

University of Groningen

Clinical Aggressology

Dennen, J.M.G. van der

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version

Publisher's PDF, also known as Version of record

Publication date:

2005

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Dennen, J. M. G. V. D. (2005). *Clinical Aggressology: Neuropathology and (violent) aggression*. Rijksuniversiteit Groningen.

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

CLINICAL AGGRESSOLOGY: NEUROPATHOLOGY AND (VIOLENT) AGGRESSION

by Johan M.G. van der Dennen

Abstract

Neuropathological changes in the Central Nervous System (CNS) – so-called substrate changes, particularly lesions and brain neoplasms – may result in disruptions of behavioral, motivational and/or emotional systems, and chronic (morose) personality changes. Many of these syndromes present a clinical picture in which aggression – ranging from enhanced irritability and anger outbursts, deleterious mood changes, to episodic all-out violent attacks – is a conspicuous symptom.

This paper presents a review of the literature on pathological aggression associated with organic brain disorders, i.e., disorders on the basis of discernible neuropathology of the CNS, such as (a) temporal lobe epilepsy, predominantly as an interictal phenomenon; (b) as a concomitant of abnormal electroencephalograms (EEGs), particularly of the 6- and 14-per-second positive spike types; (c) the dyscontrol syndrome as an episodic behavioral disorder with diffuse brain dysfunction; (d) Minimal Brain Dysfunction (MBD) in children; (e) as a symptom of brain neoplasms, especially tumors in the limbic system, amygdaloid and hypothalamic areas; (f) as a concomitant of viral encephalitis, rabies, and other infectious diseases affecting the CNS; (g) as a result of degenerative disorders and miscellaneous cerebral traumas; (h) as a symptom of several metabolic disorders (such as phenylketonuria), and dysfunctions of the adrenergic, dopaminergic, and serotonergic systems (5-hydroxytryptamine or 5-HT and its metabolites); (i) several neuroendocrine disorders, especially those involving the sex hormones: androgens (such as testosterone), and estrogens (such as in premenstrual syndrome); (j) as an allergic reaction; (k) as a hypoglycemic reaction; (l) in confusional and delirant syndromes such as alcohol intoxication, amphetamine psychosis, etc.

Irascibility, assaultiveness and hostility as a result of chromosomal anomalies or abnormal karyotypes will be considered in another paper, as well as psychopathological syndromes (e.g., paranoid schizophrenia), passive-aggressive character, anti-social (psychopathic, sociopathic), and sadistic personality (These data only corroborate the conclusions presented below).

Among the major conclusions are that (1) a variety of neural substrate changes, preferentially involving the so-called limbic system, may result in pathological aggression, most probably by damaging the fight/flight neural circuitry; (2) Lesions of the CNS may give rise to at least two distinct kinds of pathological aggression: (a) paroxysmal rage outbursts of an impulsive, relatively uncontrollable nature, related to an 'offensive aggression' circuitry in the limbic system; and (b) self-defensive striking out and assaultiveness due to delusional beliefs, hallucinations, and paranoid ideation, preceded and accompanied by overwhelming emotions of fear and terror. This conclusion confirms the distinction in offensive and defensive systems

as reported in many animal studies. It also points to the conspicuous role of fear: indeed, fear might well be the most forceful precipitant of human violence in general.

Introduction

A highly synthetic, circumscriptive definition of ‘aggression’ might be the following. ‘Aggression’ may be considered to be a hypothetical, non-reified (Horn, 1972) motivational system, which, in situations of conflict, perceived threat or danger (pain, intrusion, novelty, thwarting, etc. (Archer, 1976)), and together with other motivational systems – some facilitating or synergistic and others antagonistic or inhibiting (e.g. fear to some extent) – generates a phenomenal category of behaviors (or a behavioral polymorphism), which is the proximate mechanism of contest competition for resources, commodities, values (Thiessen, 1976; Barash, 1977), for the use and purpose of reducing the inclusive fitness of another organism or other organisms (usually conspecifics) and/or enhancing one’s own individual inclusive fitness.

In human beings self-esteem and freedom may be considered functional equivalents of inclusive fitness (cf. Fromm, 1973; Wilson, 1978). As a behavioral polymorph, or ‘scaling’ behavioral strategy (Wilson, 1975) it may contain advertizing, bluff, display, intimidation, threat, attack, ritualized fighting (Rasa, 1980), several modes of coercion (Tedeschi et al., 1974, 1977) and manifestations of power (Rummel, 1977), some of the behaviors being overtly violent. Aggressive behavior can be indirect when the fitness consequences are mediated through resources or inanimate objects (Durham, 1976), or direct if other organisms are physically harmed or injured by the activity, when noxious stimulation is intentionally inflicted (Moyer, 1976), or the other organism is driven away, subjugated, or eliminated (Groen, 1972; Eibl-Eibesfeldt, 1974).

In humans, competing for immaterial values as much as for material resources and commodities, the enhancing of one’s own inclusive fitness may take the form of self-assertion at the cost of others (Galtung, 1964), or self-imposition on other individuals, a group, or an abstraction (Holloway, 1968); and the reduction of inclusive fitness of other individuals may take the form of reducing the freedom to act of another individual (Wilson, 1978), or a symbolic attack on the self-esteem of another individual.

Since in humans many highly sophisticated, subtle and more effective modes of coercion and influence exist, the overtly violent forms of aggression manifestation are more often than not based on specifically diminished or defective communicative competence (Horn, 1972), deficient social skills (Tedeschi et al., 1974, 1977; van der Dennen & van der Molen, 1981), or are paroxysmal attempts to restore a vulnerable sense of self-esteem (Toch, 1969; Rochlin, 1973; Fromm, 1973; Heller, 1979; van der Dennen, 1980), or are symptoms of underlying pathology.

