Neuropsychological Correlates of Violence in Schizophrenia

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Abstract

Thirty-one outpatient men with schizophrenia were assessed with various measures of lifelong history of physical violence as well as psychopathology, neuropsychological performance, and neurological intactness. Most of the results consisted of nonsignificant positive relationships between physical aggression and neuropsychological performance in these schizophrenia subjects. Some neuropsychological test performances did show significant positive correlations with levels of aggressivity. In contrast with previous studies that have established a relation between neuropsychological impairment (as opposed to performance) and violence in schizophrenia, subjects of the present study were high-functioning outpatients who may not have attained a level of neurological impairment inducing constant uncontrollable outbursts of irritative aggression in their daily living. The importance of defining in detail the clinical characteristics of the subjects studied and the type of violence assessed is discussed, and an ecological interpretation of these counterintuitive results is provided.

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Although most schizophrenia patients are not violent, the distribution of various psychiatric disorders in relation to violent behavior indicates that the aggressive patient is most likely to have a primary diagnosis of schizophrenia. For example, Lee et al. (1989) concluded in their study that patients with schizophrenia are more prone to engage in assaultive and destructive behavior than other psychiatric patients. Likewise, Fottrell (1980) reported that the greatest percentage of violent incidents occurring in psychiatric hospitals can be imputed to patients with schizophrenia. Similar observations have been reported by Karson and Bigelow (1987) and Noble and Rodger (1989). In a study on clinical aspects of criminality, Odejide (1981) observed that among mentally ill criminals, offenses against other persons were committed mainly by persons with schizophrenia. Henn et al. (1976) and Siomopoulos (1978) concluded that schizophrenia is the leading diagnosis for every type of criminal offense. Furthermore, the severity of schizophrenic symptoms seems to be directly related to the dangerousness of the antisocial behavior (Yesavage 1983; Link 1992). In addition to the schizophrenic disease process, and apparently in association with it, substance abuse disorders (alcohol or drug related) have been found to relate positively and significantly with violence in persons with schizophrenia (Swanson et al. 1990; Link 1992).

There seems to be an association between violence in schizophrenia and various signs of neurological dysfunction. Barber et al. (1986) studied the clinical characteristics of chronic psychiatric inpatients, 44 percent of whom had schizophrenia with a clear history of physical violence. They observed a high incidence of electroencephalogram (EEG) abnormalities and of loss of

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consciousness following head injury in their patients. Heath (1982) reported a correlation between aggressive behavior in schizophrenia and EEG abnormalities of the basal frontal regions. Heinrichs (1989) concluded that, among patients with concomitant schizophrenia and frontal cerebral damage, only the neurological factors were significantly associated with the antisocial behaviors. Lewis and colleagues (1979, 1983) found significant incidence of prepsychotic symptoms and neurological signs in cohorts of extremely violent children. In aggressive adults with schizophrenia, a significant incidence of neurological soft signs has also been reported (Quitkin et al. 1976).

The possible existence of a subgroup of violent people with schizophrenia and a specific brain dysfunction has led to a few recent neuropsychological inquiries of this hypothesis. Krakowski et al. (1989) compared schizophrenia inpatients with a high degree of violence, low violence, and no violence on various neuropsychological and neurological variables (violence was defined as the frequency of assaultive behavior committed on the psychiatric ward during a definite period of observation). Their results indicated a selective visuospatial impairment for the high-violence group. They also observed greater neurological impairment in this group, manifesting itself in the form of a higher incidence of neurological soft signs. Adams et al. (1990) categorized schizophrenia patients into neuropsychologically impaired and nonimpaired groups and observed a strong positive relation between this classification and lifelong history of antisocial behavior but not inpatient violence. However, the sample consisted of unusually violent and highly criminalized incarcerated patients. The outpatient violence manifested a marked bimodal distribution as a function of neuropsychological impairment, leaving the authors puzzled.

