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Resting EEG deficits in accused murderers with schizophrenia

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1. Introduction

Efforts to advance the understanding of the empirically established schizophrenia/violence relationship have been impeded by researchers hesitant to move beyond its reification (Raine, 2006) and by a public which misperceives its true scope; which has unfortunately contributed to a negative stigmatization of all individuals with schizophrenia—the majority of which who are not violent (Joyal et al., 2004; Swanson et al., 2006)—rather than a focus upon the minority that are. Increasing evidence indicates that violent persons with schizophrenia may represent a biologically-based schizophrenia subtype, with distinct electrodermal (Schug et al., 2007) and neuropsychological deficits (Schug and Raine, 2009). Electroencephalographic (EEG) investigations—more direct indicators of cortical functioning—have revealed abnormalities in individuals characterized by schizophrenia and violence separately; though to date comparison studies designed to examine resting EEG deficits unique to violent schizophrenia have not been undertaken, particularly in schizophrenic individuals characterized by the most extreme form of violence—homicide.

The classic finding in the literature on resting EEG in schizophrenia is increased slow-wave activity in schizophrenic persons (i.e., 1–8 Hz, or delta and theta; Clementz et al., 1994; Sponheim et al., 1994), often accompanied by decreased alpha activity (Iacono, 1982; Itil et al., 1972; Itil et al., 1974). This cortical “slowing” is believed by some authors to represent trait-related features of schizophrenia (due to its demonstrated presence in medicated and unmedicated patients, longitudinal stability, and observed frequency composition similarities among first-episode and chronic patients; Clementz et al., 1994; Miller, 1989;

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Sponheim et al., 1994) and by others to represent the therapeutic effects of antipsychotic medications (Centorrino et al., 2002; Freudenreich et al., 1997; Pillay et al., 1996; Shagass, 1991; Stevens, 1995; Wichniak et al., 2006). While diffuse and more-localized slow band activity increases have been identified in the resting EEGs of schizophrenic patients (i.e., over the whole cortex, anterior cingulate gyrus, temporal and posterior cortical regions; Mientus et al., 2002; Miyauchi et al., 1990), excess slow band activity appears to be more prominent in the frontal areas (Miller, 1989). It has been proposed that slowing of EEG activity represents greater brain activation which is consistent with findings of increased slow band activity during hallucinations in schizophrenia and heightened creativity in normal controls (Clementz et al., 1994; Miller, 1989), though another interpretation is that frontally pronounced EEG slowing in schizophrenia represents hypofrontality—one of the most prominent and consistent findings in neuroimaging studies of schizophrenia (Basile et al., 2004; Mientus et al., 2002). EEG slowing correlates with reduced cerebral blood flow and glucose utilization in schizophrenia patients (Wuebben and Winterer, 2001), and hypofrontality may be of particular interest in the study of homicidally violent schizophrenia, given the implication of impaired frontal executive processes in both schizophrenia and antisocial and violent behavior separately (Barkataki et al., 2005; Morgan and Lilienfeld, 2000) and PET findings of reduced prefrontal glucose metabolism in murderers (Raine et al., 1997).

Literally hundreds of EEG studies have assessed populations of criminals, delinquents, and violent offenders (Raine, 1993), which generally describe EEG abnormalities in frontal and temporal regions (Gatzke-Kopp et al., 2001). Early qualitative EEG studies of homicidal violence reported prevalence, types, and locations of EEG abnormalities among samples of murderers (often described as diffuse or focalized in temporal regions; Hill and Pond, 1952; Mundy-Castle, 1953; Stafford-Clark and Taylor, 1949), though these studies were methodologically limited and without control groups (Langevin et al., 1987). In subsequent decades, descriptive and comparison studies following in this qualitative tradition (Blake et al., 1995; Driver et al., 1974; Green et al., 2001; Kahn, 1971; Langevin et al., 1987; Langevin et al., 1982; Okasha et al., 1975; Sakuta and Fukushima, 1998; Sayed et al., 1969; Sendi and Blomgren, 1975; Winkler and Kove, 1961) and incidental qualitative EEG data from descriptive and case studies involving murderers (Chesterman et al., 1994; Lewis et al., 1988; Lewis et al., 1985; Mouridsen and Tolstrup, 1988; Pontius and LeMay, 2003; Szymusik, 1971) have continued to indicate—in varying levels of detail—similar EEG abnormalities including frontal and temporal anomalies in some cases; though EEG methodology—particularly resting EEG—is not always specified (making cross-study comparisons difficult). More-recently, computerized quantitative studies of resting EEG in murderers have reported right hemispheric, frontal, and temporal abnormalities (without controls; Evans and Park, 1997), increased temporal but not frontal slow wave and beta1 activity (Gatzke-Kopp et al., 2001), and overall reduced (most prominently occipital and temporal) alpha power, bilaterally increased occipital delta and theta power, and increased left temporal delta power in the resting EEGs of murderers compared to controls (Lindberg et al., 2005). Additionally, temporal EEG slowing is in line with imaging findings of reduced temporal functioning in violent individuals with and without schizophrenia (Yang et al., 2008), and neuropsychological findings of reduced performance on indices of temporal lobe functioning in antisocial schizophrenic persons compared to their non-schizophrenic and non-antisocial counterparts (Schug and Raine, 2009). In sum, resting EEG findings from schizophrenia research, taken together with those from homicide studies, suggest frontal and temporal regions as promising for examining markers for cortical dysfunction (i.e., EEG slowing) which may characterize homicidally violent schizophrenic persons.