It should be noted that ‘violence’ in this conception is not synonymous with ‘aggression’. Indeed, van der Dennen (1980) argued that in humans most high-level violence (i.e. collective

political violence, particularly purges, institutionalized terror, genocides, civil wars and interstate wars) and most low-level violence (i.e. interindividual violence in the criminal domain) is not aggressively motivated at all (in the sense of 'affective', 'arousal' or 'angry' aggression), but consists of purposely calculated, instrumental and more or less 'rational' acts. He pleaded for a conceptual dissociation of the categories 'aggression' and 'violence', aggression being one source of violence among many; and violence being one manifestation of aggression among many. In this paper, however, we will focus on those manifestations of aggression which have a more or less violent quality, in the conventional sense that the corporal integrity of the victim is being violated in an agonistic encounter, resulting in pain, tissue damage, physical injury, incapacitation, mutilation, or death.

'Aggression' has usually been defined either as a behavioral category, or as a motivational construct. Aggression in the latter sense may range from mild irritability, through occasional flaring up of anger, to paroxysmal fits of fury. In the clinical literature it usually also comprises the relatively long-lasting attitudes and states of mind like hostility, hatred, resentment, spite, vindictiveness, assaultiveness, destructiveness, etc., as well as relatively enduring moods like peevishness, sulkingness, negativism, etc. It is a multilevel, multiplex construct.

Violent Aggression as a Pathological Symptom

It has often been argued that a certain amount of aggression would be necessary for the healthy pursuit of social, sexual, and even intellectual endeavors. There is, however, no evidence in the studies of achievement motivation – contrary to the Lorenzian assertion – that aggression is indispensable for intellectual pursuit. McClelland et al. (1953) concluded that there was no correlation between the instigation to aggression and achievement motivation. Indeed, as Kim (1976) argued, there is much to be said for the hypothesis that aggression (at least violent aggression), far from being normal, healthy or indispensable, is a pathological symptom.

Many investigators have advocated similar notions. Scott (1975), for instance, proposed that maladaptive agonistic behavior, particularly that resulting in destructive violence, can be attributed to the disaggregation of a system or systems on some level of organization (gene pool, physiology, organism, society, ecosystem). Thus Scott views violent aggression as a symptom of systemic breakdown, disruption or pathology. In a similar vein Moreno (1981) concluded that while aggression may be adaptive social behavior, violence may be pathological.

On the other hand, evolutionary biologists, ethologists and many psychoanalysts and psychologists alike have pointed to the adaptive character of the aggressive motivational system in evolution and in ontogeny. Aggression is thus regarded as an indispensable, indeed necessary, coping device or emergency mechanism with a high degree of survival value in the struggle for existence.

These *prima facie* contradictory views may not be as contradictory as they seem, provided that the proper distinctions are made. Defensive, reactive aggressive behavior against predators or conspecifics is obviously adaptive as Craig (1928) already noted. Offensive attack may, in certain circumstances, be adaptive as a behavioral strategy to enhance an organism's inclusive fitness. It is not, however, an activity an organism should indulge in casually. All-out violence may lead to short-term gains but is often self-defeating in the long run.

Rasa (1980) has argued that self-defensive aggression is aversive, while 'property-protective' aggression is appetitive (at least in animals), on the argument that it would be disadvantageous for an organism not to seek to better its position, and therefore the chances of passing on its gene material to the next generation.

Be that as it may, violent aggression as a symptom of underlying pathology is frequently encountered in clinical practice. Pathological aggressiveness is an epiphenomenon of many neuropathological and psychopathological syndromes (e.g. Lion, 1975; van der Dennen, 1977; Sarteschi et al., 1978). Probably most of the violent patients seen in clinical and forensic settings are patients with severe character disturbances of the explosive, anti-social, or passive-aggressive types. Impulsivity is a common feature in these cases and the violence usually has a paroxysmal, episodic quality to it. Violent ideation, too, is commonly encountered in practice. Most clinicians have seen the patient who is afraid of losing control over violent urges and who fears, say, harming his child or hitting his spouse. Such cases often are accompanied by much psychomotor agitation and anxiety (Lion, 1975).

In syndromes of organic origin, to which the following section of this paper will be devoted, single or multiple episodes of pathological aggressiveness may appear in (a) temporal lobe epilepsy – predominantly as an interictal phenomenon; (b) as a concomitant of abnormal EEGs, particularly of the 6- and 14-per-second positive spikes type; (c) the dyscontrol syndrome as an episodic behavioral disorder with diffuse brain dysfunction; (d) Minimal Brain Dysfunction (MBD) in children; (e) as a symptom of brain neoplasms, especially tumors in the limbic system, amygdaloid and hypothalamic areas; (f) as a concomitant of viral encephalitis, rabies, and other infectious diseases affecting the CNS; (g) miscellaneous brain trauma (e.g. degenerative disorders); (h) several metabolic disorders (e.g. Lesch-Nyhan syndrome); (i) several neuroendocrine disorders (e.g. the premenstrual syndrome); (j) as an allergic reaction; (k) as a hypoglycemic reaction; (l) as a result of chromosomal anomalies or abnormal karyotypes (e.g. XYY and Klinefelter syndrome) – the evidence, however, is highly controversial and will not be considered in this paper; (m) in confusional and delirious syndromes such as alcohol intoxication, amphetamine psychosis, etc.

In many instances of violent aggression associated with an acute toxic psychosis state, the violence is bizarre, erratic, undirected, and to be understood as a desperate defensive response to terrifying delusions of a paranoid thought disorder. In these cases of pathologically altered brain structure, the changes may affect behavior, as Mark & Ervin (1970) pointed out, "because they effect how the brain perceives, *fails* to perceive, or *misperceives* incoming stimuli. For instance, a person whose violence is related to the presence of brain disease

generally does not attack without what *he* considers to be provocation. What happens is that the brain misperceives some incoming stimulus – a harmless gesture, or a joking remark, let us say – as extremely threatening or enraging, when it is in fact not so”.