The present study was designed to highlight the relationship between specific testing of neuropsychological and neurological function and lifelong violence in schizophrenia. If one looks closely at the association of central nervous system pathologies and aggressivity, the contribution of the frontal cerebral areas appears critical to several authors (Blumer and Benson 1975; Cummings 1985; Fuster 1989). Consequently, most of the neuropsychological measures chosen in this study were specifically sensitive to damage in the various areas of the frontal cortex. It was also thought justified to investigate the relation between neurological soft signs and violence, and between psychopathology and violence, in schizophrenia, as both have been reported to be of significance (Lewis et al. 1979; Krakowski et al. 1989).

Method

Subjects. Thirty-one male outpatients with schizophrenia completed the entire protocol of the present investigation and met all the inclusion criteria, which are presented next. They had all been hospitalized in one of three psychiatric hospitals in Montreal. The total set of active files of schizophrenia outpatients in these hospitals varies from 2,000 to 3,000. All were referred by staff psychiatrists who were aware that we were interested in recruiting "violent" men with schizophrenia, but who in fact had been asked to refer "any" men with schizophrenia. After referral and an individual interview by a trained clinical psychologist, the diagnoses were derived from the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott and Spitzer 1978). The SADS was used to determine the primary psychiatric diagnosis as well as the absence or presence of alcoholism or drug abuse disorders for at least one period of the patient's life. In this regard, 16 cases had had a drug abuse disorder, and 10 of the 16 had suffered from alcoholism. Swanson and colleagues (1990) found that in a large sample of violent psychotic persons, 41.64 percent had a substance abuse disorder, while in another large sample of nonviolent psychotic persons, only 4.93 percent had a substance abuse disorder. The percentage of substance abuse disorder in the present sample (51.16%) may therefore be somewhat high. As for qualification of the primary diagnosis of schizophrenia, 10 subjects were classified as paranoid, 8 as undifferentiated, 7 as schizoaffective, and 6 as residual. Thirteen subjects had an additional secondary diagnosis of antisocial personality. All were receiving several psychiatric medications at the time of the study, including neuroleptics in all cases. Mean chlorpromazine-equivalent daily dose was 808 mg (standard deviation [SD] = 553, range = 67–2,400 mg).

Exclusion criteria consisted of the following: female sex (because schizophrenic violence floors in women); inpatient status (we were interested in patients with a stabilized disease process and neuroleptic maintenance doses); unwill-

ingness to sustain several days of interviewing and testing (six subjects were lost on account of this criterion); unavailability because of current employment (no subject was lost on account of this criterion); having a primary diagnosis other than schizophrenia or a secondary diagnosis of another psychosis (e.g., manic-depressive); having an organic disorder (e.g., dementing process); or having a systemic disease (e.g., pulmonary, cardiovascular). Six patients were excluded, after referral, owing to the presence of one or several of these diseases. Patients who had never caused injury to another person were also excluded, because the correlational design of the present investigation would have been compromised by a flooring effect on this variable. Four patients were excluded by this criterion.

A second group of subjects was also included in this study. They were 30 normal male subjects who had been screened for neurological, psychiatric, alcoholic, and drug abuse disorders. They were matched with the schizophrenia subjects for age and education and served as controls on some neuropsychological measures as a means of determining the actual level of performance of the subjects with schizophrenia.

Assessment of Psychopathology. Since the main focus of this investigation was to explore potential neuropsychological correlates of schizophrenic violence, it was judged worthwhile to include assessment of psychopathology along the positive-negative dimension. Indeed, negative symptomatology in patients with schizophrenia has been linked more often to neuropsychological and neurological impairment than positive symptomatology (Merriam et al. 1990). The relation between schizophrenic violence and the positive-negative dimension has not yet been explored as far as we know. To evaluate symptomatology on the axis of negative-positive symptoms, the Positive and Negative Syndrome Scale (PANSS) protocol was administered (Kay et al. 1987, 1988). Table 1 further describes this group of subjects.

