COMA AND THE ETIOLOGY OF VIOLENCE, PART 2

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Coma and the etiology of violence are explored by the author through a review of the literature. Animal studies, post-traumatic psychic disorder studies, post-traumatic anger and violence studies, tumor and lesion of the limbic system studies, temporal lobe epilepsy studies, episodic dyscontrol syndrome studies, and minimal brain dysfunction studies were reviewed in Part 1, which was published in the December 1986 issue of the Journal. Part 2 concludes the review with clinical surveys on violent individuals and studies on clinical treatment of violence.

These studies reveal the etiologic significance of central nervous system dysfunction in the production of violent behavior. Because central nervous system factors are involved in some instances of violent behavior, physicians clearly have a role in the early identification of potentially violent subjects and in the intervention or treatment of individuals who have been violent toward others. Studies have consistently found that lower socioeconomic groups are more predisposed to brain injury from trauma, and several studies have indicated that this is true for segments of the black community. Therefore, investigations in the relationship between central nervous system injury and violence should be a major goal of the black community. Black physicians should assume a lead role in these inquiries and in the prevention and treatment of violence, specifically black-on-black murder.

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There have been several studies on psychopathic or delinquent children and adolescents who have exhibited violent behavior and who have been brought to the attention of psychiatrists.

CLINICAL SURVEYS OF VIOLENT INDIVIDUALS

In 1925 Healy[110] reported on 4,000 cases of delinquency, noting cases in which a change of personality had directly followed a severe concussion. Further, he reported that there were a larger number of injuries to the head among delinquents than among nondelinquents, and that 3.5 percent of his sample had an injury to the head of some severity, each having suffered at least a period of unconsciousness. Kasanin[111] found (in 5,000 cases at the Judge Baker Foundation) that among 120 cases diagnosed as psychopathic personalities, 10 percent of these had had a serious brain injury during childhood and adolescence and conduct disorder followed the trauma. Bender[51] studied 33 children and adolescents who had killed; she found that in the 19 subjects who had stabbed, repeatedly struck, or shot their victims, 16 of these had schizophrenia, brain disease and/or epilepsy. The final diagnostic count of these cases was: three were intellectually defective, 12 were schizophrenic (nine recognized in childhood and three recognized first in adulthood; not one was diagnosed as schizophrenic at the time of the incident), three were epileptics, seven had chronic brain syndromes without epilepsy but impulsive disorders, and ten had psychoneurotic depressions reactive to the situation.

Lewis and her colleagues[112-116] have done extensive research on violence in children and found several interesting trends relevant to this review. In one study[112] she reported on 285 children referred to juvenile court and found psychomotor epileptic symptoms in 18 (6 percent); this finding

was frequently associated with aggressive behaviors and offenses. These patients had episodes of loss of fully conscious contact with reality and long staring spells or falling; 16 of the 18 also had paranoid symptomatology in the form of delusions. Eleven of the 14 available electroencephalograms (EEGs) showed an abnormality but only three had a temporal lobe focus. She notes a striking finding of serious trauma to the central nervous system in 15 of the 18.

In another study[113] Lewis et al found incarcerated adolescents had significantly more accidents and injuries, specifically head or face injuries than hospitalized adolescents. In addition, she found 18 percent of the violent incarcerated male adolescents she studied had psychomotor seizures.[114]

In a later study Lewis[115] and her colleagues studied 55 children; of these, 21 were homicidally aggressive. The most significant behavior distinguishing the homicidally aggressive from the nonhomicidally aggressive children was suicidal behavior (57 vs 23 percent). Of the 21 homicidal children 48 percent definitely had a history of seizures (many had experienced febrile seizures) compared with 7 percent of 30 nonhomicidal children. (Further, an additional 10 percent of the homicidal children probably had seizures). There was also a tendency for more of the homicidally aggressive children to have had a history of head injury (57 vs 30 percent) and to have averaged a greater number of head injuries as well. In 62 percent of the households that produced these violent children, the fathers had been physically violent toward the mothers compared with only 13 percent in the nonhomicidal children. Alcoholism was also significantly more common in the fathers of homicidally aggressive children (52 vs 10 percent). The most significant factor distinguishing mothers of homicidally aggressive children from nonhomicidally aggressive children was a history of psychiatric hospitalization (43 and 7 percent, respectively).