The main psychopathological syndromes which may be responsible for pathologically aggressive behavior include:

- (a) schizophrenic psychosis – mainly in its form of excited catatonia, in the course of hallucinatory deliria of a terrifying, or persecutory nature, as a sudden reaction to harmless stimuli. There are forms, coming from one or more schizophrenic episodes, which are called ‘defective’, where the most striking behavior consists in sudden and intermittent aggressive outbursts of a pseudopsychopathic type;
- (b) the manic phase in cyclothymia – this occurs sometimes during the psychotic phase of the illness, or in the presence of schizophrenic elements (so-called mixed psychoses);
- (c) paranoia – paranoid syndromes of development, which give progressively to an abnormal personality with fanatically suspicious traits, have the constant feature of pathological aggressiveness;
- (d) morbid jealousy;
- (e) depression – aggression can result from depression such as in cases of involuntal, puerpural or reactive depressions where the patient reacts in despair by striking out against other people. In other cases it may lead to suicide or automutilation;
- (f) violence related to sexual perversions like sadomasochism, and the vicious violence encountered in lust murder and some cases of rape is particularly enigmatic because of its savagely cruel and sometimes atavistic character;
- (g) pathological aggression as a qualitatively altered pattern of human behavior – there are people who, mainly in their adult life and apparently without being affected by any of the psychic illnesses mentioned above or without being addicted, show a pattern of interpersonal relationship which can be described as violent or destructive. These subjects have been called ‘psychopathic sociopaths’, due to their manifest anti-social and asocial nature, which brings them to attack, strike, and destroy other people, without any intelligible motivation. This lack of any apparent motivation should distinguish such sociopaths from criminals who are engaged in destructive violence for personal or lucrative reasons. However, the borderline between these two categories seems indistinct and it is certainly very likely that a sociopathic individual will commit a crime because of the characteristics linked to his peculiar relational model with others and society. The behavioral anomalies of psychopathic sociopaths typically consist in (1) repeated episodes of impulsive, violent, and assaultive aggressiveness towards other people, their own families and society in general; (2) a total absence or an extreme inadequacy of critical evaluation of their own behavior; (3) a tendency to persist compulsively in behaving aggressively; (4) a failure to respond to pharmacological and/or to psychological

treatments; (5) The most striking feature of these patients is the highly dulled affects and the complete lack of emotional awareness of other people, who are perceived as objects; (6) Also typical is the absence of guilt feelings ('moral idiocy').

Furthermore, some personality disorders have been described with a heightened proneness to aggressiveness, e.g., the borderline personality, the oligophrenic personality, the neurotic personality, the compulsive personality, etc. Finally, extreme overcontrol and inhibition of primary aggression may predispose to later explosive breakthroughs (Megargee, 1966 et seq.) and to secondary aggression (hostile or violent behavior that is entirely disproportional or even unrelated to current provocation). A total dampening of primary aggression also may lead to seriously inhibited character development or a chronic, life-long apathy (Solomon, 1970), or to a variety of so-called psychosomatoses with psychocutaneous, cardiovascular, gastro-intestinal or respiratory symptom formations (e.g. review in Buss, 1961).

In a living human being aggression is always mixed with other impulses, defenses, emotions and character traits. Symptom formation occurs when an aggressive impulse is warded off and the energy is discharged into a pathway of organic functions (Musaph, 1969; Groen, 1957; Wolff, 1969). It is a hard clinical fact, according to Bastiaans (1969), that psychosomatic syndromes do not arise when activated aggression has not been suppressed or repressed beyond a certain degree. These syndromes are thus regarded as being based on a defective aggression regulation.

“Consideration has to be paid to the function of aggressiveness, which should ensure the preservation of the individual’s homeostasis with respect to himself and his environment. More specifically, this function allows the individual to properly react to threat or dangers and to obtain the self-affirmation when there is a real need for it. This belongs to the legitimate sphere of aggressiveness, which instead becomes wrong when it loses its adaptive role and begins to operate autonomously in situations which do not require its emergence. This new aggressiveness may be intermittent or continuous, reversible or irreversible. It corresponds to clinical judgments of ‘an outburst of unmotivated aggressiveness’ or of a ‘bad-tempered, aggressive character’; in the worst cases the subject is classed as an aggressive sociopath. Apart from the case in which anatomopathological lesions affect the brain structures involved in aggressive response (Valzelli, 1971), it should be concluded that a change has taken place in the perception and/or interpretation of what constitutes a danger, obstacle or threat to the organism. The facts, situations, objects or people which do not represent a real threat to the individual are interpreted as if they did, and provoke an aggressive reaction which is inappropriate, as it has no real purpose, and is not required by the organism’s homeostasis” (Sarteschi et al., 1978).

Neuropathology and Aggression: A Literature Review

Siomopoulos (1978) examined the distribution of various offenses among various psychiatric diagnoses in a sample of 342 Black and 109 White male indicted felons who were adjudicated unfit to stand trial. Schizophrenia was the leading psychiatric diagnosis among the total

population. Significantly, organic brain syndromes were associated exclusively with crimes of high degree of violence.

It is also significant that violent male prisoners can be successfully discriminated with a relatively simple neuropsychological test battery (Spellacy, 1977, 1978).

It is especially the focal brain lesions in the amygdala-uncal-hippocamal areas, whether atrophic due to interference with blood supply, or neoplastic, which result in personality changes in about 50 % of the patients, characterized by intolerance of frustration, impulsiveness and irritability (Hill, 1964). Thus it seems justified to include the organic brain syndromes – behavior disorders on the basis of lesions, neoplasms or dysfunctions of the central nervous system (CNS) – in a class of high vulnerability to aggression and/or violence.

Excluded from this review is the extensive literature on stereotaxic neurosurgery and otherwise artificially induced lesions, together with the many electrical brain stimulation studies.

Temporal Lobe Epilepsy (TLE)

The *furor epilepticus* was already well-known in ancient times. The psychiatric study of epileptics began in the early-19th century with Esquirol. After 1850, Morel, Griesinger and Falret developed the clinical picture of what is now termed psychomotor or temporal lobe epilepsy, with emphasis on its psychiatric aspects (Blumer, 1976). Szondi (1952) and Geschwind (1975) have given accurate descriptions of the particular setting of aggressiveness in epilepsy. Several reviews and monographs deal with the topic of aggressivity and epilepsy (Taylor, 1969; Slater & Roth, 1969; Mark & Ervin, 1970; Monroe, 1970; Mirsky & Harman, 1974; Goldstein, 1974; Blumer, 1976; Moyer, 1976; Mark, 1978; Gonzalez de Rivera, 1981; Gigli et al., 1981).

