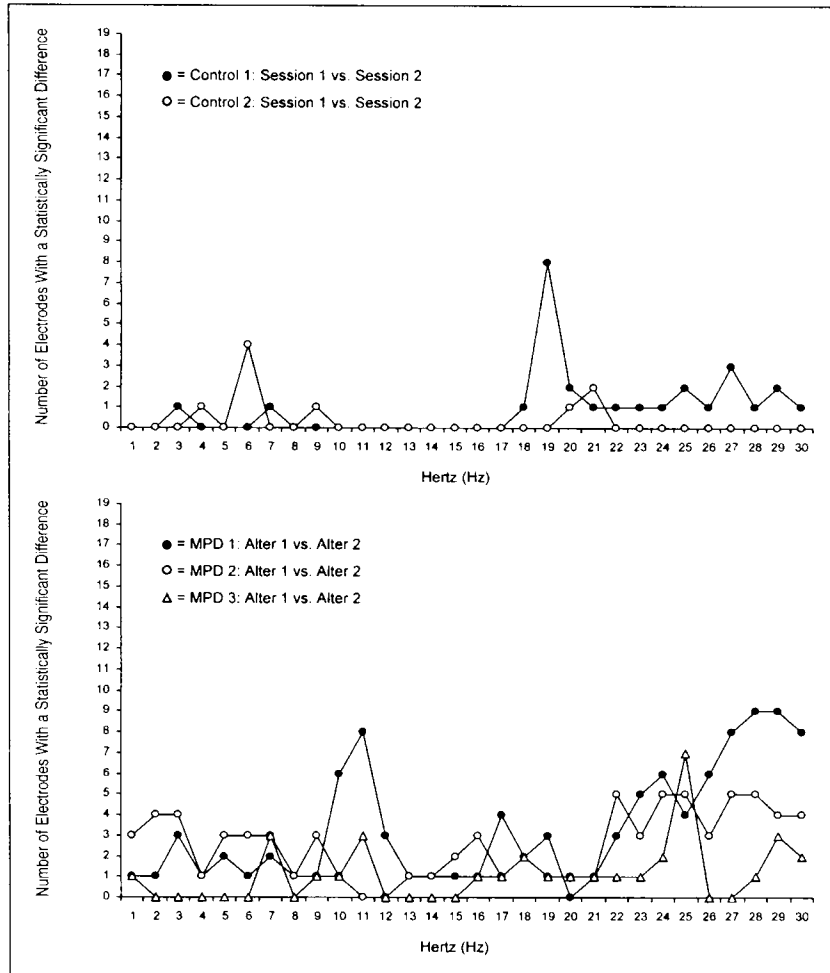
Prior to recording EEG, all subjects were screened with a urine analysis (UA). UA results indicated that 5 of the 6 subjects were negative for alcohol, illicit substances, and psychotropic medications. MPD 3 tested positive for a benzodiazepine (session 1) and barbiturate (sessions 1 and 2). MPD 3 was not excluded from the study because of the limited sample size and difficulty finding patients with a diagnosis of MPD. The effects of psychotropic medications on EEG are considered in the discussion section.

Data Processing

NeuroGuide 1.7.9²⁷ was used to edit the EEG files prior to performing statistics. The method for selecting clean EEG was based on criteria used by Thatcher, North, and Biver²⁸ and Thatcher et al.²⁹ The NeuroGuide "automatic

Figure 1.

Statistically significant differences in FFT relative power between sessions for 2 controls (top graph) and between alters (bottom graph) for 3 participants with MPD. The horizontal axis is the 1-Hz frequency band that independent t-tests were run on. The vertical axis is the number of electrodes in which statistical significance ($p \leq .001$) was found for that frequency band.



selection” was used to select artifact-free segments of EEG that were representative of an eyes closed and relaxed state of arousal. Following the automatic selection, each EEG file was visually edited until it was between 60 and 90 seconds long and had an average electrode split-half reliability that was greater than .95.