In a prospective study of children who murder, Lewis and her colleagues[116] noted five characteristics (history of extreme violence, psychotic illness in a first-degree relative, witness or victim of violence in the family, psychotic symptoms, and major neurologic impairment) that taken collectively significantly differentiated nine children (six had severe head injury) who went on to murder from those 24 who did not. Lewis and her colleagues[116] speculate that the prevalence of head trauma, perinatal problems, or seizures in their samples indicates a problem of central nervous system dysfunction—the kind often associated with lability of mood and impulsivity. The theory is that the central nervous system dysfunction, combined with a vulnerability to psychotic disorganization, contributes to children's self-destructive and homicidally aggressive behaviors.

Malmquist[117] studied 20 adolescents charged with murder and found suicidal preoccupations may be common the week before the homicide. Inamdar and her colleagues[118] studied violent and suicidal behavior in psychotic adolescents and found that 27.5 percent had been both violent and suicidal, with male subjects significantly more likely to be violent and suicidal. These findings parallel a study by Whitlock and Broadhurst[119] that compared 50 suicidal patients, 50 nonsuicidal psychiatric patients, and 50 healthy persons and found that suicidal patients had significantly higher violence indicies than either controls. The relationship between suicidal and homicidal impulses is also seen in the finding that 12 to 18 percent of the calls to suicide hotlines are in fact calls related to homicidal impulses.[6] Of interest to this review is the finding that suicidal and homicidal impulses have been correlated in blacks,[120] and at least one study notes a significant prevalence of head injury (14 percent) occurring within the year of a completed suicide in a black population.[121]

In clinical studies of prison inmates who have committed violent crimes, neural factors in the generation of this violence have been implicated. Gunn and Bonn[122] found that the prevalence of epilepsy in prison was several times greater than in the general population and noted that other studies found that murderers were more prone to epilepsy than the general population. A representative sample of epileptic prisoners was no more criminally violent than a matched control group of prisoners. However, this study found epileptics to be more impulsive than controls. Thus, their finding of greater impulsivity in epileptic prisoners fits with previous observations that individuals with abnormal EEGs are more likely to commit motiveless homicidal violence.

Hill[123] has written on "epileptoid" features in psychopathic personalities that cause abnormal irritability, resulting in attacks of rage with poor reflection, a twilight state, and markedly clouded

memory; jealousy and alcohol are the precipitants. He notes that 65 percent of the aggressive psychopaths (patients whose histories repeatedly show hostile acts against individuals and a tendency to self-injury) he studied had abnormal EEGs, and 33 percent had had head injuries. However, Hill notes EEG abnormalities in aggressive persons are independent of previous head injury, and that, although head injury can produce aggressiveness and dysrhythmia, these results are probably not related; he feels dysrhythmia is more common in the naturally aggressive psychopath than in the psychopath who is aggressive as a result of head injury.[124] Hill[125] also studied 110 murderers and found that they had 11 percent more theta-wave excess and 6.2 percent more post-temporal slow wave focus than controls. Of these murderers, there were ten with temporal lobe foci, and nine of these were in the posterior regions.

Williams[126] studied 1,250 prisoners; over one half were incarcerated for a crime of major violence (murder, attempted murder, inflicting bodily harm, and rape or attempted rape) and the remaining prisoners committed crimes that had included bodily violence such as robbery with violence. A random sample of 333 was closely studied. It was found that the EEGs of the habitually aggressive inmates were abnormal in 65 percent of the cases, which compared with 24 percent of inmates who had committed major violent crimes but were not habitually aggressive and with 12 percent of the inmate population at large. When those who were mentally retarded, had epilepsy, or who had had a major head injury were removed from the tally, it was found that the EEG was abnormal in 57 percent of the habitual aggressives, but only in 12 percent of the nonhabitually aggressive inmates who had committed a major violent crime (the same as the population at large). The EEG abnormalities in the habitual aggressives were seen bilaterally in 64 percent of the cases and involved the frontal cortex. In addition, the temporal lobes were affected in all (the anterior part three times as often as the posterior), while rhythms known to be associated with temporal lobe dysfunction (theta wave, 4 to 7 second) were present in 80 percent.