From the psychiatric viewpoint, the more or less pronounced proneness toward irritable and angry-explosive behavior in certain epileptics is merely one aspect of a complex personality and behavior change which may develop after onset of overt seizures (Blumer, 1974, 1975, 1976; Mirsky & Harman, 1974; Gastaut et al., 1955). This irritable, at times threatening and abusive behavior is episodic, and combines paradoxically with a highly good-natured (hyperethical and sometimes hyperreligious) attitude which may be almost entirely predominant. What is loosely referred to as ‘violence’ or ‘aggressiveness’ in epileptics represents a very real problem, but is distinctly different from the deviant aggression commonly found in passive-aggressive and antisocial personality disorders, as well as from the one less commonly found in paranoid, catatonic or manic-depressive patients (Lion & Penna, 1975).

According to Minkowska (1937) the concept or the ‘epileptoid’ character is bipolar including viscosity and explosivity. A certain relationship of the ‘aggressiveness’ in epileptics with the impulsive behavior of ‘epileptoid’ or explosive personality disorders is probable but not well-

established (Blumer, 1976).

Mirsky & Harman (1974) emphasized the multidetermined etiology of epilepsy, which encompasses, in addition to the life-long stress of seizures, defective cognitive mechanisms of long standing, possible social class differences in the acceptability of aggressive acts, and the accumulated anger and frustration derived from an impoverished, punishing and perhaps painful childhood. "One wonders also, whether the catastrophic rage or violence described in some patients may be understood as a means of warding off catastrophic feelings of anxiety, inadequacy or worthlessness". Such patients may also have more than one seizure type, as suggested by Rodin et al. (1976).

By far the most common focal cerebral disorders associated with poor control of destructive impulses and rage are those which also give rise to limbic system epilepsy (Mark, 1978). Already more than a century ago, Maudsley (1874) described the auras of temporal lobe epilepsy, and the ictal and interictal violence and assaultive behavior seen in his patients. Violence was a feature of the episodic, short duration, epileptic mania described by Gowers (1881), while instances of violent actions during automatisms are evident in the case histories cited by Jackson (1958). Gastaut et al. (1955) recorded paroxysmal rages in 50 % of their epileptics of the temporal lobe type. Falconer et al. (1958), reporting on 50 patients whose temporal lobe epilepsy was associated with a predominantly unilateral spike focus, found as the most common personality disturbance in 38 % a "pathological aggressiveness occurring in outbursts in an otherwise adjusted individual". Another 14 % had "an often milder but more persistent aggressiveness associated with a continued paranoid outlook". Rages became so severe in 38 epileptic patients of Roger & Dongier (1950) that they had to be confined to mental hospitals. They all had classical neurological symptoms of temporal lobe epilepsy, evidence of EEG foci in scalp leads from temporal or inferior frontal regions or both (Mark, 1978). Ounsted et al. (1968) found that 36 of 100 temporal lobe epileptics developed repeated episodes of 'catastrophic rage'.

Dramatic and indicative as these studies may seem, there is also counterevidence. Penfield & Jasper (1954) concluded that "Neither localized epileptic discharge nor electrical stimulation is capable of awakening the emotions of anger, joy, pleasure, or sexual excitement". Similarly, Gloor (1967) concluded that "rage with or without aggressive behavior is an extremely rare ictal phenomenon". Currie et al. (1971) studied 666 temporal lobe epileptics seen in a general hospital. Only 16 had rage attacks as part of their seizures. Of 50 patients treated by Paillas (1958) with surgery of the temporal lobe, 26 had affective symptoms as part of their seizure patterns. These symptoms consisted of fear or a generalized unpleasant feeling in 18; only 2 had 'angry impulses'.

Fear, however, may precipitate attack behavior. This was true in 2 of Mark's (1978) patients with inlying electrodes. These two patient. had an aura containing an element of fear immediately preceding their destructive or assaultive acts.

Individuals manifesting temporal lobe epilepsy show a higher percentage of personality disorders than do persons with other types of neural malfunctioning (Moyer, 1976). Ictal rage

does occur but it is less common than either fear or depression (Williams, 1956, 1965, 1968; Paillas, 1958; Weil, 1959; Ounsted et al., 1966; Gloor, 1967; Currie et al., 1971; Rodin, 1973). Ictal behavior involving considerable violent aggression or even homicide has been reported (MacDonald, 1961; Walker, 1961; Roth & Harper, 1962; Fenton & Udwin, 1965; Falconer, 1967; Brewer, 1971; Dinnen, 1971; Gunn & Fenton, 1971; Gunn, 1978), but it actually appears to be quite rare; and is not usually purposeful (Freud, 1928; Walker, 1961; Fenton & Udwin, 1965; Serafetinides, 1965; Falconer, 1967; Taylor, 1969).

There is evidence that uncontrolled, impulsive, assaultive behavior is not uncommon as an interictal behavior pattern, particularly among institutionalized temporal lobe epileptics (Gastaut et al., 1953, 1955; Gastaut, 1954; Liddell, 1953; Falconer et al., 1958; Gloor, 1960; Keating, 1961; Nuffield, 1961; Glaser et al., 1963; Schwab et al., 1965; Serafetinides, 1965, 1970; Ounsted, 1969; Walker & Blumer, 1972), and chronic epileptic patients are capable of actions of the most malicious and petty spite, combining them with self-justification (Slater & Roth, 1969), though among noninstitutionalized TLE patients no more criminal or anti-social behavior is found than in the rest of the population (De Haas, 1963; Livingston, 1964; cf. Alström, 1950; Gunn & Bonn, 1971; Mirsky & Harman, 1974). Gigli et al. (1981) found no relationship between aggressiveness and criminality in epileptics.