While qualitative and quantitative EEG studies alike have included schizophrenic individuals within homicide samples (Chesterman et al., 1994; Gatzke-Kopp et al., 2001;

Mundy-Castle, 1955; Okasha et al., 1971; Sayed et al., 1969; Stafford-Clark and Taylor, 1949), determining resting EEG characteristics specific to this group of schizophrenic murderers becomes problematic because: (1) non-violent individuals with schizophrenia are not included for comparison in these studies, and (2) either the exact number of schizophrenic individuals in these samples is not reported or schizophrenia/non-schizophrenia statistical comparisons of resting EEG are not conducted. The present study sought to address both of these methodological shortcomings, by simultaneously testing the hypotheses that murderers with schizophrenia would be characterized by more-pronounced resting EEG slowing in frontal and temporal regions relative to both non-violent schizophrenic patients (to ascertain why only *some* individuals with schizophrenia become violent) and murderers without mental illness (to determine if the homicidal violence observed in schizophrenic individuals is in some way etiologically distinct from that of homicidal persons in general; see Schug and Raine, 2009). Additionally, we sought to determine if any group differences in resting EEG could represent characteristics of a distinct homicidal schizophrenic subgroup rather than the influences of general mental illness alone.

2. Methods

2.1. Participants

Participants (162 men and women in Nanjing, China) were classified into five diagnostic groups: individuals accused of homicide without psychiatric illness ($n = 31$); individuals accused of homicide who also suffer from schizophrenia or other psychoses ($n = 32$); individuals accused of homicide who suffer from non-psychotic psychiatric disorders (i.e., homicide psychiatric controls [HPCs]), including mental disorders due to brain damage or vascular disease, epilepsy, hallucinosis due to alcohol use, alcohol dependence, depression, mania, acute stress disorder, and twilight—a state of altered consciousness ($n = 14$); non-violent patients with schizophrenia ($n = 33$); and normal controls (i.e., without psychiatric illness; $n = 47$). Participants were recruited from the Nanjing Brain Hospital at Nanjing Medical University—accused murderers were detainees who were undergoing forensic psychiatric evaluation,* non-violent individuals with schizophrenia were hospital inpatients, and normal controls were employees and community members, screened for history of mental illness. Groups did not differ significantly in age, gender composition, or Hollingshead's Two Factor Index of Social Position (Miller, 1983); though all schizophrenia and homicide groups demonstrated significantly reduced Full Scale IQ (assessed via the WAIS-RC; Gong, 1992) in comparison to normal controls (see Table 1).

Written informed consent was obtained from all participants according to specifications outlined in the Belmont Report. The study and all of its procedures were approved by the Institutional Review Board at the University of Southern California.

2.2. Diagnostic Measures

Two psychiatrists (including C.H., who supervised all diagnostic procedures) confirmed by consensus the lifetime presence of Axis I and Axis II psychopathology—psychotic disorders, mood disorders, personality disorders (paranoid, schizoid, dissocial, other), substance use disorders, and epilepsy—obtained via semi-structured diagnostic interviews based upon the Chinese Classification of Mental Disorders Version 3 (CCMD-3; Chinese Society of Psychiatry, 2001) and fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychological Association, 1994). Interviews were conducted independently by 1-4 psychiatrists (for diagnostic reliability purposes), and inter-rater reliability was established among principal raters by selecting cases for whom all had made independent diagnostic ratings ($n = 51$) and calculating recommended Cohen's kappa

statistics (Dewey, 1983; Sim and Wright, 2005) between each. Using diagnosis of schizophrenia (any type) as an outcome variable, Cohen's kappa statistics were high, ranging from 0.82 – 0.94 (*SEs* 0.06 – 0.10, all *ps* < .001; Landis and Koch, 1977). Additionally, data related to current antipsychotic medication use and history of hospitalization for head injury were also collected during diagnostic interviews. Finally, forensic data (i.e., history of assaultive behavior, previous criminal records) were collected from all murderer groups.

Given the comorbidity of antisocial personality disorder (ASPD) and substance use disorders (SUDs) reported to characterize antisocial individuals with schizophrenia (Hodgins, 2004; Moran and Hodgins, 2004; Mueser et al., 1997), and the effects these conditions separately may have upon EEG functioning (Costa and Bauer, 1997; Lijffijt et al., 2009; Lindberg et al., 2005), it was important in the present study to assess for comorbidities with these illnesses. Overall rates for antisocial personality and substance use disorders in this sample, however, were very low. Only one participant (0.6% of the entire sample) was diagnosed with dissociative personality disorder (the CCMD-3 analogue to the DSM-IV's ASPD), and two participants (1.2% of the total sample) were diagnosed with mental disorders due to substances. As such, any potential confounding effects of comorbid ASPD or SUDs upon EEG functioning were considered negligible. Additionally, neither schizophrenia group contained individuals with comorbid mood or substance use disorders, or personality disorders of any type.