Assessment of Aggressivity and Violence-Related Variables. Psychiatric files, criminal records, and indepth interviews of the schizophrenia patients were the tools necessary for completing the aggressiveness protocol developed at the Institut Psychiatrique Philippe Pinel de Montréal (IPPPM) (*Grille d'Histoire d'Agressivité;* Tremblay and Hodgins 1988). This protocol, now available in English from the IPPPM as *History of Aggressiveness Protocol* (Collin and Braun 1994), is used to evaluate the lifelong history of violent behavior. In this protocol, an "aggressive act" is scored by using a conservative criterion: physical injury must have clearly been very likely caused to a human victim. On the other hand, it is deemed that any violent physical contact (e.g., a punch) automatically meets this criterion. Examples of aggressive behavior not retained include verbal abuse, spitting, throwing cold water, throwing an eraser, and so on. Different measures were derived from this evaluation, but owing to their high level of intercorrelation, only the most meaningful ones are used in the following analyses—namely, "number of violent injuries to persons throughout the entire life span," (minor, moderate, or major) "number of convictions and of acquittals because of mental illness related to violent incidents," and "total length of sentences." Table 2 presents major descriptive information

Table 1. Biographic and clinical description of the schizophrenia subjects

Variables	Mean	SD	Minimum	Maximum
Age (years)	36	6.0	25	49
Education (years)	10	2.7	5	19
Global index of psychopathology (PANSS)	28	9.0	16	56
Total positive symptomatology (PANSS)	13	5.3	7	25
Total negative symptomatology (PANSS)	14	5.9	7	26
Age at first hospitalization (years)	23	4.6	17	32
Number of hospitalizations	11	9.1	1	39
Total length of hospitalization (days)	1,402	896.7	72	3,416

Note.—Typical scores of chronic schizophrenia inpatients on the positive, negative, and global psychopathology subscales of the PANSS are 21.57, 22.90, and 43.56, respectively (Kay et al. 1987). Typical scores of acute inpatient cases are 18.44, 19.96, and 39.48 (Kay et al. 1988). SD = standard deviation, PANSS = Positive and Negative Syndrome Scale (Kay et al. 1987, 1988).

Table 2. Description of violent behavior of the schizophrenia subjects

Variables	Mean	SD	Minimum	Maximum
Incidents of physical aggression causing injury	7.8	5.2	2	27
Convictions and acquittals related to such aggressions	0.4	0.6	0	2
Age at first conviction (years)	22.1	4.8	17	36
Total length of sentences (days)	228.8	428.8	1	1,624
Minor injuries caused	6.7	4.8	1	22
Moderate injuries caused	0.7	1.0	0	3
Major injuries caused	0.1	0.2	0	1
Deaths caused	0.1	0.3	0	1
Aggressions while intoxicated	1.1	1.5	0	5
Aggressions while not intoxicated	6.7	4.7	2	23

Note.—In *History of Aggressiveness Protocol* (Collin and Braun 1994), incidents of physical aggression are defined as violent acts on people having caused bodily harm (i.e., injury). SD = standard deviation.

obtained from the assessment of violence.

Neuropsychological Measures. As a means of establishing the actual level of performance of the psychiatric patients, neuropsychological tests commonly used to describe people with schizophrenia were administered to the two groups. Those measures included the number of correct words on the Controlled Oral Word Association Test (Benton et al. 1983); the number of categories, perseverative errors, and category breaks on the Wisconsin Card Sorting Test (WCST; Heaton 1981); and time to completion on the Trail Making Test forms A and B (Reitan and Davidson 1974).