Another study of insane murderers showed that these individuals had EEG abnormalities about four times more often than the control group.[127] Stafford-Clark and Taylor[128] studied 64 murderers and found that the murders could be grouped in the following manner: (1) murder incidental to the commission of another crime, (2) a clear motive for the murder or intended violence during the commission of another crime, (3) essentially motiveless murders, (4) murders with a strong sexual element, and (5) murders in which the murderer was insane. In groups 1 and 2, there was a preponderance of normal over abnormal EEGs, but in the unmotivated murders (group 3) the reverse was true. There was a marked preponderance of abnormal records in groups 3 (73 percent) and 5 (86 percent), which strongly suggests that whatever the cerebral dysfunction that such abnormal records indicate, it is significantly correlated with a capacity to commit violent acts of an apparently motiveless character. In contrast, group 1 (the "accidental murder" group) contains less than 10 percent of abnormal records, which approximates the proportion in the general prison population.

Other miscellaneous clinical studies on criminal populations include the work by Gibbons, Pond, and Stafford-Clark[129] who noted that nearly one half of the criminal psychopaths studied who had suffered prior head injury were originally convicted for violent offenses, which was a much higher proportion than in psychopaths without complications, or psychopaths with epilepsy.

Frazier[130] studied 23 single murderers and eight multiple murderers and found organic brain factors in four single and two multiple murderers (temporal lobe epilepsy in two single murderers, mental retardation in two single murderers, and organic brain syndrome caused by neoplasm in one multiple murderer, and early dementia from central nervous system infection in another). He noted episodic dyscontrol was present in eight single and three multiple murderers, and episodic psychosis without external reality testing was present in 15 murderers (three of whom were multiple murderers).

Finally, Langevin et al[131] studied sexually aggressive inmates and, though they found a nonsignificant difference between sexually aggressive inmates who had temporal lobe pathology (30 percent) compared with controls who had temporal lobe pathology (11 percent), they did find that the sadistic sexually aggressive inmates had significantly more temporal lobe pathology (56 percent) compared with nonsadist sexual aggressive inmates (0 percent). One third of the sadists had indices over the cutoff point for significant brain

damage compared with 8 percent of the nonsadist sexually aggressive inmates and 18 percent of the controls.

Clinical studies of patients who come to emergency rooms for treatment because of violent tendencies have already been discussed in the Episodic Dyscontrol Syndrome (Part 1) section. However, a study by Climent and Ervin[132] has particular relevance to this review. These authors compared 40 violent emergency room patients with 40 controls and found that violent subjects were more likely to have been beaten as children and more likely to have owned and used knives. Alcoholism in the father and mother was more common in the parents of the violent group. Of the 19 violent subjects who reported head injuries before 15 years of age, nine reported unconsciousness along with the injury; in contrast, in the control group only three (of the 12 who also reported head injury before the age of 15) reported unconsciousness. Thus, significantly more violent subjects reported head injury with unconsciousness (the severity of injury being judged by coma). Five of the violent subjects reported convulsions before the age of 10 compared with none in the control group. After 10 years of age, 15 of the violent subjects suffered from convulsions as opposed to none in the non-violent group. The violent subjects also suffered significantly more headaches. The authors hypothesize that these findings point to the likelihood of violent behavior being influenced by organic brain disease. Violent subjects also had more suicidal thoughts.

STUDIES ON CLINICAL TREATMENT OF VIOLENCE

Lion, Bach-y-Rita, and Ervin[133,134] raised significant questions to determine which patients exhibited violent behavior that might have been caused by a neurologic disorder. These questions included the following: Were there symptoms such as headaches or altered states of consciousness that accompanied the violent act or impulsiveness? Are there subtle personality changes over time (implying neoplasm)? Is there a marked alteration in sexual function or memory implying temporal lobe process? Is there a past history of infection such as meningitis or head injury that may have led to brain impairment? Is there an impairment of intellectual ability or a history of learning difficulty? Is there a family history of epilepsy or a history of convulsions in childhood? Do aura or seizure-like states exist? Is there a history of repetitive rage reactions or periodically reoccurring temper tantrums that suggest temporal lobe epilepsy? Similarly, Elliott[97] suggested asking: Do you have difficulty in controlling your temper? Have you been charged with traffic violations for dangerous driving? Are you especially sensitive to alcohol? Clearly, such questions asked during a standard review of systems would tend to identify patients who are at risk for violent behavior from central nervous system causes. Further, physicians who routinely asked for a history of assault could uncover families of violence and could initiate appropriate interventions—sociologic, interpersonal, intrapsychic, or biologic.