2.3. EEG Recording Procedures

Participants were tested in a temperature-controlled, light- and sound-attenuated room. Participants were instructed to keep their eyes closed and to minimize all hand and other body movements for the duration of the resting EEG recording. EEG was recorded from 16 scalp sites according to the international 10/20 system: Fz, Cz, Pz, Oz, Fp1, Fp2, F3, F4, F7, F8, T3, T4, P3, P4, O1, and O2. All electrodes (silver-silver chloride) were referred to linked ear lobes, and a ground electrode was attached to the center of the forehead. Resting EEG was recorded using a 16-channel electroencephalograph (Nicolet Biomedical Bravo) with bandpass (0.1 – 70 Hz) and notch (50 Hz) filters, and a sensitivity of 10 microvolts. Impedance was kept below 10 k Ω . Physiological signals were acquired using a 16-bit 16 channel analog-to-digital converter with 1 KHz maximum sampling rate, using a sampling frequency of 256 Hz. Horizontal and vertical EOG data were also simultaneously collected. Resting EEG was recorded during a three minute rest period at the beginning of a one-hour psychophysiological testing session.

2.3.1. EEG data reduction

Spectral power was calculated with Persyst FFT computing software (using a Hanning FFT window of one-second duration, and non-overlapping samples) from the following frequency bands: delta (0.01- 4.00 Hz), theta (4.01 – 8.00 Hz), alpha1 (8.01 – 10.50 Hz), alpha2 (10.51 – 13.00 Hz), beta1 (13.01 – 20.00 Hz) beta2 (20.01 – 30.00 Hz), and beta3 (30.01 – 50.00 Hz). Spectral power was calculated for each frequency band for each electrode site. From the original three-minute rest period EEG recording, one sixty-second window—free of electrooculographic artifacts (i.e., eye blinks) and movement—was selected for quantitative analysis. EEG window selections were made by visual analysis (neurophysiologic evaluation, double blind with respect to diagnosis) and classification. Data from left hemispheric sites (Fp1, F3, F7, P3, T3, O1) were averaged into one left hemispheric measure. Data from right hemispheric sites (Fp2, F4, F8, P4, T4, O2) were averaged into one right hemisphere measure. Data from medial prefrontal (Fp1 and Fp2), dorsomedial prefrontal (F3 and F4), dorsolateral prefrontal (F7 and F8), temporal (T3 and

T₄), parietal (P₃ and P₄), and occipital (O₁ and O₂) sites were averaged into individual lobe measures.

2.4. Statistical Analyses

To avoid Type I error in conducting multiple analyses across individual electrode sites and power bands, a series of repeated-measures MANOVAs with one between-subject factor (GROUP with five levels) and two within-subject factors (HEMISPHERE with two levels and LOBE with six levels) was employed to first assess for group-by-hemisphere, group-by-lobe, and group-by-hemisphere-by-lobe interactions in each band. Significance levels were adjusted using the more-conservative Greenhouse-Geisser correction if the assumption of sphericity was violated. If significant interactions were detected, secondary one-way ANOVAs along with post-hoc multiple comparisons (Bonferroni) were subsequently conducted on individual hemisphere, lobe, or hemisphere-lobe site groupings. Subsequent analyses were conducted in individual power bands at hemisphere, lobe, or hemisphere-lobe sites where significant group differences were revealed to assess for sample-wide EEG effects of current antipsychotic medication use and head injury history (see Table 1; no analyses for substance use disorder effects were conducted as participants were predominantly free of substance use disorders).

In cases where serious violations of the assumptions underlying traditional statistical techniques were detected, additional modern methods were used—including trimming and bootstrapping techniques. Trimming 20% of the data can substantially reduce poor power due to outliers, skewness, and variance (yet good power is still achieved under standard assumptions; Wilcox, 2005b), while bootstrapping techniques (Wilcox, 2003) have proven effective in analyses involving smaller group sizes. Bootstrap methods estimate appropriate critical values using the available data, rather than assuming normality to determine appropriate critical values (Efron and Tibshirani, 1993; Davison and Hinkley, 1997). In the present study, boxplots were generated to assess for violations of standard assumptions (i.e., significant outliers and/or skewness) in all analyses. For simplification purposes, the reporting here of results from modern methods indicates that boxplots had revealed the presence of significant outliers and/or skewness, and that these methods were subsequently employed to augment conventional analyses (Schug et al., 2007).

3. Results

Repeated measures MANOVAs indicated no significant interactions in the delta, alpha1, alpha2, or beta1 bands. However, repeated measures MANOVAs revealed significant group-lobe (Pillai's Trace $F(20,576) = 1.92, p = 0.010$; Greenhouse-Geisser correction $F(8.08,292.83) = 2.17, p = 0.029$) and group-hemisphere (Pillai's Trace $F(4,145) = 2.81, p = 0.028$; Greenhouse-Geisser correction $F(4,145) = 2.81, p = 0.028$) interactions in the theta band, and a significant group-hemisphere interaction (Pillai's Trace $F(4,145) = 3.99, p = 0.004$; Greenhouse-Geisser correction $F(4,377.54) = 3.99, p = 0.004$) in the beta3 band. Secondary one-way ANOVAs along with modern analyses (Benjamini-Hochberg method; Wilcox, 2005a)—conducted at individual group hemispheric and group lobe sites in these power bands where significant group differences were indicated—revealed significantly increased beta3 power in murderers with schizophrenia compared to non-violent schizophrenic patients for left hemispheric lobe sites; and significantly increased theta power in non-violent schizophrenic patients compared to other groups at all group hemispheric and group lobe sites (Figure 1 and Table 2). A series of univariate ANOVAs were also conducted while entering Full Scale IQ as a covariate. Results indicated group differences in theta and beta3 bands remained significant for all hemispheric and lobe sites except for temporal lobe sites in the theta band ($F(4, 144) = 1.44, p = .224$). However, when

the theta temporal lobe site analysis was repeated with two significant extreme outliers removed, results became significant ($F(4, 142) = 6.79, p < .001$).