Even restricting the meaning of aggression to overtly aggressive displays, all that emerges from many reports is that a proportion of epileptics have a troublesome bad temper (Taylor, 1969). EEG studies usually show that the hippocampal form of TLE is the most common and most associated with behavior disorders (Hill, 1952; Gastaut, 1953; Hughes et al., 1961). These findings, according to Taylor (1969) would be consistent with the neuropathology of TLE where the most common lesion, mesial temporal sclerosis, most regularly affects the hippocampus (Margerison & Corsellis, 1966; Falconer & Taylor, 1968; Taylor & Falconer, 1968). Acts of violence are unusual in the context of automatisms, and the latter have little role in crime (Gunn & Fenton 1969, 1971, 1972; Mirsky & Harman, 1974; Goldstein, 1974). The importance of left temporal lobe damage has been emphasized by Flor (n.d.), Mnukhin & Dinaburg (1965), and Lishman (1966). The implication of actual temporal lobe seizure in a specific directed violent attack remains difficult to prove (Glaser, 1975).

Moyer (1976) suggested that the assaultive behaviors result from damage to the inhibitory neural systems for aggression. This author also presents evidence that the neural systems involved in the seizure are different from those involved in the impulsive-aggressive syndrome, as effective anticonvulsant treatment may exacerbate the irritability and dyscontrol.

Gloor (1975) distinguished two kinds of nonictal aggressive behavior in TLE patients. The first type is aggressive behavior in conjunction with a seizure, but during the postictal confusional state. It represents a consequence of post-ictal confusion and results from the patient's misinterpretation of others' usually well-meaning attempts at restraining him. The second type is characterized by a low rage threshold. Such patients even in response to the most trifling provocation may fly into a violent rage which is totally out of proportion to the triviality of the triggering event. It is very unlike a temporal lobe fit. The neurophysiological

mechanism underlying these episodes of violent outbursts is obscure; there are no hard data that limbic system dysfunction is involved. In a former publication (Gloor, 1967), he suggested that seizure discharge might be akin to the physiological reaction to unknown threats, leading to fear or flight, while the more mundane stimuli of everyday life may provoke rage because they are known and less feared.

Individuals who come to a medical facility (emergency room, violence clinic) complaining of fear of going out of control and injuring someone, of homicidal ideation, repetitive aggressive behavior under the influence of alcohol, impulsiveness, rage outbursts, dangerous use of the automobile, or of repeated arrests for violent acts (Vide infra: Dyscontrol Syndrome), have been carefully examined (Bach-y-Rita et al., 1971; Lion et al., 1974). In one series, 37 of 79 who were given an EEG showed abnormalities, with 20 out of the 37 having temporal lobe spikes. Other studies raise more doubts about any relationship of epileptic temporal lobe abnormalities, in the absence of clinical seizures, and episodic loss of control over violent impulses (Green, 1961; Milstein & Small, 1971).

A most interesting hypothesis, however, has already been suggested (Monroe, 1970; Treffert, 1964): that individuals with the epileptic EEG abnormality and no overt seizures may be prone toward more harmful outbursts of violence than patients with clinical seizures. This hypothesis is in keeping with the clinical observation of the 'tension-releasing' property of overt generalized seizures (and occasionally of minor seizures) in certain patients. The well-accepted therapeutic use of seizures in psychiatric patients clearly attests that a seizure is not merely a neurological phenomenon, but also a mechanism for abnormal instinctual discharge (Blumer, 1976).

Recent studies: Graham & Rutter (1968) found an incidence of behavior disturbance of 34 % of epileptic children, with a preponderance of the psychomotor category. A study of 100 children with TLE reported by Ounsted (1969) revealed that 36 suffered from interictal episodes of rage.

Gross & Kaltenbäck (1975) studied a representative group of delinquents defined as aggressive toward strangers (homicide or bodily injury) and found that they showed the same incidence of epilepsy (4.6 %) as the general population, but significantly higher incidence of schizophrenia. Among epileptics, the authors conclude, aggression seems to be associated with psychotic symptoms, reactive mood states, and paranoid reactivity. During the actual crime, amnesic or transitory paranoid-hallucinatory syndromes may be postulated. Epileptic aggressors very rarely kill the victim.

Lewis (1976) found psychomotor epileptic symptoms in 18 (6 %) of 285 children referred to a juvenile court. Abnormalities appeared in 11 of the 14 available EEGs, but temporal lobe foci were noted in only 3 cases. Of these 18 children, 16 experienced paranoid symptoms that led to aggressive behavior.

Lewis et al. (1982) found 18 cases of psychomotor epilepsy among 97 delinquent male adolescents, an incidence far more prevalent than the .5 % found in the general population.

The number of psychomotor symptoms (abnormal EEGs, history of generalized seizures, and memory lapses) was correlated with the degree of violence exhibited by the subjects. Psychomotor symptoms were correlated more strongly with certain psychotic symptoms (e.g., hallucinations and paranoid ideation) than with soft neurological signs or intellectual deficits.

For TLE and aggression see also Dowzenko et al. (1975), and Sherwin (1976).

Libert (1980) described a case of a 19-yr-old male suffering from familial myoclonic epilepsy (Unverricht-Lundborg disease) who was hospitalized for aggressive behavior. Deteriorating rapidly, this patient entered into intermittent states of confusion and violent aggression lasting from several hours to several weeks.

Blumer (1976) summarizes the evidence on 'violence and epilepsy' as follows:

1. Angry-irritable behavior during the interictal phase (and sometimes accentuated preictally) is commonly found among chronic temporal lobe epileptics. It can reach threatening and violent proportions in some of them, but physical harm is rarely inflicted. This behavior is episodic and combines with a usually predominant good-natured (hyperethical and sometimes hyperreligious) attitude. Seizures may serve as a safety valve for the discharge of pent-up crude affects. The outbursts of temper can cause serious problems in the domestic sphere or in institutions, but are not a threat to the public safety.
2. Ictal violence is a most unlikely event, if it occurs at all. It is suggested, however, that outbursts of anger may occur in association with a build-up of subclinical seizure activity.
3. In rare cases, unchecked violence may take place in the confusional-amnestic postictal state when an individual meets with some interference. The importance of these states has decreased with modern seizure control.
4. Some individuals who are not epileptic but have paroxysmal EEG abnormalities (temporal lobe sharp waves or spikes in particular) seem to be more prone to react with excessive violence than patients with overt seizures. The syndrome, however, is not well outlined and needs further investigation.