Lion[135] notes that aggression is a symptom that may stem from psychosis—severe character disturbances of the explosive, antisocial, or passive-aggressive types (impulsivity is a common feature in all of these types and their violence is usually episodic and paroxysmal, indicating organic factors may or may not underlie such problems)—hypersexuality, depression, or mania. He further notes that various types of medication may alter these causes of violence.

As previously reported, several authors have noted gross clinical psychopathology (usually schizophrenia) in murderers.[11,21,51,116] Although there is considerable controversy about the dangerousness of mentally ill patients,[136-139] it is important to note that several authors have indicated the efficacy of antipsychotic medication in treating aggression stemming from psychosis.[21,140-142]

Lithium has been noted to reduce aggression in several animal studies.[21,143] Sheard[144] studied 12 inmates in a maximum security prison who were selected on the basis of a prehistory of three or more episodes of violent assaultive crimes, prison behavior characterized by continuing verbal and physical aggressive behavior, and high scores on aggressive scales. He found a significant reduction in aggressive affect (self-rated) and a reduction in the number of tickets received for physical or verbal aggression secondary to treatment with lithium. Tupin et al[145] studied 27 male convicts who had a pattern of recurrent violent behavior (in and outside of prison) and who reacted rapidly to slight provocation with anger or violence. They found several other characteristics commonly

encountered in the patients' medical profiles, such as a history of brain injury, nonspecific abnormal EEGs, and mixed psychiatric diagnoses (eight schizophrenics, four possible schizophrenics, 12 explosive personalities, and three others). These authors found a significant decrease in the mean number of disciplinary actions for violence for identical time periods before and after lithium. Prisoners reported a decrease in aggressive feelings, an increased capacity to reflect on consequences of action, an increased capacity to control angry feelings when provoked, diminished intensity of angry affect, and a more generally reflective mood. Staff also consistently reported a decrease in aggressive behavior. Sixteen of the 21 who were judged improved had an abnormal EEG or a history suggestive of brain damage (including head trauma with unconsciousness, or known central nervous system disease (eg, meningitis or epilepsy). Morrison et al[146] studied 20 inpatients (11 with frequent seizures, abnormal EEGs, and hyperaggressiveness; two with hyperaggressive behavior but normal EEGs; seven with EEGs showing 14/6 per second positive spiking but without clinical seizures) and found that 15 inpatients treated with lithium showed a reduction in the frequency of hyperaggressiveness, assaultive behavior, a decreased tendency to quarrel, and greater self-control when provoked. Finally, Rifkin et al[147] also reported that lithium aids overaggressive patients with character disorders.

As a result of the findings that associate EEG abnormalities with violence, several authors have investigated the use of anticonvulsants in the treatment of violent subjects. Several authors[148,149] have discussed the efficacy of carbamazepine in violent patients with abnormal limbic system discharges and temporal lobe pathology with sham rage attacks. Monroe[150] and his colleagues[151,152] have noted a significant number of violent acts are committed by individuals in whom central nervous system instability can be demonstrated by special EEG activation procedures using alpha chloralose as the activating agent. This instability is suggested by the circumscribed ictal phenomenon in the limbic system demonstrated on subcortical EEGs. These authors note that chlordiazepoxide or primidone led to dramatic improvement in 23 of 55 acutely disturbed patients with episodic, impulsive, aggressive behavior. Maletzky[95] reported on 19 of 22 subjects with episodic dyscontrol syndrome who responded favorably to phenytoin.