Data Analysis

NeuroStat, a supplementary component of NeuroGuide, was used to run statistics on EEG relative power. Relative power is a preferred EEG measure for between-subject comparisons because it is not influenced by skull or skin thickness.³⁰ Independent t-tests were used to detect statistically significant differences in relative power for 1-Hz frequency bands between 1 Hz and 30 Hz. Initially, the 4 controls’ EEG records, all from session 1, were compared to one another to establish interpersonal differences in relative power (Control 1 vs. Control 3, etc). Secondly, the 2 EEG records of Control 1 and Control 2 were compared to one another to assess intra-personal differences in rela-

tive power (session 1 vs. session 2). Finally, the 2 EEG records of each MPD subject were compared to one another to assess intra-personal differences in relative power between alters (alter 1 vs. alter 2).

Hypothesis Testing

Every statistical comparison of EEG resulted in 570 p-values (30 1-Hz frequency bands x 19 electrodes). The hypothesis in this study was tested by counting the number of p-values that were equal to or less than .001. The hypothesis was supported if the number of statistically significant differences ($p \leq .001$) between 2 alters’ EEGs was always less than the number of statistically significant differences ($p \leq .001$) between 2 separate controls, and similar to the number of statistically significant differences ($p \leq .001$) between the 2 EEG sessions of a single control. EEG frequencies were grouped into delta (1-3 Hz), theta (4-7 Hz), alpha (8-12 Hz), beta-1 (13-18 Hz), beta-2 (19-25 Hz) and beta-3 (26-30 Hz) frequency bands to facilitate the interpretation of the results.

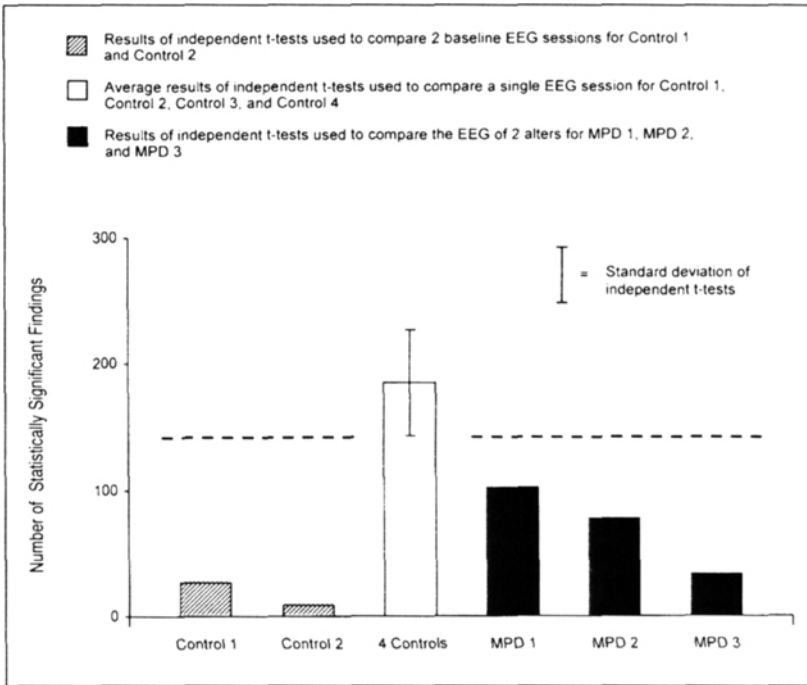


Figure 2. Statistically significant differences in FFT relative power (a) within 2 controls, (b) between 4 controls, and (c) between the two alters of 3 subjects with MPD. The vertical axis is the total number of statistically significant findings ($p \leq .001$) that resulted when independent t-tests were run on 1-Hz frequency bands between 1 Hz and 30 Hz.

RESULTS

Interpersonal EEG Variability for Healthy Controls

A between-subject comparison revealed that all 4 of the controls had many differences in 1-Hz frequency band relative power that were statistically significant at $p \leq .001$. Significant differences ($p \leq .001$) between controls were found in every 1-Hz frequency band between 1 Hz and 30 Hz. Interpersonal differences ($p \leq .001$) in alpha relative power spanned most of the cortex, whereas interpersonal differences ($p \leq .001$) in delta relative power were generally restricted to a few leads.

Intra-personal EEG Variability for Healthy Controls

Intra-personal differences in 1-Hz frequency band relative power were rarely significant at more than 3 electrodes when the 2 EEG sessions for Control 1 and Control 2 were compared to one another. Delta, alpha, and beta-1 relative power varied the least between sessions, whereas beta-2 and beta-3 relative power varied the most. A consistent finding for both subjects was a significant decrease ($p \leq .001$) in beta-2 relative power in the left temporal lobe, particularly the middle temporal lobe. The intra-personal stability of EEG relative power across sessions for Control 1 and Control 2 is depicted in Figure 1.