Subsequent independent-samples T tests and modern methods (i.e., a percentile bootstrap method comparing modified M-estimators; Wilcox, 2005a) indicated significantly increased theta power in antipsychotic-medicated compared to antipsychotic-free participants only for parietal and occipital lobe sites (though group differences approached significance for left hemispheric, right hemispheric, and temporal lobe sites); however, significant differences were not observed for left hemispheric sites in the beta3 band. Similar analyses comparing participants with and without a history of hospitalization for head injury revealed no significant group differences in theta or beta3 power for the aforementioned sites.

Additionally, to examine the strengths of the schizophrenia and homicide effects separately, and any two-way interactions, a series of univariate MANOVAs was conducted using two group dimensions: SCHIZOPHRENIA (yes/no) and HOMICIDE (yes/no). Homicide psychiatric controls were excluded from these analyses to control for potentially confounding effects of mental illnesses other than schizophrenia. Results are listed in Tables 3 and 4, and indicated significant main effects for Schizophrenia at all hemispheric and lobe sites in the theta band, with a significant Schizophrenia-by-Homicide interaction at medial prefrontal sites. In contrast, in the beta3 band, significant effects for Homicide and a significant Schizophrenia-by-Homicide interaction were observed for left hemispheric sites. Results are depicted graphically (using analyses for left hemispheric sites as examples) for theta band in Figure 2 and beta3 in Figure 3.

Furthermore, Chi Square analyses were employed to assess for group differences in history of assaultive behavior and previous criminal records among all murderer groups. Finally, a series of Chi Square analyses was conducted on individual schizophrenia symptoms to assess for group differences in symptom presentations between murderers with schizophrenia and non-violent schizophrenic patients. An independent-samples T-test was also conducted to examine potential differences in the total number of schizophrenia symptoms among these two groups. Results are listed in Table 5.

4. Discussion

The present study sought to determine if homicidally violent individuals with schizophrenia were characterized by particular deficits in EEG functioning (i.e., more-pronounced slow-wave increases in frontal and temporal regions) relative to non-violent schizophrenic patients, murderers without schizophrenia, and murderers with psychiatric conditions other than schizophrenia. Results, however, indicated that murderers with schizophrenia were instead characterized by significantly increased left hemispheric beta3 power relative to non-violent schizophrenic patients; and while non-violent schizophrenic patients were characterized by widespread EEG deficits (i.e., significantly increased theta power at all hemispheric and lobe sites) compared to other groups, murderers with schizophrenia were not.

Theta band findings are consistent with the EEG slowing and cortical hypoactivity commonly reported in schizophrenic patients (see above). Secondary analyses here indicated antipsychotic medication effects upon theta power which appeared more localized to parietal and occipital lobe site regions and not significant at prefrontal sites. Ultimately, medication effects cannot be ruled out in any region, though theta findings suggest a violent form of schizophrenia uncharacterized by the hypofrontality seen in nonviolent schizophrenic patients—perhaps indicating differential medication efficacy or dissimilar biological underpinnings of cortical arousal among these groups. Both explanations could suggest a

functional strength in violent compared to nonviolent individuals with schizophrenia, possibly speaking to intact frontal-related executive abilities which could facilitate—or at least allow for—homicidal behavior despite cognitive deficits in other areas.

Beta3 findings in murderers with schizophrenia here are in line with previous reports of asymmetrical left hemispheric fast beta activity increases in individuals with schizophrenia (Shagass, 1991), and may suggest left hemispheric dysfunction in the form of overarousal (Saletu et al., 1986; see also Raine and Manders, 1988, and Raine et al., 2002). Reasoning about context-independent situations appears to be mediated by left hemispheric regions, whereas reasoning influenced by information based on previous beliefs, values, or goals is mediated by right hemispheric and bilateral ventromedial regions (Wharton and Grafman, 1998). Left hemispheric overarousal in murderers with schizophrenia relative to non-violent schizophrenia patients may reflect over-activity in left hemispheric brain centers responsible for logical or sequential reasoning, rational or analytical thought (Wharton and Grafman, 1998), or the ability to be objective or look at parts rather than wholes (e.g., analytic, bit-by-bit processing; Delis et al., 1988). Overactive left in the absence of intact right hemispheric reasoning processes (undermined perhaps by schizophrenia-related deficits in context-relevant or even “emotional” types of memory) could explain why an individual with schizophrenia would focus upon and over-process context-independent environmental cues (at the expense of understanding important overarching contextual factors) and perceive them as confusing or even threatening while failing to call upon important right hemispheric belief, value, and goal-oriented processing resources; or perhaps over-interpret, analyze, or rationalize available information into perceptions which are congruent with a delusional framework. Either scenario could create a greater predisposition toward violence in an individual with schizophrenia relative to another with normative or even deficient left-hemispheric reasoning abilities. Ultimately, a left-hemispheric over-processing hypothesis specific to violent schizophrenia would require further testing, as EEG evidence for such a hypothesis here is suggestive but limited.

