Daniel A. Martell,¹ Ph.D.

Estimating the Prevalence of Organic Brain Dysfunction in Maximum-Security Forensic Psychiatric Patients

REFERENCE: Martell, D. A., "Estimating the Prevalence of Organic Brain Dysfunction in Maximum-Security Forensic Psychiatric Patients," *Journal of Forensic Sciences*, JFSCA, Vol. 37, No. 3, May 1992, pp. 878–893.

ABSTRACT: This is a descriptive study of 50 randomly selected male patients retained in a maximum-security state hospital for mentally disordered offenders. Data regarding the prevalence of several indicators of potential organic brain dysfunction are presented, including: (1) a diagnosis of any organic brain disorder, (2) a history of severe head injury with loss of consciousness, (3) a history of seizure activity, (4) evidence of cognitive impairment, (5) abnormal neurological findings, and (6) other relevant neurodiagnostic or historical findings. Results show that multiple indicators of potential brain dysfunction were present in 64% of the cases. At least one indicator of potential brain dysfunction were significantly more likely to have been indicted for violent criminal charges (p = 0.01). Implications of these findings for clinical treatment and forensic science decision-making are discussed.

KEYWORDS: psychiatry, mental illness, organic brain disorder, forensic psychology, neurological impairment, head injury, mentally disordered offenders, violence

The neurobehavioral components of violence are of increasing importance to forensic behavioral science. However, few studies have set out to determine the prevalence of organic brain dysfunction in a general forensic psychiatric population², despite the growing assertion by forensic science clinicians that mentally disordered offenders have significant rates of organic brain disorder [5,6]. If impaired brain functioning plays an important role in the etiology of violent behavior, one would expect the prevalence of organic brain

This paper was awarded the 1991 AAFS Psychiatry and Behavioral Science Section Fellowship Research Award. Portions of this paper were presented at the bi-annual meeting of the American Psychology-Law Society/Division 41 of the American Psychological Association, 10-12 March 1988 in Miami, FL; and at the 43rd Annual Meeting of the American Academy of Forensic Sciences, 18-23 Feb. 1991 in Anaheim, CA.

¹Nathan S. Kline Institute for Psychiatric Research, New York University School of Medicine, and director, Forensic Neuropsychology Laboratory, Kirby Forensic Psychiatric Center, New York, NY.

²Rates of organic impairment have been reported for several specific classes of mentally disordered offenders seen in forensic psychiatric settings. For example, organic impairment has been documented in: (1) 0.9% of 104 adolescent defendants [1]; (2) 41% of 54 Mariel refugees [2]; and (3) 18% of 62 sex offenders [3,4].

impairment in this population to be high. This kind of information has important implications for the evaluation and treatment of forensic psychiatric patients as well as for assessments of future dangerousness.

To date, our primary clues to the possible prevalence of brain disorders among mentally disordered offenders come from two diagnostic census studies, both of which were conducted outside the United States. Odejide [7] studied the population of "criminal lunatics" at the Lantoro Psychiatric Institution in Nigeria. Using a structured questionnaire to assess psychopathology and determine diagnoses, his data indicate that 24.6% of the subjects were either epileptic (18.9%) or suffered from organic psychoses (5.7%). Häfner and Böker, [8] working in the Federal Republic of Germany, conducted an extensive epidemiological diagnostic study of a cohort of 533 mentally disordered offenders entering the forensic mental health system over a 10-year period. Their data suggest that 33.6% of these patients had a diagnosis reflecting organic cerebral impairment. These included mental retardation (12.7%), late-acquired brain damage (8%), cerebral atrophy (7.5%), and epilepsy (5.4%).

Studies of other violent populations have documented even higher levels of brain impairment. Elliott [9] studied 286 nonpsychotic subjects with a history of recurrent uncontrollable rage attacks and found evidence of developmental or acquired brain defects in 94%. In an uncontrolled study of 15 inmates on death row in American prisons, Lewis et al. [10] found evidence suggestive of brain impairment in 100% of the cases.

Violence and Brain Dysfunction

Theoretical models from neuropsychology [11,12] and neurology [13,14] suggest that brain damage may increase the risk of violent behavior. Abnormal brain functioning may impair inhibition of violent impulses or stimulate excesses in impulsivity and behavioral dyscontrol. Either mechanism may increase an individual's propensity to aggressive or violent behavior, particularly in combination with other characterological, environmental, or situational risk factors. Researchers have begun to document the importance of several specific types of brain impairment in violent behavior [15-17], four of which were selected for examination in the present study.

Head Injury

Head injury is a major cause of acquired brain damage [18,19], often resulting in focal damage to the frontal or temporal lobes or both. Several studies have correlated damage to these specific brain regions with violent behavior [20-23]. For example, one study specifically associates temporal lobe lesions secondary to head injury with violence [24]. Another study [25] suggests that as many as 70% of those suffering a severe head injury experience significant levels of aggressive and irritable behavior. Severe head injuries have been reported in a significant proportion of murderers in Iceland [26], and 100% of death-row inmates in the United States [10].

Seizure Disorder

Seizure disorder is another important area in which brain dysfunction appears to be associated with violent behavior [27-29]. However, the precise nature of the relationship between epilepsy and violent behavior remains a controversial issue [30]. Most studies associate "irritative" lesions arising within the temporal lobes with violence, as illustrated by the literature on temporal lobe epilepsy [31-34] and the "episodic dyscontrol syndrome" [35,36]. Note that current opinion [27,30,37] associates the increased risk of

violence with behavior occurring during interictal periods, rather than with ictal or preictal behavior per se.

Cognitive Impairment

Violent behavior is also prevalent in populations exhibiting various forms of cognitive impairment. Intellectual and neuropsychological deficits, mental retardation, Alzheimer's disease, and other forms of dementia all have known associations with violent behavior [17]. Behavioral studies of mentally retarded adults have documented violent behavior in 30 to 40% of cases [38,39]. Bryant et al. [40] found a significant relationship between learning disability, neuropsychological deficits, and violent criminal behavior. Impaired scores on subscales of the Wechsler Adult Intelligence Scale have also been associated with violent behavior [41]. Programmatic studies by Yeudall and his colleagues [42-44] have documented significant neuropsychological deficits in both violent prisoners and forensic psychiatric patients. Other neuropsychologists have also demonstrated the ability of neuropsychological findings to discriminate between violent and nonviolent offenders [45-46].