Elliott[153] reported on seven patients who had belligerence secondary to acute brain damage that was successfully controlled with propranolol. Yudofsky and his colleagues[154-156] have made several reports on the usefulness of propranolol in treating rage that occurs secondary to organic brain damage. One study[154] indicated that propranolol controlled the rage and violent outbursts in four patients with irreversible chronic organic brain syndromes. A second more extensive study[155] examined 30 children and adolescents with uncontrolled rage outbursts and organic brain dysfunction (nine with minimal brain dysfunction, eleven with uncontrolled seizures and three others with definite neurogenic seizure histories, four with anti-convulsant medication for suspected seizures) and found that over 75 percent of the patients showed either moderate or marked improvement in response to propranolol treatment, although propranolol did not do much for the seizures. Finally, these authors[156] presented a case of rage and violent behavior in a patient with Korsakoff's psychosis in which propranolol controlled the rage and violent behavior. Elliott[153] suggested that catecholamine access to the brain stimulates alpha and beta adrenergic receptors to induce dyscontrol of anger and violence. Animal studies reported on earlier[20,21] support this hypothesis.

Other authors have reported that psychostimulants[157] are useful in treating children and adolescents with problems of hyperactivity (resulting in fighting, defiance, and impulsiveness) by reducing violent behaviors, and suggest that some adults with residual minimal brain dysfunction resulting in hyperaggressiveness may be aided by similar medication. Finally, although there is some controversy about whether minor tranquilizers cause a reduction in aggressive behavior or cause "paradoxical" rage reactions, one author[158] reports his experience has been that this type of medication helps anxious, aggression-prone individuals, and suggests further research in this area.

Lastly, most authors that advocate a chemotherapeutic approach to managing violent individuals underscore the additional need for psychotherapy in these persons. Lion[159] notes that patients who have aggressive personality disorders are unable to handle depression-arousing stimuli and that a major goal of therapy is to establish an ability to feel and appreciate depression stemming

from frustration. He also notes that these patients need practice in fantasizing and imagining possible consequences of their behavior. He suggests that the therapist needs to be supportive, empathic, and firm, and available in times of need. Lion and Bach-y-Rita[160] reported on their experiences with group psychotherapy and violent outpatients. They noted that two therapists in the group helped to create an atmosphere of control. This atmosphere was accomplished by seating the group around a large table and constantly emphasizing the need for verbal rather than physical expression of emotions, which helped ease the tensions of such a group. This group was viewed as an ongoing therapeutic modality available to members in time of stress, so intermittent member attendance was acceptable. Also, it was felt that insight was less a factor in helping than was the social experience of a group.

CONCLUSIONS

From this review of the literature on the central nervous system and the causes and treatment of violence, it should be clear that physicians have a role in the early identification of potentially violent subjects and the intervention or treatment of individuals who have been violent toward others. It is only through this active role taken by physicians that there will be a substantial reduction in black-on-black homicide.

The review of the literature cited in this article of animal studies, post-traumatic anger and violence studies, tumor and lesion of the limbic system studies, temporal lobe epilepsy studies, episodic dyscontrol syndrome studies, minimal brain dysfunction studies, clinical surveys on violent individuals, and studies on clinical treatment of violence all point to central nervous system factors being involved in some instances of violent behavior. Considering the consistent finding that lower socioeconomic groups are more predisposed to brain injury from trauma and that several studies[13,101,102] have indicated this is indeed true for segments of the black community, investigations of the relationship between central nervous system injury and violence are a must for the black community. Black physicians should assume the lead role in these inquiries.

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Literature Cited

6. Allen NH. Homicide prevention and intervention. Suicide Life Threat Behav 1981; 11:167-179.

11. Taylor PJ, Gunn J. Violence and psychosis, I. Risk of violence among psychotic men. Br Med J 1984; 288:1945-1949.

13. Bell CC, Thompson B, Shorter-Gooden K, et al. Prevalence of coma in black subjects. J Natl Med Assoc 1985; 77:391-395.

20. Reis DJ. Central neurotransmitters in aggression. Res Publ Assoc Res Nerv Ment Dis 1974; 52:119-148.

21. Goldstein M. Brain research and violent behavior. Arch Neurol 1974; 30:1-35.

51. Bender L. Children and adolescents who have killed. Am J Psychiatry 1959; 116:510-513.

95. Maletzky BM. The episodic dyscontrol syndrome. Dis Nerv Sys 1973; 36:178-185.

97. Elliott FA Neurological aspects of antisocial behavior. In: Reid WH, ed. The Psychopath: A Comprehensive Study of Antisocial Disorders and Behaviors. New York: Brunner/Mazel, 1978.