These conclusions are corroborated by Mark (1978) who concluded that abnormal aggressivity is clearly more frequent as an interictal than as a seizure phenomenon; and González de Rivera (1981) who concluded that postictal pathology is characterized by psychotic episodes and occasional severe aggressions. For methodological criticism see Coleman (1974). For a review of research problems on TLE and aggression see Kligman & Goldberg (1975).

Abnormal EEG and Abnormal Aggressive Behavior

Jasper et al. (1938) were the first to report the high incidence of abnormal EEGs in children

with behavior disorders, particularly poor impulse control, destructive behavior and hyperkinesis, possibly reflecting a diffuse brain pathology. The incidence of abnormality was still higher when a subgroup of hyperactive impulsive children was considered. Similar observations were made shortly after by Strauss et al. (1940). Since then a vast amount of material on this subject has been assembled (Taterka & Katz, 1955; Zimmerman, 1956; Gross & Wilson, 1964; Bayrakal, 1965; Yoshii et al., 1961 1964; Arai et al., 1966; Aird & Yamamoto, 1966; Monroe, 1970; Stevens, 1972; Christiani & Völker, 1977; Harris, 1978; Surwillo, 1980; see also Meiri, 1976). The EEGs of aggressive, adult psychopaths show a high degree of similarity to the EEGs of normal young children. Hill & Watterson (1942) were the first to note this and to suggest that the abnormal EEGs in aggressive psychopaths may result from a failure in the development of the CNS. This 'maturational-retardation' hypothesis postulates that the retarded EEG pattern seen in aggressive adult psychopaths reflects a functional cortical retardation. Although some evidence supports the maturational-retardation hypothesis (Kiloh & Osselton, 1966; Murdoch, 1972), the finding that many aggressive psychopaths have normal EEGs argues against it. Surwillo (1980), who investigated the maturational-retardation hypothesis in severely aggressive boys, found that all the aggressive children had one distinguishing trait in common: their EEGs had the characteristics of chronologically younger children.

Although temporal and subcortical foci are most frequently reported to be associated with aggressive-behavior disorders, Cohn & Nardini (1958) describe an abnormality with an occipital focus in young adults who were hostile and irritable among other symptoms. Other studies, however, failed to find a relationship between EEG abnormalities and aggressive tendencies (Knott & Gottlieb, 1943; Arthurs & Cahoon, 1964; Loomis, 1965; Stevens & Milstein, 1970). Carvalhal Ribas et al. (1974), on the other hand, found evidence of cerebral disrhythmia in 69 % of youngsters with behavior disorders with predominance of aggressiveness.

EEG-abnormality Incidence in Specific Populations

Clinical electroencephalography has been used to study the incidence of abnormality in various psychiatric populations and criminal offenders. The earliest studies are those by Hill & Watterson (1942), Harty et al. (1942), Jenkins & Pacella (1943); Hill (1944), and Stafford-Clark & Taylor (1949). Jenkins & Pacella concluded that whereas delinquency does not show any relationship to EEG abnormalities the habitual expression of aggressiveness does, while Stafford-Clark & Taylor concluded that the abnormality in the EEG is directly proportional to the unpredictability of the crime. Other positive correlations have been reported by Silverman (1943, 1944), Jones et al. (1955), Verdeaux & Verdeaux (1955), Winkler & Train (1959), and Assael & Kohn Raz (1967).

Aggressive psychopaths show significantly more abnormal responses (theta activity) than either nonaggressive psychopaths or nonpsychopaths (Hill & Watterson, 1942; Silverman, 1943, 1944; Gibbens, 1958; Gibbens et al., 1959; Murdoch, 1972).

Results by Blackburn (1975), however, are not in accord with the view that a high prevalence

of theta activity characterizes aggressive offenders.

A survey of 105 murderers showed that about half of them had abnormal EEGs (Hill & Pond, 1952). They found more abnormalities in psychotic and motiveless murders than in incidental or clearly motivated homicides. Of habitually aggressive prisoners, 65 to 80 % had EEG abnormalities, compared to 24 % of those who had committed one violent crime, and 12 % of the general population (Hill, 1944, 1952, 1963; Williams, 1969, 1975), while the incidence of EEG abnormality in 32 murderers to be judged 'insane' was 65 % as opposed to nonpatient controls which was 15 % (Sayed et al., 1969; see also Hill, 1944, 1952; Gibbs et al., 1945; Brown-Mayers & Straub, 1953; Levy & Kennard, 1953; Ellingson, 1954; Kennard et al., 1958; Pond, 1961, 1963; Rogina & Serafetinides, 1962; Winkler & Kove, 1962; Small, 1966; Mitsuda, 1967; Stevens et al., 1968; Kido, 1973; cf. also Monroe, 1970; Mark & Ervin, 1970; Goldstein, 1974; Moyer, 1976; Harris, 1976; Surwillo, 1960; Buikhuisen, 1982).

Suicidal ideation and attempts have been mentioned by Goldstein (1974) as among the determinants of violent and aggressive acts. Indeed, Struve et al. (1972) found a higher incidence of paroxysmal EEG abnormality among patients with suicidal ideas or attempts as compared to patients without these symptoms. Krynicky (1978) reported that paroxysmal activity, particularly in the frontal area, appeared to be the most important EEG feature related to assaultive behavior in a group of repetitively assaultive adolescents. The neuropsychological data give some support to the hypothesis that severe aggression is related to left hemisphere dysfunction.

The results show a diversity of diagnostic criteria, definitions of criminal behavior, and variability in EEG techniques (cf. Mark & Ervin, 1970). Nevertheless, deviation of cerebral physiology may be one, among many, of the factors involved in habitually violent offenders. Indeed, "One of the most striking facts is that abnormalities in the EEG tend to become commoner as the section of the population brought under investigation becomes more abnormal, particularly if the abnormality includes propensities for aggressive and explosive behavior" (Surwillo, 1980).