To explore in greater detail the relationships between violence and neuropsychological functioning, a wide range of other measures was administered to the clinical group only. Those measures include the quantitative and qualitative scores on the Porteus Maze Test (Porteus 1965); the performance and inertia scores on the French Canadian version of the picture arrangement subtest of the Wechsler Adult Intelligence Scale (WAIS; McFie and Thompson 1972; Wechsler 1981); the performance score on the Rey-Osterrieth Complex Figure Test (Osterrieth 1944); the number of errors on the Trail Making Test, forms A and B; and, finally, the median reaction time as well as the number of commission errors on a stimulus-discrimination (go/no-go) task. We designed this latter task for use on a microcomputer. The task consisted of a set of 150 trials requiring the subject to press the space bar to respond to either a white square or a buzz sound as fast as possible. The two types of stimuli were randomly equiprobable. Once this simple reaction time condition was completed in view of creating a positive response habit, the subject was required to respond only to the squares (n = 50) and not the buzzes (n = 50). Median reaction time of correct responses and number of commission errors on this task were retained for further analysis.

To support the idea of implication of the frontal cortex in violent behavior, the neuropsychological measures were selected according to their potentially localizing qualities. In fact, the qualitative error score on the Porteus Maze Test seems to be mostly affected (positively) by orbitofrontal damage (Crown 1952; Petrie 1952; Tow 1955; Porteus 1965; Gow and Ward 1982). Commission errors on a go/no-go paradigm also seem to be affected by damage in this area (Drewe 1975). Both these measures reflect impulsivity, which typically results from orbitofrontal lesions. Some evidence tends to link low verbal fluency and inertia on the picture arrangement subtest of the WAIS with mesial frontal damage (McFie and Thompson 1972; Hécaen and Ruel 1981). Perseverative errors on the WCST correlate with dorsolateral frontal damage more than with orbitofrontal damage (Milner 1964), even though they do not seem to distinguish frontal lesions in general from posterorolandic lesions (Anderson et al. 1990). This particular deficit is considered one of decreased mental flexibility. The performance score on the Rey-Osterrieth Complex Figure Test is more dependent on the integrity of the posterorolandic cortex than on the anterior areas, as it requires mostly visuospatial integration (Eslinger and Grattan 1990). The other neuropsychological measures obtained in this study were considered nonspecific in regard to their localizing properties.

Neurological Assessment. A 108-item version of the Nathan Kline

Institute (NKI) neurological scale of soft signs was administered to the schizophrenia subjects. Earlier versions of this neurological scale have been found suitable for clinical practice and research (Brizer et al. 1987; Convit et al. 1988; Krakowski et al. 1989).

Results

Methodological Considerations. The schizophrenia subjects in this sample were clearly neuropsychologically impaired. See table 3 and Braun et al. (in press) for details concerning differences between the schizophrenia and control subjects.

The violence measure with the widest range, and the measure most representative of a pure disposition to violent acts, was "number of incidents of physical aggression causing injury" (see table 2). The distribution of this variable was unfortunately skewed (though not complicated by outliers), making parametric analysis of its relation to other measures questionable. Consequently, we carried out a nonparametric procedure (a two-group comparison at median using the chi square test) that is not biased by skewness. Although none of the neuropsychological tests or the clinical measures, or the neurological scale related significantly to the aggression measure, valences of relationships were identical to those obtained by parametric correlation analysis. This encouraged us to proceed (table 4) with more sensitive and sophisticated multivariate (i.e., partial correlational) analyses. The measures of the present study most directly informative of the schizophrenia disease process were the PANSS negative scale, positive scale, and global scale. None of these scales correlated significantly with lifelong "number of physical aggressions against another person causing injury." Aside from the schizophrenia disease itself, several "clinical" and biographic variables could also conceivably relate to violence. Correlations of such variables with lifelong "number of physical aggressions against another person causing injury" were the following: age at time of schizophrenia onset, $r = -0.23$; number of psychiatric hospitalizations, $r = 0.01$; total duration of psychiatric hospitalization, $r = 0.05$; age, $r = -0.24$; education, $r = -0.19$; and level of daily neuroleptic intake, $r = -0.23$. All of these were far from significant, the critical alpha (uncorrected) of 0.05 requiring an r of 0.34 or more. No significant relationship was observed among the subgroups by secondary diagnosis of schizophrenia type and the various neuropsychological, neurological, and violence-related measures. Furthermore, the results of the subjects with an additional diagnosis of antisocial personality did not differ significantly from those of the other subjects ($r =$