Intra-Personal EEG Variability for Subjects with MPD

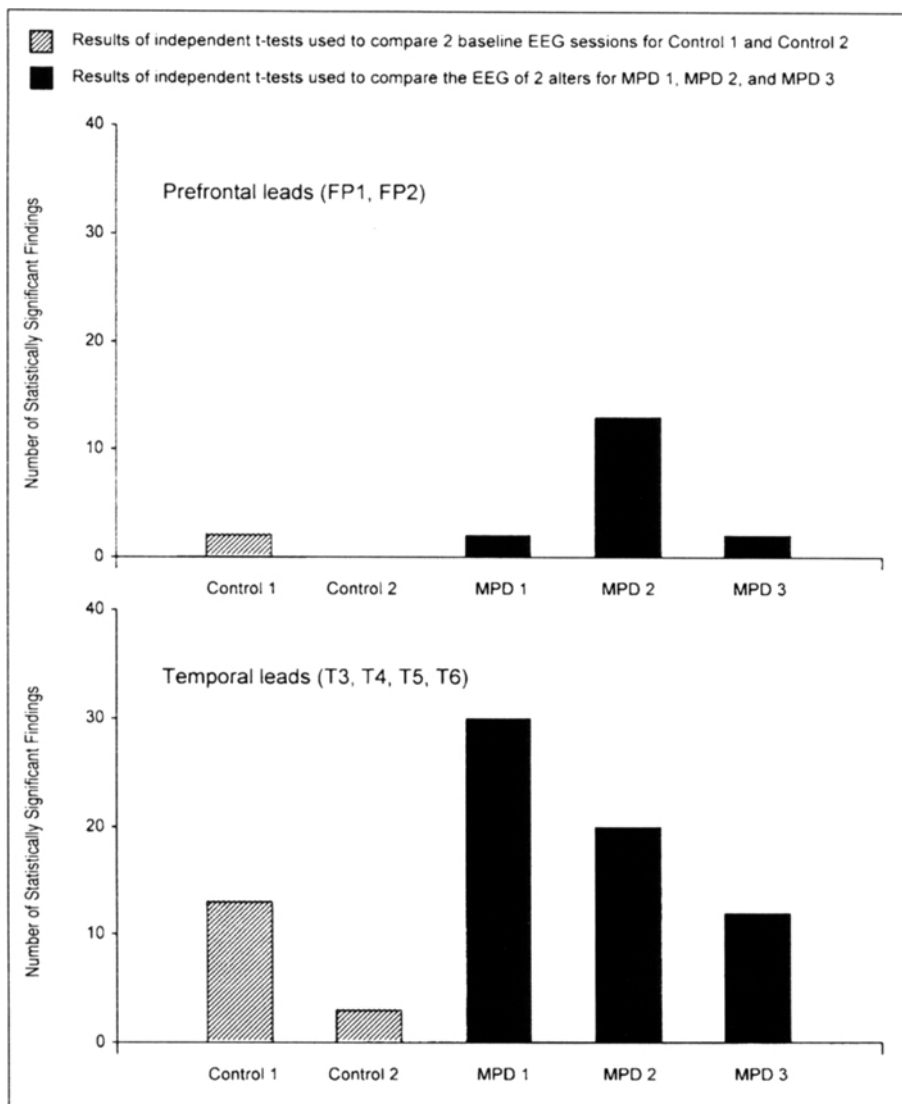
Intra-personal differences ($p \leq .001$) in fast activity, particularly beta-2 and beta-3 were detected throughout the frontal and temporal lobes whenever 2 alters EEGs were compared to one another. The most significant findings were in the left middle temporal lobe. A consistent finding across subjects was a significantly greater amount ($p \leq .001$) of

beta-2 or beta-3 relative power in the right prefrontal lobe during the first alter compared to the second. In the case of MPD 2 significant differences in prefrontal relative power, both slow and fast activity, were quite pronounced. Prefrontal fast activity was significantly higher ($p \leq .001$) during the first alter of MPD 2, whereas prefrontal slow activity was significantly lower ($p \leq .001$). In addition to the aforementioned differences in beta activity, the two alters of MPD 1 demonstrated varying amounts of ($p \leq .001$) of alpha relative power in the posterior regions, particularly the occipital and right temporal lobes. The intra-personal stability of EEG relative power across alters for MPD 1, MPD 2, and MPD 3 is depicted in Figure 1.