Neurological Impairment

Neurological abnormalities also appear to play a role in violent behavior. Monroe et al. [47] were among the first to document abnormal neurological findings in recidivistically violent prisoners. More recently, programmatic studies by the violence research group at New York University Medical Center and Manhattan Psychiatric Center [48–50] have repeatedly implicated neurological impairment in the behavior of violent psychiatric inpatients.

The present study begins to examine the prevalence of several indicators of organic brain disorder in a random sample of N = 50 male patients from a maximum-security state hospital for mentally disordered offenders. These pilot data were collected to determine if more controlled and extensive neurodiagnostic studies would be warranted. Six indicators of potential brain dysfunction were examined through a review of chart material: (1) a *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R) diagnosis of any organic brain syndrome, (2) a history of head injury with loss of consciousness, (3) evidence of cognitive impairment, (4) seizure disorder, (5) neurological abnormalities, and (6) other relevant neurodiagnostic or historical findings. Each of these indicators was selected on the basis of the research literature discussed above because of its potential association with violent behavior.

Methods

Setting

This study was conducted at Kirby Forensic Psychiatric Center (KFPC), a maximumsecurity state hospital for mentally disordered offenders located on Ward's Island in New York City. The center is the city's primary forensic psychiatric evaluation and treatment setting for both men and women, with a catchment area including all of Manhattan and portions of the outer boroughs. The patient population at KFPC is typical of that found in similar forensic psychiatric facilities in other major metropolitan areas.

Subjects

A random sample of 50 male patients was selected from the population of 125 male patients retained at KFPC. The ages of subjects in the sample ranged from 18 to 73 (M

= 33.26, SD = 11.79 where *M* is the mean, and SD is the standard deviation), accurately reflecting both the mean age and range of ages in the KFPC male population as a whole. The racial composition of the sample also reflects that of the entire facility. The majority of subjects in the sample were Black (46%), followed by Hispanic (34%), and White (20%).

Admissions to the facility are court-ordered, primarily under two articles of the New York State Criminal Procedure Law (CPL). Patients admitted under CPL Article 330.20 were found not responsible by reason of mental disease or defect (not guilty by reason of insanity or NGRI). These patients are retained for the evaluation and treatment of "dangerous" mental disorders. The NGRI patients comprise approximately 40% of the total hospital population and 36% of the present sample. Patients admitted under CPL Articles 730.40 through 730.50 were found "unfit to proceed" in court (incompetent to stand trial) and are admitted for evaluation and treatment to restore them to competency. Competency patients comprise approximately 60% of the hospital population and 64% of the present sample.

Procedure

Chart Review—Hospital records (including standardized psychological, psychiatric, criminal-legal, social work, and admission assessments) were reviewed for demographic, diagnostic, forensic, and historical data. Demographic data included age at the time of admission and racial designation. Forensic science data included each subject's instant offense and current legal status (that is, NGRI or incompetent to stand trial).

Data on five indicators of organic brain dysfunction were collected. The first was a diagnosis of any organic brain disorder (for example, senile dementia of the Alzheimer's type, organic hallucinosis). The second was documented evidence of a history of severe head injury, defined as involving loss of consciousness. The third was any history of seizure activity. The fourth was evidence of cognitive impairment as reflected in a diagnosis of mental retardation or test evidence of cognitive functioning at or below the borderline intelligence quotient (IQ) level (79). An IQ of 79 and below represents cognitive functioning in the bottom 6.7% of the adult population [51]. The fifth indicator was any other documentation of abnormal brain structure or function (for example, as reflected in the results of neuropsychological testing, neurodiagnostic findings, or significant historical events suggestive of compromised brain sequelae).

Quantified Neurological Examination—Convit et al.'s [48] quantitative neurological scale was used to measure neurological integrity. The scale follows the traditional neurological examination format, however individual signs and symptoms are scored zero if normal and one if abnormal. Scores for both hard and soft neurological signs are provided. Soft signs are abnormal clinical features that cannot be precisely localized or lateralized within the brain. These include findings such as motor overflow, graphesthesia, and stereognosis. Only subjects exhibiting two or more soft signs were scored as abnormal. Hard signs are clinical findings that can be reliably localized within the brain. These include a range of reproducible behavioral evidence of brain dysfunction. To be scored as abnormal, any given subject had to exhibit two or more hard signs. The neurological scale total score has an inter-rater reliability of coefficient of 0.98 (Pearson's R), with Kappa coefficients for individual items ranging from 0.69 to 1.00.

Findings

Prevalence Estimates

The data obtained for this study illustrate the scope and the diversity of potential brain impairment in the sample. Data for each subject are presented in Table 1, which permits

Neurodiagnostically	Suggestive Findings	20-point IQ drop over 10 vears			Head banger as child		Hearing loss)				Abnormal right hemi- sphere neuropsy-	chological findings			Father banged pa- tient's head as	child/car accident	Multiple head inju- ries/Shot self in	licau	
	Cog	I	I	I	I	I	I	I	I		I	I		I	+	I		I	+ +	I
History of	Seizure	I	I	I	i	I	I	I	+	I	I	I		I	I	ì		I	ŧ	1
	Hi	1	I	I	+	I	I	I	I		I	I		I	I	+		+	I	+
cal Signs	Hard	1	i	+	E	+	+	I	I	-	ł	+		1	E	i		E	ł	E
Neurological Signs	Soft	I	+	+	E	+	+	I	ł	-	÷	I		I	E	I		E	i	E
	Diagnosis	Drug-induced OBS	Schizophrenia, Paranoid	Schizophrenia, Undif	Schizophrenia, Undif	Schizophrenia, Undif	Alzheimer's	Antisocial PD	Organic hallu-	Cinations Schirzonhronio	scnizopnrenia, Paranoid	Bipolar-Manic	4 	Borderline PD	Organic Delu- sion Disa- bility	Schizophrenia, Paranoid		Delusional Disability	Organic Delu- sion Disa-	bility Antisocial PD
Legal	Status	NGRI	NGRI	NGRI	CST	NGRI	CST	CST	CST	IGUN	INUN	CST		NGKI	NGRI	NGRI		CST	NGRI	NGRI
	Crime	Murdr	Murdr	Murdr	Murdr	Murdr	Murdr	Murdr	Murdr	Munde	Muru	Murdr	-	Murdr	Murdr	Murdr		Murdr	Mansl	AttMr
	Case	-	3	ŝ	4	S	9	1	8	c	ע	10	;	II	12	13		14	15	16