Table 3. Results obtained by the schizophrenia group on the neuropsychological and neurological measures

Measure	Mean	SD	Minimum	Maximum
Qualitative score (Porteus Maze Test)	40.0	24.7	8	89
Commission errors (go/no-go)	9.8	15.0	0	74
Verbal fluency (COWAT) (correct words)	31.0	8.7	18	54
Inertia score (WAIS–R subtest)	4.9	3.5	0	15
Perseverative errors (WCST)	35.6	21.6	10	95
Global score (Rey-Osterrieth Complex Figure Test)	60.9	9.7	35	72
Quantitative score (Porteus Maze Test)	121.5	28.2	70	170
Scaled score (WAIS–R subtest)	7.3	4.8	2	18
Categories achieved (WCST)	2.3	1.8	0	6
Category breaks (WCST)	1.0	1.2	0	4
Go/no-go reaction time (seconds)	275.7	52.7	185	415
Time (Trail Making Test A)	67.3	36.1	26	174
Time (Trail Making Test B)	152.4	86.1	49	404
Number of errors (Trail Making Test A)	0.4	0.7	0	2
Number of errors (Trail Making Test B)	0.9	1.2	0	4
Total score (NKI scale)	9.9	6.5	2	36

Note.—Controlled Oral Word Association Test (COWAT; Benton et al. 1983); go/no-go (Drewe 1975); NKI (Nathan Kline Institute [Brizer et al. 1987, Convit et al. 1988; Krakowski et al. 1989]); Porteus Maze Test (Porteus 1965); Rey-Osterrieth Complex Figure Test (Osterrieth 1944); Trail Making Test, Forms A and B (Reitan and Davidson 1974); Wechsler Adult Intelligence Scale–Revised (WAIS–R; Wechsler 1981), Wisconsin Card Sorting Test (WCST; Heaton 1981), SD = standard deviation.

Table 4. Correlations and partial correlations between the number of incidents of physical aggression causing injury and the various neuropsychological and neurological measures

Measure	*R*	*Rp1*	*Rp2*
Qualitative error score (Porteus Maze Test)	−0.26	−0.12	−0.25
Commission errors (go/no-go)	−0.33	−0.36	−0.45
Verbal fluency (COWAT)	0.45[1]	0.80[1]	0.58[2]
Inertia on WAIS–R Picture Arrangement	−0.04	−0.20	−0.01
Perseverative errors (WCST)	−0.28	−0.36	−0.19
Performance score (Rey-Osterrieth Complex Figure Test)	0.17	0.45	0.26
Quantitative performance (Porteus Maze Test)	−0.04	−0.27	−0.37
Picture arrangement performance (WAIS–R)	0.06	0.12	−0.14
Categories achieved (WCST)	0.48[2]	0.70[1]	0.66[1]
Category breaks (WCST)	0.03	0.13	−0.09
Reaction time (go trials)	0.11	−0.17	0.19
Time to completion (Trail Making Test A)	−0.07	−0.45	−0.16
Time to completion (Trail Making Test B)	−0.19	−0.30	−0.12
Errors (Trail Making Test A)	−0.06	0.56	0.27
Errors (Trail Making Test B)	−0.04	0.17	0.03
Soft signs (NKI)	−0.37[2]	−0.51	−0.23