The 6- and 14-per second Spikes and Aggression

One EEG abnormality that appears to be specifically associated with impulsive, aggressive behavior is the 6- and 14-per second positive spike in the temporal area (Gibbs & Gibbs, 1951; Schwade & Otto, 1953; Schwade & Geiger, 1953, 1960; Henry, 1963; Hughes, 1965; Monroe, 1970; Moyer, 1976). "The aggressive behavior shown by these subjects is not the more random and confused type found in the ictal and immediate postictal state. The rage and destructive attacks occur with little or no provocation and are frequently carried out with skill and precision. Under the impulse of an explosive episode of aggression, the individual appears overwhelmed by his own momentum and is unable to inhibit the act" Moyer (1976) reports. The aggressive acting out is followed by relief of tension and little, if any, feelings of guilt or remorse (Bender, 1953; Stehle, 1960; Schwade & Geiger, 1960; Monroe, 1970). Relatively motiveless murders have been committed by individuals showing this spike pattern

(Schwade & Otto, 1953; Winfield & Ozturk, 1959; Stehle, 1960; Schwade & Geiger, 1960; Woods, 1961), while delinquents showing the pattern are more likely to be recidivists and involved in crimes of violence (Yoshii et al., 1963). The incidence of the 6- and 14-per second spiking in the population at large, as well as the anatomical neurophysiological origin and meaning of the pattern, are still subject to controversy. Moyer (1976) suggests that it may reflect some damage in the inhibitory mechanisms that reduces the threshold for aggression in individuals thus affected. It may not even be an abnormality at all (Henry, 1963; Lombroso et al., 1966; Eeg-Olofsson, 1970; Woerner & Klein, 1974; Goldstein, 1974).

Conclusions

“A review of the literature indicates that there is no one-to-one relationship between EEG findings and psychiatric conditions or criminal activities. The relationship is either statistical or inferential” Mark (1978) concludes. The findings usually consist of bioelectrical immaturities, such as slow alpha and theta activity, although distinct abnormalities are also reported (Revitch & Schlesinger, 1978).

As approximately half of the deviant populations investigated show no indication of abnormal EEG tracings, these are neither a necessary nor a sufficient condition for abnormal aggressive or criminal behavior. Strauss (1959) already concluded: “It seems evident now that the electroencephalographic abnormality indicates only one factor which helps to create the clinical picture. One sees children with the same electroencephalographic abnormality and without behavior disorder, and also children with the same type of behavior disorder and no electroencephalographic abnormality”, and Surwillo (1980) concludes a recent survey: “There is a lack of uniformity in the EEG assessments and no further evidence has been produced which could alter Ellingson’s (1954) conclusion that ‘no specific relationships have been established between EEG abnormalities and specific symptom’.”

Dyscontrol Syndrome

Mark et al. (1967), Mark & Ervin (1970), cf. Ervin et al. (1955), Sweet et al. (1969); Taylor (1969) identified a syndrome due to limbic system and temporal lobe abnormalities which they labeled the ‘dyscontrol syndrome’, the characteristic symptoms of which were considered to be (not necessarily all simultaneously present): (1) a history or physical assault, especially wife and child beating; (2) pathological intoxication; (3) a history of impulsive sexual behavior, at times including sexual assaults; and (4) a history of traffic violations and accidents. ‘Pathological intoxication’ they call a condition in which even a small amount of alcohol triggers acts of senseless brutality (Vide infra). Mark & Ervin point to parallel observations by Menninger (1963) and his coworkers who described disorders of impulse control from a psychodynamic point of view. One order of dyscontrol defined by Menninger as ‘ego rupture’ is manifested by two general syndromes: (1) The chronic, repetitious occurrence of relatively milder (although serious) aggression; and (2) sudden, explosive outbursts of very serious aggression. Males predominate by 9:1. The propensity for violence

begins in early childhood (Robins, 1966; Detre et al., 1975).

Monroe (1970 et seq.) has presented both the phenomenologic and psychodynamic analyses of a group most frequently showing paroxysmal EEG bursts of 3-4 per second activity with some notching, a group he designated as 'primary dyscontrol'. This group of subjects are characterized as individuals who demonstrate frequently recurring single acts or short series of acts with a common intention which occur abruptly, are motivated by intense emotions (usually fear and rage) and result in a relief of tension or perhaps more specific need gratification. Monroe also presents an elaborate clinical taxonomy of episodic behavioral disorders, with many case histories.

Andy & Jurko (1972) described a 'hyperresponsive syndrome' the main characteristics of which are hyperkinesia, aggression, and pathological affect. Detre & Feldman (1963; see also Detre et al., 1975) described a syndrome of 'explosive personality disorder'. Behar & Stewart (1982) describe 'aggressive conduct disorder' in children as a psychiatric syndrome. These syndromes may show considerable overlap.

The dyscontrol syndrome was corroborated by Waller & Whorton (1973), Maletzky (1973), and Maletzky & Klotter (1974). Mark & Ervin's observation that any single act of violence is not isolated, atypical behavior, but one in a series of violent incidents is supported by Wolfgang's (1958) study of 588 homicides committed in Philadelphia.

According to Benson & Geschwind (1975) the role of temporal lobe seizures in dyscontrol violence remains unsettled. For the histopathological evidence of limbic system involvement in the dyscontrol syndrome see Girgis (1977). A review is provided by Elliott (1976).

Pathological Intoxication

Pathological intoxication is not synonymous with ordinary drunkenness. It has been a recognized clinical entity and it was initially described by Krafft-Ebing in 1869 (quoted in Banay, 1944). The state is characterized by its dramatic and sudden onset. Detre et al. (1975) describe the syndrome of pathological intoxication as follows:

"The term refers to a state in which the individual engages in a violent act after drinking, an act for which he will have little or no recollection. Such behavior may sometimes be elicited by small amounts of alcohol, much less than would be required for ordinary intoxication. Blood alcohol levels below 30 mg per 100 ml have been recorded in these cases (Pincus & Tucker, 1974). Pathological intoxication is not associated with slurred speech and incoordination and may last for only a few minutes. It occurs most often when alcohol is imbibed under circumstances 'conducive' to violence, i.e., at a bar or a party, and it has been difficult to reproduce this state of intoxication by administering alcohol in any quantity in a laboratory setting or by intravenous injection. Those who become pathologically intoxicated may not be chronic alcoholics and the condition is not limited to individuals with a criminal disposition. It is relatively rare, even among brain-damaged individuals. Yet 90 per cent of

pathological-intoxication cases are associated with brain damage, epilepsy, retardation, or psychosis (Bowman & Jellinek, 1941). Electroencephalographic change may also be associated with episodes of pathological intoxication (G.N. Thompson, 1963)".