Left cerebral lesion on CT/aphasia/R	WCARIICSS			Visual field deficits			Frontal lobe findings on neuropsycholog-	1001 10010		Head banger/diffuse severe neuropsy- chological abnor-	Atypical OBS from infancy, attention	exp & rec Aphasia/ right-side hearing	1088	Fetal alcohol syn-	Diffuse abnormalities on neuropsycholog- ical tests
+	ł	I	I	+ +	I	I	I	I	ł	+ +	+	+ +	I	ł	+ +
I	+	ł	I	I	I	I	I	I	I	I	I	I	1	I	I
+	+	I	1	I	I	I	ł	I	ł	I	I	I	I	ł	+
+	+	+	+	+	+	ł	÷	+	в	I	+	+	I	E	+
I	+	+	I	+	+	I	I	÷	в	+	+	ł	+	E	+
Schizophrenia, Residual	Alcoholic De-	Schizoaffec-	uve Schizophrenia, Paranoid	Schizophrenia, I Indif	Schizophrenia, Paranoid	Schizophrenia, Undif	Schizophrenia, Residual	Schizophrenia, 1 Indif	Bipolar, Ma-	Schizophrenia, Undif	Atypical OBS	Mental Retar	Bipolar, Ma-	Psychotic	Schizophrenia, Disorg
NGRI	CST	CST	CST	CST	NGRI	NGRI	CST	CST	CST	CST	CST	CST	CST	CST	NGRI
AttMr	AttMr	AttMr	AttMr	Asslt	Asslt	Asslt	Asslt	Asslt	SexOf	SexOf	SexOf	SexOf	SexOf	Robry	Robry
17	18	19	20	21	22	23	24	25	26		28	29	30	31	32

PSYCHIATRY AND BEHAVIORAL SCIENCE SECTION AWARD PAPERS 883

				TABLE	TABLE 1—Continued	pa			
		Legal		Neurological Signs	ical Signs		History of		Neurodiaenostically
Case	Crime	Status	Diagnosis	Soft	Hard	H	Seizure	Cog	Suggestive Findings
33	Robry	CST	Schizophrenia, Undif	1	+	ł	1	+ +	
34	Robry	CST	Psychotic NOS	+	+	1	1	! -	
35	Robry	CST	Schizophrenia, Residual	I	÷	+	I	I	
36	Robry	CST	Psychotic NOS	E	E	+	I	1	
37	Robry	NGRI	Schizophrenia, Paranoid	+	+	I	1	I	
			SPE	CIFIC NEUR	SPECIFIC NEURODIAGNOSTIC FINDINGS	FINDINGS			
38	Robry	CST	Schizophrenia,** Undiff	E	8	+	+	ł	Hypoxia 2° to hang- ing self at 15 years old
39	Robry	CST	Mixed Drug	1	1	1	I	l	
40	Arson	CST	Bipolar, Ma-	9	ì	I	ł	I	
41	Arson	CST	Schizophrenia, Democid	+	÷	I	1	I	
42	Arson	NGRI	Schizoaffec-	I	Ì	1	I	Ι	
43	CPWpn	CST	uve Major Depression	1	+	+	I	I	CVA (stroke) with right-side Hemi-
44	CPWpn	CST	Schizoaffec-	B	H	I	+	I	
45	GrLar	CST	Schizophrenia,	+	+	I	1	I	
46	Burgl	CST	Schizophrenia, I adif	I	+	I	I	l	
47	Burgl	CST	Dysthymia	B	ш	Ι	I	I	

			16 (32%)	
I	I	I	9 (18%)	
I	I	I	4 (8%)	
I	I	Ι	11 (22%)	
+	ł	+	26 (52%) [65%]	
+	+	I	20 (40%) [50%]	tardation ubstance
Schizophrenia,	Poly-Sub- stance	Abuse Poly-Sub- stance Abuse	8 (16%)	 = indicator present = indicator absent = cognitive function in the range of mental retardation = missing data = incompetent to stand trial = not guilty by reason of insamity = morder = manslaughter = attempted murder = sexual offense = assult = assult = eriminal possession or sale of a controlled substance = robbery = criminal mischief = burglary = criminal mischief
NGRI	NGRI	CST		 indicator present indicator absent cognitive function in the range o missing data incompetent to stand trial incompetent to stand trial not guilty by reason of insanity murder murder attempted murder assault assault robbery robbery eriminal possession of a veapon grand larceny burglary
CrMch	Drugs	Drugs	TOTALS % of 50 % of 40	 indicator present indicator absent cognitive functior missing data micompetent to st not guilty by reast not guilty by reast murder murder murder attempted murder assault exual offense assault exual offense assault possessic eriminal possessic argrand larceny eriminal mischief
48	49	50	T01 % (" + + + m CST CST CST CST Murdr Murdr Mansl AttMr Assit Secof CPWpn CrMch Burgl Burgl

the examination of both the prevalence of specific indicators in the total sample, as well as the unique combinations of findings which characterize individual cases. Data on each indicator will be presented below, followed by an examination of the cumulative prevalence of these data.