101. Clark K. Dark Ghetto. New York: Harper & Row, 1965.

102. Ramos SM, Delany HM, Freefalls from heights: A persistent urban problem. J Natl Med Assoc 1986; 78:111-115.

110. Healy W. A review of some studies of delinquents and delinquency. Arch Neurol Psychiatry 1925; 14:25-30.

111. Kasanin J. Personality changes in children following cerebral trauma. J Nerv Ment Dis 1929; 69:385-406.

112. Lewis DO. Delinquency, psychomotor epileptic symptoms and paranoid ideation: A triad. Am J Psychiatry 1976; 133:1395-1398.

113. Lewis DO, Shanok SS, Cohen RJ, et al. Race bias in the diagnosis and disposition of violent adolescents. Am J Psychiatry 1980; 137:1211-1216.

114. Lewis DO, Pincus JH, Shanok SS, Glaser GH. Psychomotor epilepsy and violence in a group of incarcerated adolescent boys. Am J Psychiatry 1982; 139:882-887.

115. Lewis DO, Shanok SS, Grant M, et al. Homicidally aggressive young children: Neuropsychiatric and experimental correlates. Am J Psychiatry 1983; 140:148-153.

116. Lewis DO, Moy E, Jackson LD, et al. Biosocial characteristics of children who later murder: A prospective study. Am J Psychiatry 1985; 142:1161-1167.

117. Malmquist CP. Premonitory signs of homicidal aggression in juveniles. Am J Psychiatry 1971; 128:461-465.

118. Inamdar SC, Lewis DO, Siomopoulos G, et al. Violent and suicidal behavior in psychotic adolescents. Am J Psychiatry 1982; 139:932-935.

119. Whitlock FA, Broadhurst AD. Attempted suicide and the experience of violence. J Biosoc Sci 1969; 1:353-368.

120. Hendin H. Black suicide. New York: Harper & Row, 1971.

121. Swanson WC, Breed W. Black suicide in New Orleans. In: Schneidman ES, ed. Suicidology: Contemporary Developments. New York: Grune & Stratton, 1976, pp 99-128.

122. Gunn J, Bonn J. Criminality and violence in epileptic prisoners. Br J Psychiatry 1971; 118:337-343.

123. Hill D. Cerebral dysrhythmia: Its significance in aggressive behavior. Proc R Soc Med 1944; 37:317-330.

124. Hill D, Watterson D. EEG studies on psychopathic personalities. J Neurol Psychiatry 1942; 5:47-65.

125. Hill D. EEG in episodic psychotic and psychopathic behavior. Electroencephalogr Clin Neurophysiol 1952;

4:419-442.

126. Williams D. Neural factors related to habitual aggression. Brain 1969; 92:503-520.

127. Sayed ZA, Lewis SA, Britain RP. An electroencephalographic and psychiatric study of 32 insane murderers. Electroencephalogr Clin Neurophysiol 1969; 27:332-335.

128. Stafford-Clark D, Taylor FH. Clinical and EEG studies of prisoners charged with murder. J Neurol Neurosurg Psychiatry 1949; 12:325-330.

129. Gibbons TCN, Pond DA, Stafford-Clark D. A follow-up study of criminal psychopaths. J Ment Sci 1959; 105:108-115.

130. Frazier SH. Murder—single and multiple. Res Publ Assoc Nerv Ment Dis 1974; 52:304-312.

131. Langevin R, Bain J, Ben-Aron M, et al. Sexual aggression: Constructing a predictive equation. In: Langevin R, ed. Erotic Preference, Gender Identity, and Aggression in Men, in press.