4.1. Murderers with Schizophrenia: A Biologically Distinct Subgroup?

While findings did suggest factors which might explain why only some individuals with schizophrenia become violent, evidence for a biologically distinct violent schizophrenia subtype was mixed. EEG characteristics distinguished murderers with schizophrenia only from nonviolent schizophrenic patients, and could have been attributable to general violence or additive schizophrenia and homicide effects. Several key pieces of evidence were considered, however, which may not support these alternative explanations. First and foremost, while theta band findings appeared to be predominantly driven by schizophrenia and beta3 findings by homicide, the significant interaction between schizophrenia and homicide on beta3 power for left hemispheric sites indicate these effects were not purely additive. For murderers without psychiatric illness, theta power was slightly increased though nonetheless similar to normal controls—even comparatively *reduced* in some regions; and slightly increased beta3 power in this group was much closer to normal controls (those of murderers with schizophrenia were increased almost twofold) while those of nonviolent schizophrenic patients were *decreased* relative to normal controls (Figures 2 and 3). Together, this pattern of results would speak to some degree against general violence effects and to a greater degree against additive effects of both conditions upon EEG functioning.

Second, we considered that individuals with schizophrenia who murder—compared to those that do not—may reflect a more-severe form of schizophrenia (in terms of symptomatology) rather than a distinct subtype. Results, in fact, did reveal significant differences in symptom presentations between nonviolent schizophrenic patients (characterized more so by repeated mood-incongruent auditory hallucinations, control/influence/passivity delusions and thought

broadcasting, incongruous emotion or apathy, and catatonic behavior) and murderers with schizophrenia (characterized instead by increased disorganized speech/thought disorder symptoms and perceptual, mood, and bizarre delusions; Table 5). Additionally—and perhaps more compelling—murderers with schizophrenia demonstrated significantly *reduced* total number of schizophrenia symptoms compared to nonviolent schizophrenic patients (suggesting a *less*-severe form of the illness in the former, in terms of symptom presentation). Together, these findings may constitute further support for what may be a violent schizophrenia subtype rather than the more-extreme ranges of schizophrenia symptomatology—though future research should continue to address this very important distinction.

Third, we considered that schizophrenic murderers here may represent the extreme end of the violence spectrum, more-likely characterized by repetitive rather than one-time violence. A significant literature relating to the anomalous EEG functioning of repetitively-violent (including—though not necessarily—schizophrenic) individuals has been developed (Convit et al., 1991; Raine, 1993; Wong et al., 1997), with results generally indicating anterior EEG slowing and temporal abnormalities in these individuals. On balance, other forensic inpatient samples have indicated that violent psychotic offenders are often first-time offenders who perpetrate severe physical assaults against intimates (Nijman et al., 2003). In the present sample, however, the majority of murderers overall were not characterized by repetitive violence or criminal history. Chi Square analyses revealed that groups did not differ significantly in proportions of these two forensic variables, though schizophrenic murderers were characterized by increased rates of repetitive violence history which approached significance compared to HPCs when analyzed separately, $\chi^2(1) = 3.56, p = 0.059$. In the end, while results do not suggest that schizophrenic murderers here represent the extreme ends of schizophrenia and violence spectrums (or necessarily first-time assaultive offenders, for that matter), this line of inquiry should continue to be considered in future attempts to identify a biologically distinct schizophrenia subtype.

Finally, failure to find significant EEG group differences among murderers with schizophrenia and HPCs could suggest effects more attributable to general mental illness rather than a violent schizophrenia subtype, though this may be less likely, given—for example—the differential EEG complexities and disorganization and functional MRI hemispheric activation patterns reported among patients with schizophrenia and those with depression (Flor-Henry, 1984; Li et al., 2008). Additionally, failure to find HPC group differences may simply reflect aberrant HPC data distributions due to diagnostic heterogeneity (perhaps compromising group validity), or the conservative Type I error correction required in five-group analyses. Ultimately, general psychiatric factors should be among methodological considerations in future examinations of violent schizophrenia, and results here—regardless of HPC findings—nonetheless speak to what may cause only *some* individuals with schizophrenia to become violent.

4.2. Limitations and Strengths of the Present Study

Key methodological considerations in the present study are the potential influences of antipsychotic medications and/or head injury history upon EEG functioning; though these were at least partially addressed and qualified with the use of secondary analyses (with the caveat that our high-threshold operationalization of head injury history—hospitalization—may not encompass all instances of head injuries). Additionally, gamma band activity (largely overlapping in frequencies and bandwidth with beta3 here; Basar-Eroglu et al., 2007; Hong et al., 2004) is thought to represent a stable personal trait variable (Lenz et al., 2008), which may speak more to a fixed beta3 characteristic of murderers with schizophrenia here rather than an impermanent medication-induced quality. Future studies incorporating both medicated and unmedicated groups (analyzed separately) could help

disentangle these medication influences, and further the understanding of antipsychotic medication effects upon EEG functioning in individuals with schizophrenia who do and do not become violent.