Organic Diagnoses—A DSM-III-R diagnosis reflecting organic brain impairment was present for 16% of the 50 cases sampled. The organic diagnoses are presented in Table 1. They include alcoholic dementia, primary degenerative dementia of the Alzheimer's type, organic delusional disorder, organic hallucinosis, mental retardation, and atypical organic brain syndrome. The most prevalent nonorganic diagnosis was schizophrenia (46%), followed by the affective disorders (12%). Smaller proportions of the sample had diagnoses of schizoaffective disorder (6%), delusional disorder (2%), or psychotic disorder not otherwise specified (NOS) (6%). The remaining subjects were diagnosed as having a personality disorder (6%), or substance abuse disorder (6%). It is notable however, that 57% of the subjects with a nonorganic psychiatric diagnosis also had two or more indicators of potential brain dysfunction. For example, Case 17 had a diagnosis of schizophrenia, residual type. However, he also had a history of head injury with loss of consciousness, evidence of neurological hard signs, cognitive functioning in the borderline range, and computerized tomography (CT) scan evidence of a left hemisphere cerebral lesion with secondary right-sided weakness and aphasia.

Head Injury—A history of severe head injury, defined as involving loss of consciousness, was present in 22% of the 50 subjects. Head injuries were acquired at various ages and resulted from a variety of traumas. Records indicated that at least four of these subjects had acquired head injuries secondary to child abuse. For example, Subject 13 was repeatedly abused by his father, who would bang his head against the floor or a wall until the subject lost consciousness. Other patients acquired head injuries later in life, as the result of car accidents, fights, and other blunt traumas. One subject shot himself in the head with a hand gun. Of these subjects, 45% had a history of multiple head injuries.

Seizure Disorder—A history of seizure activity was among the least prevalent findings. Only 8% of the subjects had a documented history of seizure activity. By comparison, this rate exceeds that reported by Häfner and Böker [8] among mentally disordered offenders in the Federal Republic of Germany (5.4%), but it is lower than that reported by Odejide [7] in Nigeria (18.9%).

Cognitive Impairment—Evidence of cognitive impairment was present in 18% of the subjects. Four percent scored in the range of borderline mental retardation (full-scale IQ between 70 and 79), while an additional 12% fell in the mentally retarded range of intellectual functioning (full-scale IQ below 70). While this rate is already higher than the 12.7% reported by Häfner and Böker [8], the present estimate is likely to be fairly conservative since few subjects had reports of actual IQ testing.

Neurological Abnormalities—By far the most prevalent findings were neurological abnormalities. Unfortunately, ten subjects refused to consent to the quantified neurological examination. However, abnormal neurological findings were obtained in 75% of the remaining 40 consenting patients. Soft signs were observed in 50% of these patients, while hard signs were present in 65%. The greater prevalence of hard signs is notable because hard signs generally reflect more significant, localizable brain dysfunction. Hard and soft signs occurred together in 40% of the subjects.

Suggestive Historical and Neurodiagnostic Findings—Finally, a wide array of other historical or neurodiagnostic findings suggestive of brain impairment were discovered in the chart material. Relevant findings were noted for 32% of the subjects. These ranged

from focal findings on CT scan and neuropsychological assessments to cerebral-vascular accident, hypoxia, aphasias, sensory-perceptual deficits, mutism, and fetal alcohol syndrome.

Cumulative Prevalence Estimates—Taking into account all the indicators of brain dysfunction discussed above, two or more indicators were present in 64% of the subjects. Eighty-four percent of the subjects had at least one indicator suggesting potentially abnormal brain functioning. These estimates suggest that brain impairment may be present in a substantial majority of mentally disordered male offenders.

The most conservative estimate, that is, relying solely on a diagnosis of organic brain dysfunction, would suggest that just 16% of the cases are organic. However, attention to relevant historical data found in the patients' charts increased this estimate to 50%. Going a step further to obtain neurological scale data from consenting patients (40 of the 50 subjects) identified an additional 34% of the cases, bringing the prevalence estimate to 84%. A more controlled study including thorough neuropsychiatric histories coupled with neuropsychological and neurodiagnostic testing, or both, could potentially reveal even higher rates of brain abnormality.

Correlates of Brain Dysfunction

Given the high prevalence rates reported above, relationships were explored between evidence of potential brain dysfunction and (1) demographics, (2) legal status, and (3) violent criminal indictments. Subjects with two or more indicators of potential brain dysfunction were classified as organic. This yielded a group of n = 32 organic subjects, and a comparison group of n = 18 nonorganic subjects.

Demographics—The average age of subjects in the organic group (M = 34.3, SD = 13.3) was three years older than that of the nonorganic group (M = 31.3, SD = 8.5). However, this difference in age failed to reach statistical significance, t(47.09) = 0.97, p > 0.5. Breaking the sample down by racial designation, Hispanic subjects had the highest rate of potential organic brain impairment (82.4%). In contrast, only 56.5% of the Black subjects and 50% of the White subjects fell in the organic group. However, the apparent overrepresentation of Hispanic subjects among those with brain impairment was not statistically significant, $\chi^2 (2, N = 50) = 3.89$, p > 0.05. Hence, these demographic findings suggest that indications of brain impairment are evenly distributed among male patients of all ages and racial backgrounds.

Legal Status—Patients in the sample fell into two legal status categories: NGRI cases (36%) and competency to stand trial (CST) cases (64%). Sixty-seven percent of the NGRI cases, and 63% of the competency cases were classified as organic. No significant difference was obtained in the prevalence of organic brain syndrome (OBS) among these two classes of patients, χ^2 (2, N = 50) = 0, p = 1.0. The fact that brain impairment is equally prevalent in these two groups of patients suggests that it may have little bearing on insanity acquittals. A higher rate among NGRI subjects might indicate that brain-impairment increases the likelihood of acquittal by reason of mental disease or defect.