Note—*R* = univariate correlation *Rp1* = correlation with age, education, number of psychiatric hospitalizations, total length of psychiatric hospitalizations, age of schizophrenia onset, and daily neuroleptic dose partialed out. *Rp2* = correlation with PANSS total symptom score, alcoholism, and drug abuse disorder partialed out. Alpha levels are controlled for each partial correlation, but not tablewise. Radical adjustment of type I error using the additive (Dunn) method would require an *r* value of 0.5 and a *p* value of 0.003 as an equivalent to the standard alpha criterion of $p = 0.05$ (two-tailed) for the univariate coefficients. Controlled Oral Word Association Test (COWAT; Benton et al. 1983), go/no-go (Drewe 1975); NKI (Nathan Kline Institute [Brizer et al. 1987; Convit et al. 1988; Krakowski et al. 1989]); Porteus Maze Test (Porteus 1965); Rey-Osterrieth Complex Figure Test (Osterrieth 1944); Trail Making Test, Forms A and B (Reitan and Davidson 1974); Wechsler Adult Intelligence Scale–Revised (WAIS–R, Wechsler 1981); Wisconsin Card Sorting Test (WCST; Heaton 1981).

[1] $p = 0.01$.
[2] $p = 0.05$.

0.23, $p = 0.3$). Subjects with secondary diagnoses of alcoholism and/or drug abuse disorder were not significantly more violent than the rest of the sample ($p = 0.5$). However, it is noteworthy that substance abuse presented a trend toward positive correlation with violence, as was found by previous investigators. Finally, a subsample of 26 patients receiving antiparkinsonian medication did not differ from the rest of the sample ($p = 0.4$).

Neurological and Neuropsychological Functioning and Violence in Schizophrenia. Exploring the relationship between neuropsychological functioning and lifelong history of violence in schizophrenia was the main objective of this study. We therefore proceeded with correlational analysis of those variables. First, univariate correlational analyses were done of the "lifelong number of aggressions against another person causing injury" with the neuropsychological and neurological variables. Partial correlations were also carried out, removing in a single pass the combined variance potentially attributable to sampling bias—namely, age, education, number of psychiatric hospitalizations, total length of psychiatric hospitalizations, age at onset of schizophrenia, and daily chlorpromazine-equivalent neuroleptic dose. Although none of these variables were individually related to violence in this sample, we wanted to make sure they were not additively or otherwise distorting in a multivariate manner the correlations of interest.

Another partial correlation analysis was carried out to remove surface indicators of general psychopathology (PANSS total symptom score, alcoholism, drug abuse disorder) to determine the distinct relationship between congenital neuropsychological or neurological impairment and violence. Table 4 presents the results of these correlational analyses.

Identical analyses were done with the other two most important measures derived from the assessment of violence, that is, "number of convictions and acquittals because of mental illness related to violent incidents" and "total length of sentences." However, the results obtained were nearly identical to those presented in the previous table and are therefore not presented.

Although the test of significance for each partial correlation was controlled for number of parameters (type I error), the tests of significance were not adjusted ex-

perimentwise. Of the 16 neuropsychological tests (including the NKI scale) presented in table 3, 14 indicated a positive relationship between neuropsychological performance and aggression. Of these tests, two consistently reached significance in the parametric analyses before and after correction for (1) psychopathology ratings and substance abuse and (2) potential sampling bias, but not after additive correction for type I error (see note to table 4). The two negatively correlated measures (Rey-Osterrieth Complex Figure Test and reaction time) were far from significant. In short, the ensemble of the results suggests a subtle overall positive relationship between neuropsychological performance and violent physical aggression in this sample of schizophrenia subjects.

Discussion

As can be derived from table 3 and from Braun et al. (in press), the schizophrenia sample subjects were clearly impaired at the general neuropsychological level. Although this group of outpatients was neuropsychologically impaired at a level typical of schizophrenia outpatients (Goldstein 1987; Taylor and Abrams 1987), it seems reasonable to assume that they were quite functional in relation to the samples of most of the studies of violence in persons with schizophrenia cited in the introduction, because these outpatients were functional enough to participate in a lengthy assessment requiring good cooperation.