Generalizability of the present study's findings may be limited given the very low incidence of substance use disorders in this Chinese sample (i.e., 1.2%). This finding, however, is consistent with the literature. Zhang and Snowden (1999) also reported significantly lower lifetime prevalence of drug/alcohol use disorders in Asians (2.3%/7.1%) compared to Whites (6.0%/12.7%) in U.S. samples; and prevalence rates of alcohol use disorders (5.8%), sedative/hypnotic/anxiolytic drug use disorders (< 1%), cocaine use disorders (< 1%) and other substance use disorders (< 1%) in an epidemiological study of four Chinese provinces during 2001-2005 (Phillips et al., 2009) appear comparatively low (e.g., relative to lifetime prevalence rates of 15% for alcohol dependence reported in other studies; APA, 2000). For alcohol use disorders, lower prevalence rates among Asian cultures may even reflect a deficiency (in perhaps half of Japanese, Chinese, and Korean individuals) of an enzyme—aldehyde dehydrogenase—that eliminates low levels of acetaldehyde, the first breakdown product of alcohol (which may lead to negative physical symptoms accompanied by alcohol consumption; APA, 2000).

Other methodological limitations include the poor spatial resolution of EEG relative to brain imaging techniques (Raine, 1993), and smaller diagnostic group sizes which may have affected affect statistical power and generalizability—though the latter was at least partially addressed via the use of modern statistical methods. Also, interpretation of group differences between murderers with schizophrenia and non-violent schizophrenic patients must be made with caution, given the potential differences in current and history of psychiatric symptomatology which could not be addressed here. Additionally, conviction data were not available for this sample, and the use of accused (i.e., charged) as opposed to convicted murderers could be considered a methodological limitation to the present study, given—particularly from a purely legal standpoint—the operationalization of a murderer may not be valid until the completion of the adjudication process. On balance, other empirical investigations of murderers (Firestone et al., 2000)—including classic (Hill & Pond, 1952; Stafford-Clarke & Taylor, 1949) and more-recent EEG studies (Lindberg et al., 2005)—have utilized samples of charged as opposed to convicted offenders; and samples which included offenders who were not yet convicted but being assessed for competency to stand trial have been used in imaging studies (e.g., Raine et al., 1997). Furthermore, given China's extremely high conviction rate in criminal trials (i.e., approximately 98%; Guo, 2010), it is likely that nearly all of the offenders in this Chinese sample would have been classified as murderers even according to this more-conservative operationalization (i.e., the legal standard of being convicted), and were thus viewed as appropriate for a study of homicide. Nonetheless, generalizations of the present study's results should be made cautiously given this important point.

These limitations, however, are offset at least partially by several methodological strengths—including sound diagnostic procedures using a culturally-relevant classification system (i.e., the CCMD-3), the use of modern statistical methods to deal with aberrant data distributions, and a design incorporating five diagnostic groups allowing for comparisons of murderers and non-murderers, with and without schizophrenia, and for comparisons with healthy controls to allow for the assessment of comparative group deficits within a normative functional context. Additionally, the inclusion of HPCs allows for interpretations of differences among murderers with and without schizophrenia beyond those ascribable to mental illness in general. Together, these elements serve to strengthen the present study's methodology, relevance, and generalizability of findings in other ways, which compensate to some extent for perceived study weaknesses.

4.3. Implications

The identification of a distinct homicidal schizophrenia subtype may have important implications in research, treatment, forensic, and social realms. First, research endeavors of either schizophrenia or violence separately must take into account the potential presence and confounding influence of the other condition—failure to do so may obscure or even mask findings and lead to spuriously heterogeneous results across studies. For example, disparate results achieved in past studies of electrodermal functioning in schizophrenia (Schug et al., 2007) could be clarified by considering violence as a moderator. Second, a greater understanding of the biological nature of violent schizophrenia might lead to the development of more effective pharmacological approaches specifically tailored for this group (particularly relevant given limited evidence here for what may be differential efficacy of traditional antipsychotics among violent and nonviolent persons with schizophrenia)—perhaps targeting symptoms more-related to left hemispheric over-processing. Additionally, a greater understanding of the etiological processes at work in violent schizophrenia may lead to the identification of the biological precursors of this condition—information which could be incorporated into intervention programs for children and adolescents designed to reduce violent behavior before the onset of schizophrenia (Hodgins, 2004). Finally, in the social realm, identifying a separate group of individuals with schizophrenia who are prone to homicidal violence may serve to reduce the stigma attached to schizophrenia in general, which is significantly amplified by sensational (albeit rare) cases of violence perpetrated by schizophrenia individuals. This stigma—which undermines efforts to obtain important community resources for schizophrenia individuals such as treatment and housing (Hodgins, 2004)—arguably constitutes a grave social injustice against individuals with schizophrenia who are not violent, and does little to advance the cause of those who are.

In conclusion, while limited EEG evidence here is mixed for a biologically distinct subtype of violent schizophrenia, results do suggest biological factors which distinguish violent individuals with schizophrenia from their counterparts who are not violent. Future EEG research should attempt to replicate these findings and continue to address whether violent individuals with schizophrenia in fact represent a unique schizophrenia subtype, the manifestation of a particular grouping of schizophrenia symptomatology, or perhaps a combination of inter-laced risk factors which comprise a biopsychosocial interactional trajectory toward violence specific to schizophrenia. Either explanation would have important implications in research, treatment, and social perception.

*In China, only the police, the prosecution or the court can decide to conduct a mental examination. Neither the defendant nor his/her lawyer can initiate an evaluation; they can only apply for supplementary evaluations or re-evaluations after results have been produced by an officially-initiated examination. Under Chinese law, convicted murderers are sentenced to death, though special protections are provided for those diagnosed with mental illness (Guo, 2010).