Violent Crime—The small number of subjects in this sample indicted for nonviolent offenses (12%) limits what these data can say about violent crime. However, several notable trends did emerge. All of the subjects with a DSM-III-R diagnosis of organic brain disorder had been arrested and charged for violent crimes. Of these patients, 75% were charged with murder, manslaughter, or attempted murder. The remaining 25% were charged with violent sex offenses. Examining just the most violent offenses (that is, murder, manslaughter, or attempted murder), 30% of the 20 subjects in the sample charged with these crimes carried a DSM-III-R diagnosis of organic brain disorder.

When patients with chart indicators of potential brain impairment (that is, head injury with loss of consciousness, seizure disorder, cognitive impairment, and neurodiagnostic findings) were combined with the subjects with an organic DSM-III-R diagnosis, a significant association between these measures and indictment for violent crimes was obtained (Fischer's exact test, p = 0.01). Of the subjects indicted for violent crimes, 52.3% had a diagnosis or history suggesting organic deficits while none of the subjects indicted for nonviolent crimes had similar findings. This result is tempered however, by the relatively small proportion of nonviolent offenders in the sample.

No significant associations were observed between neurological impairment (hard signs, soft signs, or both) and violent crime in this sample. It is also notable that the significant findings between other indicators of brain disorder and violence disappear when the neurological impairment data are included. The high prevalence of neurological findings among all the offenders in the sample resulted in limited variation between the groups. The high rate of abnormal neurological findings may reflect the sensitivity of the neurological exam to detect subtle forms of impairment, which may be unrelated to violent criminal behavior. Alternatively, the relationship between neurological impairment and violent behavior may be reflected in lateralization patterns of neurological deficit.³

Discussion

Results from this study suggest that a substantial majority of male forensic psychiatric patients exhibit evidence of potential organic brain abnormalities, ranging from fairly subtle findings to evidence of profound brain impairment. Cumulative prevalence estimates show that roughly two thirds of the subjects in this random sample (64%) have multiple indicators suggesting compromised brain functioning. Eighty-four percent of the subjects had at least one indicator of potential brain impairment, yet only 16% of the cases carried a DSM-III-R diagnosis reflecting organicity. This finding underscores the need for detailed clinical examinations of functional brain integrity in the majority of clinical forensic evaluations.

While these findings appear to support biological views regarding the etiology of violent behavior, they also suggest the need for further research. Impaired brain functioning may prove to be a risk factor for violent behavior among the mentally ill, but more careful studies are needed to document this relationship adequately. Future research needs to explore the role of localized brain dysfunction in violent behavior and its interaction with other personal and environmental risk factors. Investigations into the role of drug and alcohol abuse, psychopathology, situational stressors, and socialization environment may all make important contributions to our understanding of the relationship between brain dysfunction and various types of criminal violence in mentally disordered offenders.

The present study also has certain intrinsic limitations. Chart review methods provide admittedly gross and insensitive measures of true brain damage. With the exception of the quantitative neurological scale data, few of these indicators can document the location or extent of actual brain lesions. The neurological scale data, while more sensitive, are limited by the fact that a number of the subjects refused to consent to examination. Informed consent procedures often introduce bias affecting the representativeness of randomly selected samples of patients. While chart review methods overcome the problem of patient refusal rates, they are limited by the quality, reliability, and comprehensiveness of the hospital record. Finally, this study does not include data on organic impairment

³D. A. Martell, A. Convit, and C. Evangelista, "Lateralized Brain Dysfunction and Violent Behavior in a Maximum-Security Hospital for the Criminally Insane," unpublished manuscript, Nathan S. Kline Institute for Psychiatric Research and the Department of Psychiatry, New York University Medical Center, New York, NY, 1990.

in female forensic psychiatric patients. It remains to be determined if women exhibit commensurate rates of organic brain dysfunction.

Despite these methodological limitations however, these findings strongly suggest the need for further study. Future research is clearly required to determine the prevalence and extent of brain dysfunction in forensic psychiatric populations with greater accuracy and specificity.

Implications for Forensic Evaluation and Treatment

To the extent that these findings accurately reflect the potential prevalence of brain disorders in mentally disordered offenders, they have important implications for applied forensic clinical work. Careful attention to brain integrity may prove fruitful for forensic psychiatric evaluation and diagnostic procedures, forensic psychiatric treatment, and forensic psychiatric decision making.

Evaluation and diagnosis—As these data suggest, indications of organic brain damage may sometimes be underemphasized or overlooked in diagnostic evaluations of forensic psychiatric patients. Many male patients with nonorganic psychiatric diagnoses exhibit neurological abnormalities or have histories of severe head injury, seizure disorder, or cognitive impairment. Most DSM-III-R diagnoses require the clinician to rule out "organic factors" in the initiation or maintenance of the patient's behavioral disturbance. To the extent that brain abnormality may play a contributory role in the etiology of violent behavior, careful differential diagnosis including special attention to brain integrity would benefit routine forensic clinical evaluations.

Appropriate attention to brain function might include a structured neuropsychological interview, with attention to perinatal and birth complications, developmental milestones, history of physical abuse, learning disabilities, academic achievement, head injuries, seizures, hypertension, and other relevant historical data. A careful neurological exam could also be important. When brain impairment is suspected, referral for further neuropsychological or neurodiagnostic testing or both would be indicated prior to arriving at a final diagnostic formulation.

Treatment—Beyond diagnosis, knowledge of organic brain disorder has important implications for forensic psychiatric treatment. Brain dysfunction can have a significant impact on a patient's response to many forms of treatment and lack of attention to brain integrity can result in counterproductive, iatrogenic outcomes. Conversely, knowing that a patient is brain damaged raises another set of issues. The often irreversible nature of brain damage can lead to frustration on the part of treatment providers, who may feel limited in their ability to effect change in an organic patient. However, it is a mistake to equate organicity with untreatability.

Special attention has been paid to issues of the treatment of violent and aggressive behavior in the presence of brain impairment, beginning with psychopharmacological intervention. Brain damaged patients are often significantly more sensitive to the side effects of psychotropic medications, and generally warrant lower doses and close monitoring [52]. In some cases, psychiatric medications may be counterproductive and would be contraindicated. In other cases there are specific medications such as Tegretol[®] and the beta-blocker propranolol, which can be effective in managing violent or aggressive behavior secondary to brain impairment [53,54]. These issues are treated in much greater detail by Hales and Yudofsky [52].