Number of Statistically Significant Differences in EEG Relative Power (a) Between Controls, (b) Within Controls, and (c) Between Alters

On average, there were 185.2 ± 42.1 statistically significant differences ($p \leq .001$) in relative power between the EEG records of 2 controls, 71 ± 35.0 statistically significant differences ($p \leq .001$) in relative power between the EEG records of 2 alters, and 18 ± 12.7 statistically significant differences ($p \leq .001$) in relative power between the 2 EEG records of Control 1 and Control 2. Thus, our hypothesis was partially supported. Unexpectedly, MPD 1 and MPD 2 experienced more changes in EEG across sessions than MPD 3, Control 1, or Control 2, in part because of the significant shifts in temporal or prefrontal lobe beta. Figures 2 and 3 highlight these results. The results from all 19 electrodes are displayed in Figure 2, whereas only the results from the temporal and prefrontal leads are displayed in Figure 3.

Figure 3. Statistically significant differences in fast beta (19-30 Hz) relative power at prefrontal (top graph) and temporal (bottom) leads. The vertical axis is the total number of statistically significant findings ($p \leq .001$) that resulted when independent t-tests were run on 1-Hz frequency bands between 19 Hz and 30 Hz.



DISCUSSION

Consistent with previous reports, some of the MPD subjects had significantly different EEGs depending on the personality they were in.^{20,22} When the results from these subjects were compared to the results from 2 separate control groups, an interesting pattern emerged. In general, the 2 EEG records of a MPD subject (alter 1 vs. alter 2) were more different from one another than the 2 EEG records of a healthy control (session 1 vs. session 2), but less different from one another than the EEG records of 2 healthy controls (Control 1 vs. Control 2). Thus, although a patient's EEG changed according to alter, it still maintained its specificity.

Unlike either of the controls, MPD 1 exhibited a fairly pronounced change in alpha relative power at the occipital and temporal leads. In particular, MPD 1 had more occipi-

tal alpha (11 Hz) during her 5-year-old alter than during her 32-year-old alter. This finding contradicts what is known about the effects of maturation on EEG. For the most part, alpha waves above 10 Hz are not well established until adulthood. On the contrary, it is slow wave activity that predominates the EEG during childhood.^{31,32} Because 5-year-olds are expected to have less, not more, posterior alpha than adults, the decrease in occipital alpha that occurred with MPD 1 was probably the result of an increase in drowsiness or mental activity, two conditions known to attenuate occipital alpha.^{31,32}

Much of the EEG variance between alters was attributable to differences within the beta-2 and beta-3 frequency bands, hereafter referred to as "fast beta." Extreme differences in prefrontal or temporal lobe fast beta were char-

acteristic of two of the MPD subjects. Unlike either of the controls, all 3 of the MPD subjects demonstrated a significant decrease in prefrontal fast beta, predominately on the right side, when their first EEG was compared to their second. Interestingly, some researchers believe that the right prefrontal cortex helps regulate the representation of the self-concept.³³

Similar to the results from this study, Hughes et al.²⁰ and Cocker et al.²² reported that EEG activity above 18 Hz varied the most between alters at the temporal or frontal leads. Growing evidence suggests that fast beta is positively correlated with metabolic activity in the temporal and frontal regions.³⁴ Additional data suggest that changes in temporal and frontal lobe activation occur during dissociation.³⁵⁻³⁹ Although it is possible that dissociation was related to some of the EEG changes that occurred in our study, a careful interpretation of the data suggests that additional factors could have played a significant role.

The presence of prefrontal fast beta, particularly on the right side, may be associated with increased vigilance or fear. The right prefrontal cortex is believed to regulate threat-related emotions.⁴¹⁻⁴³ Increased anterior right side activation can occur while people experience or anticipate fearful events.⁴¹⁻⁴⁴ Perhaps the MPD subjects in our study were more tense and anxious during their first EEG because they were less familiar with the examiners and testing procedures. Secondly, they may have had more stressful memories associated with their first alter compared to their second. Either one of these factors could have resulted in more prefrontal fast beta during their first EEG.