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Figure 1.
 Resting EEG Spectral Power Across Frequencies at Hemispheric and Lobe Measure
 Electrode Sites with Significant Group Differences
 Note. Asterisks (*) denote significant group differences. See Table 2 for specific group
 differences and *p* values.

Figure 2.
Estimated Marginal Means of Left Hemispheric Sites—Theta

Figure 3.
Estimated Marginal Means of Left Hemispheric Sites—Beta3

Table 1

Diagnostic Group Demographic Characteristics

	Diagnostic groups					ANOVA or Chi Square results		
	N	S	H	SH	HPC	Test statistic	p	Significant group differences (p < .05)
Age	35.81 (12.40)	33.67 (10.05)	31.28 (12.50)	36.58 (11.36)	33.86 (12.01)	$F(4,145) = 0.95$	0.438	--
Gender						$\chi^2(4) = 2.47$	0.650	--
Male	35 (74.5%)	27 (81.8%)	22 (71.0%)	27 (84.4%)	10 (71.4%)			
Female	12 (25.5%)	6 (18.2%)	9 (29.0%)	5 (15.6%)	4 (28.6%)			
Hollingshead's Two Factor Index of Social Position (SES)	3.72 (1.22)	3.47 (1.31)	3.95 (1.36)	4.15 (0.97)	4.36 (0.84)	$F(4,145) = 2.03$	0.094	--
Full-Scale IQ	102.91 (14.25)	87.36 (15.08)	83.29 (16.69)	87.46 (12.91)	86.14 (13.68)	$F(4,145) = 10.94$	< 0.001	N > S, H, SH, HPC
Current antipsychotic medication use	0 (0.0%)	33 (100.0%)	1 (3.4%)	8 (29.6%)	3 (21.4%)	$\chi^2(4) = 105.70$	< 0.001	S > N, H, SH, HPC; SH > N, H; HPC > N, H *
History of hospitalization for head injury	5 (10.9%)	2 (6.1%)	7 (29.2%)	2 (7.4%)	3 (21.4%)	$\chi^2(4) = 8.65$	0.071	H > N *, S, SH
History of assaultive behavior	--	--	9 (33.3%)	14 (48.3%)	2 (16.7%)	$\chi^2(2) = 3.88$	0.144	SH > HPC *
Previous criminal record	--	--	10 (43.5%)	7 (26.9%)	4 (30.8%)	$\chi^2(2) = 1.56$	0.457	--

Note. N = Normal controls. S = Schizophrenia. H = Homicide. SH = Schizophrenia-Homicide. HPC = Homicide psychiatric controls.

^aSignificant group differences using Benjamini-Hochberg method (Wilcox, 2005) only.

* Trend toward significance.

Table 2
Resting EEG Spectral Power at Hemispheric and Lobe Measure Electrode Sites with Significant Group Differences

Power band and sites	Diagnostic groups (Spectral power measured in microvolts-squared/Hz)					ANOVA results	
	N	S	H	SH	HPC	F(4,145)	p
Theta left hemispheric	4.68 (2.49)	9.54 (6.97)	4.81 (2.44)	7.52 (10.15)	5.06 (1.16)	4.26	0.003
Theta right hemispheric	4.74 (2.58)	10.69 (9.06)	4.83 (2.52)	7.63 (9.97)	5.16 (0.92)	5.16	0.001
Theta medial prefrontal	5.43 (2.68)	13.18 (11.98)	6.20 (2.91)	8.54 (9.94)	6.58 (1.58)	5.91	< 0.001
Theta dorsomedial prefrontal	6.12 (3.26)	13.42 (11.44)	6.43 (3.54)	9.30 (10.78)	6.69 (1.60)	5.33	< 0.001
Theta dorsolateral prefrontal	4.18 (2.24)	8.04 (6.51)	4.45 (2.10)	6.05 (9.13)	4.67 (1.14)	2.95	0.022
Theta temporal	3.17 (2.99)	5.22 (3.82)	3.12 (1.81)	4.62 (8.34)	3.08 (0.87)	1.48	0.212
Theta parietal	5.47 (3.62)	11.29 (9.52)	5.04 (3.65)	8.48 (11.88)	5.19 (1.86)	4.16	0.003
Theta occipital	3.90 (2.22)	9.54 (11.13)	3.67 (2.94)	8.46 (15.22)	4.45 (2.51)	3.19	0.015
Beta3 left hemispheric	2.69 (3.17)	1.41 (0.95)	3.07 (3.65)	5.28 (6.83)	5.00 (9.93)	2.98	0.021

Note. N = Normal controls. S = Schizophrenia. H = Homicide. SH = Schizophrenia-Homicide. HPC = Homicide psychiatric controls.

^aSignificant group differences using Benjamini-Hochberg method (Wilcox, 2005) only.