As for the relationship between violence and cerebral impairment in schizophrenia, the results obtained in this study seem at first quite illogical. In fact, they suggest a higher level of violence in the more cognitively functional individuals, although often nonsignificantly. This sometimes significant relationship persists even when the effect of potentially confounding variables has been statistically removed. Such a finding does not support other neurologically or neuropsychologically oriented investigations (Krakowski et al. 1989; Adams et al. 1990). We believe that this finding could be related to the rather high level of functioning manifested by our subjects. In the above-cited studies, subjects had generally deteriorated more profoundly and were more violent. Evidence of the high level of functioning of the present sample includes its low scores on the PANSS subscales (see table 1, particularly the note). In contrast with the present study, Krakowski et al. (1989), for example, studied inpatients who manifested a high incidence of assaultiveness on psychiatric wards. We believe that this type of violence may reflect a highly impulsive, irritative, uncontrolled, and unplanned neurological type of aggressivity not so frequently seen in our relatively functional outpatients.

This hypothetical explanation is further supported by the findings of Planansky and Johnston (1977), which demonstrated that physical aggression occurred much more frequently in schizophrenia subjects who showed adequate social adjustment between hospitalizations than in continuously incapacitated individuals. Likewise, Roy et al. (1987) reported that violent schizophrenia outpatients performed significantly better on performance subtests of the WAIS than nonviolent schizophrenia outpatients. In light of all previous findings, the present findings suggest that whereas schizophrenia inpatient violence may be neurologically based, schizophrenia outpatient violence is not necessarily so. Furthermore, outpatient violence occurred unequivocally more in the least cognitively impaired cases. We were not able to link the phenomenon to any other variable (alcoholism, drug abuse, age, education, neuroleptic medication, etc.). Nor could the presence of an additional diagnosis of antisocial personality account for it, even though such patients usually show less deterioration in the premorbid level of functioning (Schanda et al. 1992). The prevalence of the "antisocial personality" subdiagnosis in the present sample (42%) seems high. Our guess is that some of this prevalence is due to referring psychiatrists' awareness of the goals of the present investigation, and some to exclusion of totally nonviolent cases. Finally, we suspect that prevalence of "antisocial personality" concomitant with schizophrenia may have been underinvestigated and underreported in the schizophrenia literature.

In people with schizophrenia who are unable to live in any community setting (i.e., chronic inpatients), a more "neurological" type of aggressivity is probably more frequently observed. This type of violence could manifest itself in outbursts of assaultive behavior on an almost daily basis and is probably related to a low threshold of frustration tolerance and to the severity of the symptoms. Conversely, violent aggressiveness was not so prevalent in the present sample, making significant correlations more difficult to achieve.

It thus appears essential to focus very carefully on the description of the type of violence one is con-

sidering in any attempt to relate it to neuropsychological or neurological impairment. It seems just as important to distinguish outpatients from inpatients in this research domain. Further, it is necessary to beware of selection bias, even within the outpatient samples. For example, atypically more violent outpatients, contrary to the present sample, manifest a significant negative correlation between neuropsychological performance and level of violence (Adams et al. 1990). A wide range of different antisocial behaviors can be manifested by a great diversity of individuals who, although in the same diagnostic category, exhibit varying levels of preserved functioning. This important heterogeneity of individuals and behaviors adds further complexity to the already difficult task of relating cognitive or emotional dysfunctions to anatomical cerebral substrates.

These findings and interpretations also indicate that the etiology of outpatient schizophrenia violence requires further research. One hypothesis to be pursued is the following. In the context of massive deinstitutionalization of patients with psychoses in industrialized countries, the highest functioning patients may have more social contact, "social" drinking, and drug abuse in public. The low-functioning ones will probably avoid social contact and, consequently, substance abuse. Aggressiveness will be higher in the high-functioning ones, not because of an intrinsic (endogenous) determinant, but because of greater exposure to aggression-provoking situations (i.e., interpersonal conflicts) aggravated by disinhibition resulting from substance abuse. However, within the range of extreme outpatient violence, the reverse relationship may prevail, brain insults then being a major source of variance. To test such a hypothesis, less stringent exclusion criteria would have to be used—particularly inability to complete such a lengthy and complex assessment procedure as the one used in the present investigation—and the higher end of the range of outpatient violence would have to be actively sampled.

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