In the case of MPD 3, the difference in fast beta between alters was likely a medication effect. As previously reported, MPD 3 had a benzodiazepine in her system during the first EEG, but not during the second. Benzodiazepines are known to increase fast beta in the anterior segments of the brain.³¹ Hence, it is not surprising that the first alter of MPD 3 generated more frontal lobe beta than the second alter did. Furthermore, MPD 3 demonstrated minimal changes in EEG while being the only subject with a mood-stabilizing agent (i.e., barbiturate) in her system on both days EEG was recorded. This finding is significant in that one of the major criticisms of research on MPD is that alter related differences in EEG are attributable to changes in mood not identity.^{23,24} Perhaps this criticism applies here. On the other hand,

mood-stabilizing anticonvulsants have been reported to exert stabilizing effects on dissociative phenomena as well.⁴⁵

Limitations

The findings in this study should be interpreted with caution due to limitations in the methodology of this study and in current EEG techniques. Obviously, the small sample size of this study limits the extent to which the results can be generalized to larger populations. For this reason, it is recommended that this study be replicated on a much larger sample. In addition, it is possible that the restrictive laboratory conditions necessary for recording clean EEG (eyes closed, seated and still) were not ideal for eliciting full shifts in personality. Last, but not least, the MPD subjects did not rate their mood while in different personality states. Hence, it is difficult to assess the degree to which mood may have influenced EEG.

Future Directions

Additional research on the stability of EEG in normative and psychiatric populations is necessary to clarify the significance of alter-specific changes in EEG. Future researchers should continue to investigate the extent to which subjects can influence EEG by performing cognitive tasks,^{46,47} pretending to have multiple personalities,^{20,21,23} entering trance-like states (e.g., meditation),⁴⁶⁻⁵¹ and focusing on emotionally laden memories with varying degrees of self-talk⁵² and visual imagery.⁵³ It would also be interesting to acquire multiple EEGs from the same alter to assess the test-retest reliability of that alter's EEG. Poor reliability across multiple EEG records would suggest that the alter does not have a specific brain wave pattern. Finally, it is important for future investigators to help clarify which parts of the brain and which EEG measures demonstrate the greatest between-subject variance.

CONCLUSIONS

Some patients diagnosed with dissociative disorders demonstrate unusual changes in EEG that coincide with reported shifts in personality. With that said it is still unclear as to whether or not these EEG changes are directly linked to dissociation. Future research is necessary to establish the extent to which variables unrelated to dissociation (e.g., anxiety, medication) can influence these patients' EEGs.