132. Climent CE, Ervin FR. Historical data in the evaluation of violent subjects. Arch Gen Psychiatry 1972; 27:621-624.

133. Lion JR, Bach-y-Rita G, Ervin FR. The self-referred violent patient. JAMA 1968; 205:503-505.

134. Lion JR, Bach-y-Rita G, Ervin FR. Violent patients in the emergency room. Am J Psychiatry 1969; 125:1706-1710.

135. Lion JR. Conceptual issues in the use of drugs for the treatment of aggression in man. J Nerv Ment Dis 1975; 160:76-82.

136. Bell CC, Palmer JM. Violence among inpatients, letter to the editor. Am J Psychiatry 1983; 140:516-517.

137. Bell CC, Palmer JM. Survey of the demographic characteristics of patients requiring restraints in a psychiatric emergency service. J Natl Med Assoc 1983; 75:981-987.

138. Bell CC, Palmer JM. Security procedures in a psychiatric emergency service. J Natl Med Assoc 1981; 73:835-842.

139. Bell CC. Interface between psychiatry and the law on the issue of murder. J Natl Med Assoc 1980; 72:1093-1097.

140. Itil TM, Wadud A. Treatment of human aggression with major tranquilizers, anti-depressants, and newer psychotrophic drugs. J Nerv Men Dis 1975; 160:83-99.

141. Darling HF. Haloperidol in 60 criminal psychotics. Dis Nerv Syst 1971; 32:31-34.

142. Faretra G, Dooher L, Dowling J. Comparison of haloperidol and fluphenazine in disturbed children. Am J Psychiatry 1970; 126:146-149.

143. Sheard MH. Lithium in the treatment of aggression. J Nerv Ment Dis 1975; 160:108-118.

144. Sheard MH. Effect of lithium on aggression. Nature 1971; 230:113-114.

145. Tupin JP, Smith DB, Clanon TL, et al. The long-term use of lithium in aggressive prisoners. Compr Psychiatry 1973; 14:311-317.

146. Morrison SD, Erwin CW, Gianturco DT, et al. Effect of lithium on combative behavior in humans. Dis Nerv Syst 1973; 34:186-189.

147. Rifkin A, Quitkin F, Carrillo C, et al. Lithium carbonate in emotionally unstable character disorder. Arch Gen Psychiatry 1972; 27:519-523.

148. Tunks ER, Dermer SW. Carbamazepine in the dyscontrol syndrome associated with limbic system dysfunction. J Nerv Ment Dis 1977; 164:56-63.

149. Stewart JT. Carbamazepine treatment of a patient with Kluver-Bucy syndrome. J Clin Psychiatry 1985; 46:496-497.

150. Monroe RR. Anticonvulsants in the treatment of aggression. J Nerv Ment Dis 1975; 160:119-126.

151. Monroe RR, Dale R. Chlordiazepoxide in the treatment of patients with "activated EEGs." Dis Nerv Syst 1967; 28:390-396.

152. Monroe RR, Wise SP. Combined phenothiazine, chlordiazepoxide, and primidone therapy for uncontrolled psychotic patients. Am J Psychiatry 1965; 122:694-698.

153. Elliott FA. Propranolol for the control of belligerent behavior following acute brain damage. Ann Neurol 1977; 1:489-491.

154. Yudofsky S, Williams D, Gorman J. Propranolol in the treatment of rage and violent behavior in patients with chronic brain syndromes. Am J Psychiatry 1981; 138:218-220.

155. Williams DT, Mehl R, Yudofsky S, et al. The effect of propranolol on uncontrolled rage outbursts in children and adolescents with organic brain dysfunction. J Am Acad Child Psychiatry 1982; 21:129-135.

156. Yudofsky SC, Stevens L, Silver J, et al. Propranolol in the treatment of rage and violent behavior associated with Korsakoff's psychosis. Am J Psychiatry 1984; 141:114-115.

157. Allen RP, Safer D, Covi L. Effects of psychostimulants on aggression. J Nerv Ment Dis 1975; 160:138-145.

158. Azcarate CL. Minor tranquilizers in the treatment of aggression. J Nerv Ment Dis 1975; 160:100-108.

159. Lion JR. The role of depression in the treatment of aggressive personality disorders. Am J Psychiatry 1972; 129:347-349.

160. Lion JR, Bach-y-Rita G. Group psychotherapy with violent outpatients. Int J Group Psychother 1970; 20:185-191.