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Resting EEG deficits in accused murderers with schizophrenia

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ABSTRACT

Empirical evidence continues to suggest a biologically distinct violent subtype of schizophrenia. The present study examined whether murderers with schizophrenia would demonstrate resting EEG deficits distinguishing them from both non-violent schizophrenia patients and murderers without schizophrenia. Resting EEG data were collected from five diagnostic groups (normal controls, non-murderers with schizophrenia, murderers with schizophrenia, murderers without schizophrenia, and murderers with psychiatric conditions other than schizophrenia) at a brain hospital in Nanjing, China. Murderers with schizophrenia were characterized by increased left-hemispheric fast-wave EEG activity relative to non-violent schizophrenia patients, while non-violent schizophrenia patients instead demonstrated increased diffuse slow-wave activity compared to all other groups. Results are discussed within the framework of a proposed left-hemispheric over-processing hypothesis specific to violent individuals with schizophrenia, involving left hemispheric hyperarousal deficits, which may lead to a homicidally violent schizophrenia outcome.

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 that misperceives its true scope. These impediments have unfortunately contributed to a negative stigmatization of all individuals with schizophrenia – the majority of whom are not violent (Joyal et al., 2004; Swanson et al., 2006) – and a failure to focus on the minority of patients that are violent. 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 persons with schizophrenia (i.e., 1–8 Hz, or delta and theta; Clementz et al., 1994; Sponheim et al., 1994), often accompanied by decreased alpha activity (Itil et al., 1972, 1974; Iacono, 1982). 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; Miller, 1989; Clementz et al., 1994; Sponheim et al., 1994) and by others to represent the therapeutic effects of antipsychotic medications (Shagass, 1991; Stevens, 1995; Pillay et al., 1996; Freudenreich et al., 1997; Centorrino et al., 2002; Wichniak et al., 2006). While both diffuse and more localized slow band activity increases have been identified in the resting EEGs of schizophrenia patients (i.e., over the whole cortex, anterior cingulate gyrus, temporal and posterior cortical regions; Miyauchi et al., 1990; Mientus et al., 2002), 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 (Miller, 1989; Clementz et al., 1994), though another interpretation is that

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frontally pronounced EEG slowing in schizophrenia represents hypofrontality—one of the most prominent and consistent findings in neuroimaging studies of schizophrenia (Mientus et al., 2002; Basile et al., 2004). 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 (Morgan and Lilienfeld, 2000; Barkataki et al., 2005) 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; Stafford-Clarke and Taylor, 1949; Hill and Pond, 1952; Mundy-Castle, 1955), 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 (Winkler and Kove, 1961; Sayed et al., 1969; Kahn, 1971; Driver et al., 1974; Okasha et al., 1975; Sendi and Blomgren, 1975; Langevin et al., 1982, 1987; Blake et al., 1995; Sakuta and Fukushima, 1998; Green et al., 2001) and incidental qualitative EEG data from descriptive and case studies involving murderers (Szymusik, 1971; Lewis et al., 1985, 1988; Mouridsen and Tolstrup, 1988; Chesterman et al., 1994; Pontius and LeMay, 2003) 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 persons with schizophrenia compared to their non-antisocial counterparts without schizophrenia (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 persons with schizophrenia.

While qualitative and quantitative EEG studies alike have included individuals with schizophrenia within homicide samples (Stafford-Clarke and Taylor, 1949; Mundy-Castle, 1955; Sayed et al., 1969; Okasha et al., 1975; Chesterman et al., 1994; Gatzke-Kopp et al., 2001), determining resting EEG characteristics specific to this group of murderers with schizophrenia becomes problematic because (1) non-violent individuals with schizophrenia are not included for comparison in these studies, and (2) either the exact number of individuals with schizophrenia 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 the frontal and temporal regions relative to both non-violent schizophrenia patients (to ascertain why only *some* individuals with schizophrenia become violent) and murderers without mental illness (to determine if the homicidal violence observed in individuals with schizophrenia 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 schizophrenia 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, and others), 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 the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). Interviews were conducted independently by one to four 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 the 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 to 0.94 (*SE*s 0.06–0.10, all $ps<0.001$; 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 and 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