Table 3

Resting EEG Spectral Power at Hemispheric and Lobe Measure Electrode Sites with Significant Group Differences: Schizophrenia (yes/no) and Homicide (yes/no)

		Spectral power (microvolts-squared/Hz)		
Power band and sites		<i>M</i>	<i>SD</i>	<i>n</i>
Theta left hemispheric				
Schizophrenia	Homicide			
No	No	4.68	2.49	46
	Yes	4.81	2.44	29
Yes	No	9.54	6.97	31
	Yes	7.52	10.15	30
Theta right hemispheric				
Schizophrenia	Homicide			
No	No	4.74	2.58	46
	Yes	4.83	2.52	29
Yes	No	10.69	9.06	31
	Yes	11.63	9.97	30
Theta medial prefrontal				
Schizophrenia	Homicide			
No	No	5.43	2.68	46
	Yes	6.20	2.91	29
Yes	No	13.18	11.98	31
	Yes	8.54	9.94	30
Theta dorsomedial prefrontal				
Schizophrenia	Homicide			
No	No	6.12	3.26	46
	Yes	6.43	3.54	29
Yes	No	13.42	11.44	31
	Yes	9.30	10.78	30
Theta dorsolateral prefrontal				
Schizophrenia	Homicide			
No	No	4.18	2.24	46
	Yes	4.45	2.10	29
Yes	No	8.04	6.51	31
	Yes	6.05	9.13	30
Theta temporal				
Schizophrenia	Homicide			
No	No	3.17	2.99	46
	Yes	3.12	1.81	29
Yes	No	5.22	3.82	31
	Yes	4.62	8.34	30
Theta parietal				

		Spectral power (microvolts-squared/Hz)		
Power band and sites		<i>M</i>	<i>SD</i>	<i>n</i>
Schizophrenia	Homicide			
	No	5.47	3.62	46
	Yes	5.04	3.65	29
Theta occipital	No	11.29	9.52	31
	Yes	8.48	11.88	30
Schizophrenia	Homicide			
	No	3.90	2.22	46
	Yes	3.67	2.94	29
Beta3 left hemispheric	No	9.54	11.13	31
	Yes	8.46	15.22	30
Schizophrenia	Homicide			
	No	2.69	3.17	46
	Yes	3.07	3.65	29
Beta3 left hemispheric	No	1.41	0.95	31
	Yes	5.28	6.83	30

Note. HPCs excluded from analyses.

Table 4

Resting EEG Spectral Power at Hemispheric and Lobe Measure Electrode Sites with Significant Group Differences: Schizophrenia (yes/no) and Homicide (yes/no) MANOVA Results

Power band, site, and variable	<i>df</i>	<i>F</i>	<i>p</i>
Theta left hemispheric			
Schizophrenia	1,132	12.69	0.001
Homicide	1,132	0.80	0.372
Schizophrenia * Homicide	1,132	1.02	0.314
Theta right hemispheric			
Schizophrenia	1,132	14.25	<0.001
Homicide	1,132	1.63	0.203
Schizophrenia * Homicide	1,132	1.86	0.175
Theta medial prefrontal			
Schizophrenia	1,132	14.27	<0.001
Homicide	1,132	2.10	0.150
Schizophrenia * Homicide	1,132	4.12	0.044
Theta dorsomedial prefrontal			
Schizophrenia	1,132	13.80	<0.001
Homicide	1,132	1.94	0.166
Schizophrenia * Homicide	1,132	2.62	0.108
Theta dorsolateral prefrontal			
Schizophrenia	1,132	7.99	0.005
Homicide	1,132	0.79	0.376
Schizophrenia * Homicide	1,132	1.37	0.243
Theta temporal			
Schizophrenia	1,132	4.62	0.033
Homicide	1,132	0.153	0.696
Schizophrenia * Homicide	1,132	0.113	0.738
Theta parietal			
Schizophrenia	1,132	11.95	0.001
Homicide	1,132	1.46	0.229
Schizophrenia * Homicide	1,132	0.80	0.374
Theta occipital			
Schizophrenia	1,132	10.83	0.001
Homicide	1,132	0.170	0.681
Schizophrenia * Homicide	1,132	0.073	0.787
Beta3 left hemispheric			
Schizophrenia	1,132	0.43	0.514
Homicide	1,132	8.85	0.003
Schizophrenia * Homicide	1,132	5.97	0.016

Note. HPCs excluded from analyses.

Table 5**Schizophrenia Group Symptomatology Comparisons**

Schizophrenia symptom	Diagnostic groups			
	S	SH	$\chi^2(1)$	<i>p</i>
Repeated auditory hallucinations that are usually not mood congruent	25 (78.1%)	13 (40.6%)	9.33	0.002
Loosening of association, derailment, incoherence in thinking or poverty of thought	5 (15.6%)	12 (37.5%)	3.93	0.048
Thought insertion or withdrawal, thought block or forced thinking	9 (28.1%)	7 (21.9%)	0.33	0.564
Delusion of control, influence or passivity, thought broadcasting	27 (84.4%)	11 (34.4%)	16.58	< 0.001
Primary delusions (delusional perception, delusional mood, other bizarre delusions)	4 (12.5%)	15 (46.9%)	9.06	0.003
Paralogic thinking, symbolic thought or neologism	18 (56.2%)	13 (40.6%)	1.56	0.211
Incongruous emotion or apathy	19 (59.4%)	11 (34.4%)	4.02	0.045
Catatonic syndrome, unusual or silly behavior	16 (50.0%)	5 (15.6%)	8.58	0.003
Avolition	1 (3.1%)	4 (12.5%)	1.95	0.162
	<i>M (SD)</i>	<i>M (SD)</i>	<i>t(58.41)</i>	<i>p</i>
Total number of symptoms	3.88 (1.10)	2.84 (1.42)	3.25	0.002

Note. S = Schizophrenia. SH = Schizophrenia-Homicide.