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REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. revised 4th ed. Washington, DC; 2000.
2. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. revised 3rd ed. Washington, DC; 1987.
3. Coons PM. The dissociative disorders: rarely considered and underdiagnosed. *Psychiatr Clin North Am* 1998; 21: 637-649.
4. McDowell, DM, Levin, FR, Nunes, EV. Dissociative identity disorder and substance abuse: the forgotten relationship. *J Psychoactive Drugs* 1999; 31: 71-83.
5. Kaplan HI, Sadock BJ. Kaplan and Sadock's Synopsis of Psychiatry: Behavioral Sciences, Clinical Psychiatry. 8th ed. Baltimore: Williams & Wilkins; 1998.
6. Lewis DO, Yeager CA, Swica Y, Pincus JH, Lewis M. Objective documentation of child abuse and dissociation in 12 murderers with dissociative identity disorder. *Am J Psychiatry* 1997; 154: 1703-1710.
7. Coons PM. Confirmation of childhood abuse in child and adolescent cases of multiple personality disorder and dissociative disorder not otherwise specified. *J Nerv Ment Dis* 1994; 182: 461-464.
8. Lilienfeld SO, Lynn SJ, Kirsch I, Chaves JF, Sarbin TR, Ganaway GK, Powell RA. Dissociative identity disorder and the sociocognitive model: recalling the lessons of the past. *Psychol Bull* 1999; 125: 507-523.
9. Pope HG, Oliva PS, Hudson JI, Bodkin AJ, Gruber AJ. Attitudes toward DSM-IV dissociative disorders diagnoses among board-certified American psychiatrists. *Am J Psychiatry* 1999; 156: 321-323.
10. Lalonde JK, Hudson JI, Gigante RA, Pope HG. Canadian and American psychiatrists attitudes toward dissociative disorders diagnoses. *Can J Psychiatry* 2001; 46: 407-412.
11. Forrest KA. Toward an etiology of dissociative identity disorder: a neurodevelopmental approach. *Conscious Cogn* 2001; 10: 259-293.
12. Sar V, Unal SN, Kiziltan E, Kundakci T, Ozturk E. HMPAO SPECT study of regional cerebral blood flow in dissociative identity disorder. *J Trauma Dissociation* 2001; 2: 5-25.
13. Mathew RJ, Jack RA, West, WS. Regional cerebral blood flow in a patient with multiple personality. *Am J Psychiatry* 1985; 142: 504-505.
14. Mesulam MM. Behavioral neuroanatomy: large-scale networks, association cortex, frontal syndromes, the limbic system, and hemispheric specialization. In: Mesulam MM (ed). *Principles of Behavioral and Cognitive Neurology*. 2nd ed. New York: Oxford University Press, Inc.; 2000.
15. Schomer DL, O'Connor M, Spiers P, Seeck M, Mesulam MM, Baer D. Temporolimbic epilepsy and behavior. In: Mesulam MM (ed). *Principles of Behavioral and Cognitive Neurology*. 2nd ed. New York: Oxford University Press, Inc.; 2000.
16. Fisch B. Fisch and Spehlmann's EEG Primer. 3rd ed. Amsterdam, The Netherlands: Elsevier Science; 1999.
17. Kondacs A, Szabo M. Long-term intra-individual variability of the background EEG in normals. *Clin Neurophysiol* 1999; 110: 1708-1716.
18. Corsi-Cabrera M, Solis-Ortiz S, Guevara MA. Stability of EEG inter- and intrahemispheric correlation in women. *Electroencephalogr Clin Neurophysiol* 1997; 102: 248-255.
19. Putnam FW. The psycho-physiologic investigation of multiple personality disorder: a review. *Psychiatr Clin North Am* 1984; 7: 31-39.
20. Hughes JR, Kuhlman DT, Fichtner CG, Gruenfeld MJ. Brain mapping in a case of multiple personality. *Clin Electroencephalogr* 1990; 21: 200-209.
21. Hopper A, Ciociari J, Johnson G, Spensley J, Sergejew A, Stough C. EEG coherence and dissociative identity disorder: comparing EEG coherence in DID hosts, alters, controls, and acted alters. *J Trauma Dissociation* 2002; 3: 75-87.
22. Cocker KI, Edwards GA, Anderson JW, Meares RA. Electrophysiological changes under hypnosis in multiple personality disorder: a two-case exploratory study. *Aust J Clin Exp Hypn* 1994; 22: 165-176.
23. Coons PM, Milstein V, Marley C. EEG studies on two multiple personalities and a control. *Arch Gen Psychiatry* 1982; 39: 823-825.
24. Perlini AH, Spanos NP. EEG alpha methodologies and hypnotizability: a critical review. *Psychophysiology* 1991; 28: 511-530.
25. Bowman ES, Coons PM. The differential diagnosis of epilepsy, pseudoseizures, dissociative identity disorder, and dissociative disorder not otherwise specified. *Bull Menninger Clin* 2000; 64: 164-180.
26. Neuroscan (Computer software). Sterling, VA: Neurosoft, Inc.; 2001.
27. NeuroGuide – Version 1.7.9 (Computer software). St. Petersburg, FL: Applied Neuroscience, Inc.; 2004.
28. Thatcher RW, North D, Biver C. Evaluation and validity of a LORETA normative EEG database. *Clin EEG Neurosci* 2005; 36: 116-112.
29. Thatcher RW, Walker RA, Biver C, North D, Curtin R. Quantitative EEG normative databases: validation and clinical correlation. *J Neurother* 2003; 7: 87-122.
30. Congedo M, Lubar JF. Parametric and non-parametric analysis of QEEG: normative database comparisons in electroencephalography: a simulation study on accuracy. *J Neurother* 2003; 7: 1-29.
31. Hughes J. EEG in Clinical Practice. 2nd ed. Newton, MA: Butterworth-Heinemann; 1994.
32. Rowan AJ, Tolunsky E. Primer of EEG with a Mini-Atlas. Pennsylvania, PA: Butterworth-Heinemann; 2003.
33. Kelley WM, Macrae CN, Wyland CL, Caglar S, Inati S, Heatherton TF. Finding the self? An event-related fMRI study. *J Cogn Neurosci* 2002; 14: 785-794.
34. Oakes TR, Diego AP, Hendrick AM, Horras KA, Larson CL, Abercrombie HC, et al. Functional coupling of simultaneous electrical and metabolic activity in the human brain. *Hum Brain Mapp* 2004; 21: 257-20.
35. Lanius RA, Williamson PC, Boksman K, Densmore M, Gupta M, Neufeld RJ, et al. Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. *Biol Psychiatry* 2002; 52: 305-311.
36. Hollander E, Carrasco JL, Mullen LS, Trugold S, DeCaria CM, Towey J. Left hemispheric activation in depersonalization disorder: a case report. *Biol Psychiatry* 1992; 31: 1157-1162.
37. Braun BG. Neurophysiologic changes in multiple personality due to integration: a preliminary report. *Am J Clin Hypn* 1983; 26: 84-92.