Specialized psychological and behavioral intervention strategies have also been shown to be effective [55]. Eames and Wood [56] report on a behavioral modification approach with a 75% effectiveness rate in treating violent behavior associated with brain impairment. This is a treatment modality that seems to hold particular promise for the treatment

of mentally disordered offenders with brain damage. Psycho-educational and behavioral self-control approaches can help these patients to understand the nature and extent of their impairment, and the way it manifests itself in their violent or aggressive behavior or both. Helping patients learn to recognize warning signs of impending aggression (internal cues and external stressors that precede violent acting out) is important to teaching behavioral self-control. Teaching them to compensate for their limitations by recognizing and capitalizing on their relative strengths characterizes subsequent intervention strategies.

Forensic Psychiatric Decision-Making—Finally, these issues come together in forensic psychiatric decision-making around the issue of future dangerousness. These are daily decisions in forensic clinical work, and they govern a range of outcomes from patient management to transfer and release decisions. Recognizing the role of brain dysfunction in assessing a patient's potential for dangerousness should be a critical component of this decision-making process. The data gathered through interventions (that is, the efficacy of medication, the degree of insight the patient has into their organicity, and their demonstrated ability or inability to exhibit appropriate behavioral self-control) can prove to be important and helpful in making these difficult decisions.

Reconstructing a patient's history with careful attention to the confluence of central nervous system (CNS) damage and violent behavior is one useful approach. Poor judgement, impulsivity, and unprovoked explosive rage attacks often characterize the violent behavior of neurally compromised offenders. In some cases violent behavior appears only after a brain injury while in other cases brain injury can serve to diminish or eliminate violent behavior. Still other cases may exhibit a developmental pattern suggestive of minimal brain dysfunction from childhood. Such cases are characterized by a long history of undercontrolled aggressive behavior beginning with attention deficit/hyperactivity disorder in childhood and may continue into adulthood in the form of aggressive or violent criminal behavior or both.

Conclusions

These data suggest that organic brain dysfunction may be present in a significant majority of male forensic psychiatric patients. Despite their biological implications for violent behavior, indications of organic brain dysfunction are too often overlooked in forensic clinical work. The issue of organic brain impairment requires greater attention from both researchers and clinicians concerned with the diagnosis, treatment, and management of mentally disordered offenders.

Acknowledgment

The author is indebted to Antonio Convit, M.D., for the neurological scale data. Thanks to Park Dietz, M.D., Ph.D.; Joel Dvoskin, Ph.D., Richard Rosner, M.D.; and Renate Wack, for their helpful comments on earlier drafts of this article.

References

- [1] Rosner, R., Wiederlight, M., Horner-Rosner, M. B., and Wieczorek, R. R., "An Analysis of Demographic Variables in Adolescent Defendants Evaluated in a Forensic Psychiatry Clinic," Bulletin of the American Academy of Psychiatry and the Law, Vol. 4, No. 3, 1977, pp. 251– 257.
- [2] Harmon, R. B., Rosner, R., and Wiederlight, M., "The Mariel Refugee and the New York Criminal Court," Journal of Forensic Sciences, Vol. 32, No. 3, May 1987, pp. 725-275.
- [3] Bonheur, H. H. and Rosner, R., "Sex Offenders: Diagnosis, Organicity, and Intelligence," Journal of Forensic Sciences, Vol. 26, No. 4, Oct. 1981, pp. 782-792.