38. Saxe GN, Vasile RG, Hill TC, Bloomingdale K, Van Der Kolk BA. SPECT imaging and multiple personality disorder. *J Nerv Ment Dis* 1992; 180: 662-663.
39. Mathew RJ, Jack RA, West W. Regional cerebral blood flow in a patient with multiple personality. *Am J Psychiatry* 1985; 142: 504-505.
40. Tsai GE, Condie D, Wu MT, Chang IW. Functional magnetic resonance imaging of personality switches in a woman with dissociative identity disorder. *Harv Rev Psychiatry* 1999; 7: 119-122.
41. Davidson RJ, Marshall JR, Tomarken AJ, Henriques JB. While a phobic waits: regional brain electrical activity and autonomic activity in social phobics during anticipation of public speaking. *Biol Psychiatry* 2000; 47: 85-95.
42. Davidson, RJ. What does the prefrontal cortex do in affect: perspectives on frontal EEG asymmetry research. *Biol Psychiatry* 2004; 67: 219-233.
43. Davidson RJ. Affective styles and affective disorders: perspectives from affective neuroscience. *Cogn Emotion* 1998; 12: 307-320.
44. Rauch SL, Savage CR, Alpert NM, Dougherty D., Kendrick A, Curran T, et al. Probing striatal function in obsessive-compulsive disorder: a PET study of implicit sequence learning. *J Neuropsychiatr* 1997; 9: 568-573.
45. Fichtner CG, Kuhlman DT, Gruenfeld, MJ, Hughes JR. Decreased episodic violence and increased control of dissociation in a carbamazepine-treated case of multiple personality. *Biol Psychiatry* 1990; 27: 1045-1052.
46. McEvoy LK, Smith ME, Gevins A. Test-retest reliability of cognitive EEG. *Clin Neurophysiol* 2000; 111: 457-463.
47. Fink A, Grabner RH, Neuper C, Neubauer AC. Extraversion and cortical activation during memory performance. *Int J Psychophysiol* 2004; 56: 129-141.
48. Takahashi T, Murata T, Hamada T, Omori M, Kosaka, H, Kikuchi M, et al. Changes in EEG and autonomic nervous activity during meditation and their association with personality traits. *Int J Psychophysiol* 2005; 55: 199-207.
49. William J, Gruzelier J. Differentiation of hypnosis and relaxation by analysis of narrow band theta and alpha frequencies. *Int J Clin Exp Hypn* 2001; 49: 185-206.
50. Crawford H, Clarke S, Kitner-Triole M. Self-generated happy and sad emotions in low and highly hypnotizable persons during waking and hypnosis; laterality and regional EEG activity differences. *Int J Psychophysiol* 1996; 24: 239-266.
51. Oohashi T, Kawai N, Honda M, Nakamura S, Morimoto M, Nishina E, Maekawa T. Electroencephalographic measurement of possession trance in the field. *Clin Neurophysiol* 2002; 113: 435-445.
52. Ford JM, Mathalon DH. Electrophysiological evidence of corollary discharge dysfunction in schizophrenia during talking and thinking. *J Psychiatr Res* 2004; 38: 37-46.
53. Marks DF, Isaac AR. Topographical distribution of EEG activity accompanying visual and motor imagery in vivid and non-vivid imagers. *Br J Psychol* 1995; 86: 271-282.