- [4] Bonheur, H. H., "Psychodiagnostic Testing of Sex Offenders: A Comparative Study," Journal of Forensic Sciences, Vol. 28, No. 1, Jan. 1983, pp. 49-60.
- [5] Dupre, J., "Organic Brain Disorders and Violent Behavior," American Journal of Forensic Psychiatry, Vol. 11, No. 1, 1990, pp. 71-79.
- [6] Hamstra, B., "Neurobiological Substrates of Violence: An Overview for Forensic Clinicians," Journal of Psychiatry and Law, Fall-Winter 1986, pp. 349-374.
- [7] Odejide, A. O., "Some Clinical Aspects of Criminology: A Study of Criminal Psychiatric Patients at the Lantoro Psychiatric Institution," Acta Psychiatrica Scandinavia, Vol. 63, No. 3, 1981, pp. 208–224.
- [8] Häfner, H., and Böker, W., Crimes of Violence by Mentally Abnormal Offenders: A Psychiatric and Epidemiological Study in the Federal German Republic, Cambridge University Press, Cambridge, 1982.
- [9] Elliott, F. A., "Neurological Findings in Adult Minimal Brain Dysfunction and the Dyscontrol Syndrome," Journal of Nervous and Mental Disease, Vol. 170, No. 11, 1982, pp. 680-687.
- [10] Lewis, D. O., Pincus, J. H., Feldman, M., Jackson, L., and Bard, B., "Psychiatric, Neurological, and Psychoeducational Characteristics of 15 Death Row Inmates in the United States," American Journal of Psychiatry, Vol. 143, 1986, pp. 838-845.
- [11] Gorenstein, E. E. and Newman, J. P., "Disinhibitory Psychopathology: A New Perspective and a Model for Research," Psychological Review, Vol. 87, No. 3, 1980, pp. 301-315.
- [12] Yeudall, L. T., Fedora, O., and Fromm, D., "A Neuropsychosocial Theory of Persistent Criminality: Implications for Assessment and Treatment," in Advances in Forensic Psychology and Psychiatry, Vol. 2, R. W. Rieber, Ed., Ablex Publishing, Norwood, NJ, 1987, pp. 119-191.
- [13] Tancredi, L. R. and Volkow, N., "Neural Substrates of Violent Behavior: Implications for Law and Public Policy," International Journal of Law and Psychiatry, Vol. 11, 1988, pp. 13-49.
- [14] Weiger, W. A. and Bear, D. M., "An Approach to the Neurology of Aggression," Journal of Psychiatric Research, Vol. 22, No. 2, 1988, pp. 85-98.
- [15] Langevin, R., Ben-Aron, M., Wortzman, G., Dickey, R., and Handy, L., "Brain Damage, Diagnosis, and Substance Abuse among Violent Offenders," Behavioral Sciences and the Law, Vol. 5, No. 1, 1987, pp. 77–94.
- [16] Nachshon, I. and Denno, D., "Violent Behavior and Cerebral Hemisphere Function," in The Causes of Crime: New Biological Approaches, S. A. Mednick, T. E. Moffitt, and S. A. Stack, Eds., Cambridge University Press, New York, 1987, pp. 185–217. [17] Silver, J. M. and Yudofsky, S. C., "Aggressive Behavior in Patients with Neuropsychiatric
- Disorders," Psychiatric Annals, Vol. 17, No. 6, 1987, pp. 367-370.
- [18] Alexander, M. P., "Traumatic Brain Injury," in Psychiatric Aspects of Neurologic Disease, Vol. 2, D. F. Benson and D. Blumer Eds., Grune and Stratton, New York, 1982, pp. 219-249.
- [19] Reitan, R. M. and Wolfson, D., Traumatic Brain Injury. Volume 1: Pathophysiology and Neuropsychological Evaluation, Neuropsychology Press, Tucson, AR, 1986.
- [20] Heinrichs, R. W., "Frontal Cerebral Lesions and Violent Incidents in Chronic Neuropsychiatric Patients," Biological Psychiatry, Vol. 25, 1989, pp. 174-178.
- [21] Kling, A., "Frontal and Temporal Lobe Lesions and Aggressive Behavior," in Issues in Brain/ Behavior Control, W. Smith and A. Kling, Eds., Spectrum Publications, New York, 1976.
- [22] Volkow, N. D. and Tancredi, L., "Neural Substrates of Violent Behavior: A Preliminary Study with Positron Emission Tomography," British Journal of Psychiatry, Vol. 151, 1987, pp. 668-673.
- [23] Will, T. E., "Temporal Lobe Neuropsychological Dysfunction among Violent Psychiatric Inpatients," Dissertation Abstracts International, Vol. 49, No. 3, 1986, pp. 923-924.
- [24] Wood, R. L., "Behavior Disorders Following Severe Brain Injury: Their Presentation and Psychological Management," in Closed Head Injury: Psychological, Social, and Family Consequences, N. Brooks, Ed., Oxford University Press, New York, 1984.
- [25] McKinlay, W. W., Brooks, D. N., and Bond, M. R., et al., "The Short-Term Outcome of Severe Blunt Head Injury as Reported by the Relatives of the Injured Person," Journal of Neurology, Neurosurgery, and Psychiatry, Vol. 44, 1981, pp. 527-533. [26] Petursson, H. and Gudjonsson, G. H., "Psychiatric Aspects of Homicide," Acta Psychiatrica
- Scandinavia, Vol. 64, 1981, pp. 363-372.
- [27] Devinsky, O. and Bear, D., "Varieties of Aggressive Behavior in Temporal Lobe Epilepsy," American Journal of Psychiatry, Vol. 141, No. 5, 1984, pp. 651-656.
- [28] Lewis, D. O. and Pincus, J. H., "Epilepsy and Violence: Evidence for a Neuropsychotic-Aggressive Syndrome," Journal of Neuropsychiatry, Vol. 1, No. 4, 1989, pp. 413-418.
- [29] Stevens, J. R. and Hermann, B. P., "Temporal Lobe Epilepsy, Psychopathology, and Violence: The State of the Evidence," Neurology, Vol. 31, 1981, pp. 1127-1132.

- [30] Stone, A., "Violence and Temporal Lobe Epilepsy," American Journal of Psychiatry, Vol. 141, No. 12, 1984, p. 1641.
- [31] Furguson, S. M., Rayport, M., and Corrie, W. S., "Brain Correlates of Aggressive Behavior in Temporal Lobe Epilepsy," in *The Limbic System: Functional Organization and Clinical Disorders*, B. Doane and K. Livingston, Eds., Raven Press, New York, 1986, pp. 183-193.
- [32] Gunn, J. and Bonn, J., "Criminality and Violence in Epileptic Prisoners," British Journal of Psychiatry, Vol. 118, 1971, pp. 337-343.
- [33] Monroe, R. R., "Brain Dysfunction in Prisoners," in Violence and the Violent Individual, R. Hays, T. Roberts, and K. Solway, Eds., SP Medical and Scientific Books, New York, 1981, pp. 75-86.
- [34] Stone, A., "Violence and Temporal Lobe Epilepsy," American Journal of Psychiatry, Vol. 141, No. 12, 1984, p. 1641.
- [35] Monroe, R. R., "Épisodic Behavioral Disorders and Limbic Ictus," in *The Limbic System: Functional Organization and Clinical Disorders*, B. Doane and K. Livingston, Eds., Raven Press, New York, 1986, pp. 251-266.
- [36] Rickler, K. C., "Episodic Dyscontrol," in Psychiatric Aspects of Neurologic Disease, Vol. 2, D. F. Benson and D. Blumer, Eds., Grune and Stratton, New York, 1982, pp. 49-73.
- [37] Bear, D., Freeman, R., and Greenberg, M., "Interictal Behavior in Patients with Temporal Lobe Epilepsy," in *Psychiatric Aspects of Temporal Lobe Epilepsy*, American Psychiatric Association, Washington, DC, 1984.
- [38] Day, K., "Psychiatric Disorder in Middle Aged and Elderly Mentally Handicapped," British Journal of Psychiatry, Vol. 147, 1985, pp. 660-667.
- [39] Reid, A. H., Ballinger, B. R., Heather, B. B., et al., "The Natural History of Behavioral Symptoms among Severely and Profoundly Mentally Retarded Patients," *British Journal of Psychiatry*, Vol. 145, 1984, pp. 289–293.
- [40] Bryant, E. T., Scott, M. L., Golden, C. J., and Tori, C. D., "Neuropsychological Deficits, Learning Disability, and Violent Behavior," *Journal of Consulting and Clinical Psychology*, Vol. 52, No. 2, 1984, pp. 323-324.
- [41] Kunce, J. T., Ryan, J. J., and Eckelman, C. C., "Violent Behavior and Differential WAIS Characteristics," *Journal of Consulting and Clinical Psychology*, Vol. 44, No. 1, 1976, pp. 42– 45.
- [42] Yeudall, L. T., "Neuropsychological Assessment of Forensic Disorders," Canadian Mental Health, Vol. 25, 1977, p. 7.
- [43] Yeudall, L. T. and Fromm-Auch, D., "Neuropsychological Impairment in Various Psychopathological Populations." in *Hemisphere Asymmetries of Function in Psychopathology*, J. Gruzelier and P. Flor-Henry, Eds., Elsvier/North-Holland, Amsterdam, 1979.
- [44] Yeudall, L. T. and Wardell, D. M., "Neuropsychological Correlates of Criminal Psychopathy. Part II: Discrimination and Prediction of Dangerous and Recidivistic Offenders," in *Human* Aggression and its Dangerousness, L. Buliveau, C. Canepa, and D. Szabo, Eds., Pinel Institute, Montreal, 1978.
- [45] Spellacy, F., "Neuropsychological Discrimination between Violent and Nonviolent Men," Journal of Clinical Psychology, Vol. 34, No. 1, 1978, pp. 49–52.
- [46] West, L. Y., "Discrimination of Violent and Non-violent Inmates with the Standardized Luria-Nebraska Neuropsychological Battery," *Dissertation Abstracts International*, Vol. 42, No. 10, 1981, p. 4218.
- [47] Monroe, R. R., Hulfish, B., Balis, G., Lion, J., Rubin, J., et al., "Neurological Findings in Recidivistic Aggressors," in *Psychopathology and Brain Dysfunction*, C. Sahass, S. Gershon, and A. J. Friedhoff, Eds., Raven, New York, 1977.
- [48] Convit, A., Jaeger, J., Lin, S., Meisner, M., and Volavka, J., "Prediction of Assaultiveness in Psychiatric Inpatients: A Pilot Study," *Hospital and Community Psychiatry*, Vol. 39, 1988, pp. 429-434.
- [49] Krakowski, M. I., Convit, A., Jaeger, J., Lin, S., and Volavka, J., "Neurological Impairment in Violent Schizophrenic Inpatients," *American Journal of Psychiatry*, Vol. 146, No. 7, 1989, pp. 849-853.
- [50] Krakowski, M. I., Convit, A., and Volavka, J., "Patterns of Inpatient Assaultiveness: Effect of Neurological Impairment and Deviant Family Environment on Response to Treatment," *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, Vol. 1, No. 1, 1988, pp. 21– 29.
- [51] Wechsler, D., WAIS-R Manual: Wechsler Adult Intelligence Scale, Revised, The Psychological Corp., New York, 1980.
- [52] Hales, R. E. and Yudofsky, S. C., Textbook of Neuropsychiatry, American Psychiatric Association Press, Washington, DC, 1987.
- [53] Silver, J. M. and Yudofsky, S. C., "Propranolol for Aggression: Literature Review and Clinical Guidelines," *International Drug Therapy Newsletter*, Vol. 20, 1985, pp. 9-12.

- [54] Silver, J. M. and Yudofsky, S. C., "Propranolol in the Treatment of Chronically Hospitalized Violent Patients," in C. Shagass, R. C. Josiassen, W. H. Bridger, et al., Eds., *Biological Psychiatry*, Elsvier, New York, 1986.
- Psychiatry, Elsvier, New York, 1986.
 [55] Liberman, R. P. and Wong, S. E., "Psychiatric Uses of Behavioral Analysis and Therapy Procedures Related to Seclusion and Restraint," in *The Psychiatric Uses of Seclusion and Restraint*, K. Tardiff, Ed., American Psychiatric Association Press, Washington, DC, 1984.
- [56] Eames, P. and Wood, R., "Rehabilitation after Severe Brain Injury: A Follow-Up Study of a Behavior Modification Approach," *Journal of Neurology, Neurosurgery, and Psychiatry*, Vol. 48, 1985, pp. 613–619.

Address requests for reprints or additional information to Daniel A. Martell, Ph.D. Research Department Kirby Forensic Psychiatric Center Ward's Island, NY 10035

Erratum

In the article, "The Trial of Louis Riel: a Study in Canadian Psychiatry" (Vol. 37, No. 3, May 1992, p. 852), I erred in stating that Valentine Shortis was found not guilty of homicide, a verdict supported by the cabinet. In actuality, the insanity defense failed and Shortis was sentenced to death. The cabinet was evenly split over a recommendation for clemency. The Governor General, Lord Aberdeen, then commuted Shortis to "imprisonment for life as a *criminal lunatic* (italics mine), or otherwise as may be found fitting." This action exacerbated the discontent of French-Canadians over the Riel case. This decision in the Shortis case may have been a factor in the election of a Liberal, Wilfrid Laurier, who became the first French-Canadian prime minister of Canada in 1986.

Shortis remained incarcerated for 42 years; in the earlier years, he was frequently described as mentally ill. In his later years, he apparently functioned quite well and was released at age 62 in 1937; in 1941 he died suddenly of a heart attack.

Both the Jackson and Shortis cases reflect the fact that Canadian authorities were not adverse to considering the impact of mental illness in deciding the disposition of offenders, a step that was rejected in the Riel case.

I wish to thank Abraham L. Halpern, M.D., for bringing this error to my attention.

Irwin N. Perr, MD, JD

Erratum

The articles that appeared in the May issue of the journal under the Psychiatry and Behavioral Science Section Awards were erroneously labeled Case Reports on the title page.