The attack occurs early in intoxication. Consciousness is frequently impaired and the perception of the environment is distorted by delusions that are always of a persecutory nature and hallucinations that have a hostile content. Maniacal outbursts occur and include terminal fits of rage and an irresistible desire for destruction (Moyer, 1976; Skelton, 1970; Banay, 1944). A number of case reports of pathological intoxication are on record (Banay, 1944; Marinacci, 1956, 1963; Lion et al., 1969; Skelton, 1970).

There is evidence that some cases of pathological intoxication result from the action of the alcohol on the temporal lobe, as evidenced by EEG (Moyer, 1976). In one study (Marinacci, 1956, 1963) EEG records were taken on 402 patients who had been involved in incidents of confusion, abnormal behavior, destructive rage and other mental dysfunctions as a consequence of alcohol consumption. In 55 cases (14 %) anterior temporal lobe spikes were recorded, appearing from 25 seconds to 35 minutes after the first dose of alcohol. Eighteen patients had definite psychomotor episodes. No diagnostic abnormality was found in 347 cases (86 %). In another study (Bach-y-Rita et al., 1970) EEG spiking, after alcohol administration, was found in two out of ten patients, and in them only when the recordings were made from electrodes implanted in the temporal lobe in the region of the amygdala. So in the majority of cases no substrate changes can be demonstrated. Other factors must play a role.

In a Polish study of pathological intoxication (Pionkowski et al., 1975), it was determined that mental disturbances encountered could be divided in two groups:

(a) In psychoses, mental defects and epilepsy with character disorders, alcohol either intensified existing psychotic symptoms or provoked them; (b) In cases of organic brain syndromes, without clearly defined mental disturbances and in epilepsy without dementive or characterological changes, alcohol can cause intensification of intoxication symptoms. Even after a small dose of alcohol, severe consciousness disturbances, paranoidal states, and unprovoked aggressiveness with a high degree of agitation can appear.

Maletzky (1976) administered iv infusion of 25 % alcohol (400-1200 cc) to 22 male alcoholics. Behavior changes resembling a psychotic state were observed in 15 of the 22 subjects. These could be categorized into 4 groups: violent reaction (9 Ss); psychotic reaction (4 Ss) characterized by hallucinations, delusions, and distortions; mixed reaction (2 Ss), characterized by components of both of the above; and normal reaction (7 Ss), characterized by signs of alcohol intoxication alone (slurred speech, numbness, ataxia). Maletzky's data also suggest the existence of a discrete syndrome of pathological reaction to alcohol without reference to the amount of alcohol consumed.

Mendelson & Mello (1975) conclude that no association between blood alcohol levels, abnormal brain wave activity, and violence has been experimentally validated. Some patients

may exhibit increased aggressive behavior when intoxicated (Mendelson & Mello, 1974). However, other individuals become more friendly, while others show no change in behavior. “So-called ‘pathological intoxication’ is probably more closely related to host and environmental determinants than to specific effects of alcohol in the CNS”.

Neoplasms

Limbic brain tumors have been associated with increased irritability, abnormally aggressive behavior, and homicidal rage attacks (Kletschka, 1966; Goldstein, 1974; Mark & Sweet, 1974; Moyer, 1976; Mark, 1978).

The first to make precise clinicopathologic correlations with aggressive behavior was Alpers (1937), reporting on a patient with progressive development of uncontrollable rage behavior, who at postmortem examination showed atrophied hypothalamic nuclei due to a cyst. At the same time, Papez (1937) implicated the hippocampus in a substratum of emotional mechanisms, on the basis of his observations on Negri bodies, the lesions of rabies. Von Economo (1931) related lesions in the anterior hypothalamus to the aggressive and hyperkinetic states of encephalitis (cf. Goldstein, 1974).

Reports of tumors include Cushing (1929), Strauss & Kreschner (1935, 1936), Kreschner et al. (1936), Alpers (1937), Vonderahe (1940), Wortis & Maurer (1942), Hill (1944), Mulder & Daly (1952), Zeman & King (1958), Sano (1962), Malamud (1967), Reeves & Plum (1969), Sweet et al. (1969), Killefer & Stern (1970), Mullan (1971), Van Rensburg et al. (1975), Rothman et al. (1976).

The lesions and sites involved were: inferior posteromedial frontal abscess; oligodendroglioma of left hippocampus seeding into basal meninges and third ventricle; ectopic pituitary adenoma compressing the medial surface of the right temporal lobe; cystic schwannoma of the anteromedial temporal lobe; angioma of the temporal pole; subfrontal meningioma; temporal, optic chiasma, and gyrus cinguli gliomas, temporal glioblastoma, hypothalamic tumors, and colloid cyst of the third ventricle distorting the hypothalamic nuclei and causing their atrophy. The majority of these were postmortem findings.

Sano (1962) has reported on 1800 cases of brain tumor, 297 of which were in the limbic region. He concluded that increased irritability and rage attacks characterized patients with tumor involvement in the temporal lobe and anterior hypothalamus. Moyer (1976) concludes: “The evidence from brain tumors demonstrates again that man’s neural systems for aggression can be activated by internal physiological processes that result in the individual feeling and behaving in an inappropriate, hostile manner. As with other neurological dysfunctions, the behavior could result from an irritative focus of the tumor that activates some of the neurological mechanisms for aggression, or it could result from the destruction of inhibitory mechanisms”.

Interestingly, Martinius & Strunk (1979) recently reported the case of a 14-yr-old male who

brutally killed a child. In addition to other neuropsychiatric problems found in this patient, a circumscribed lesion lateral to the right nucleus amygdalae was discovered, which coincided anatomically with the glioma found in Charles Whitman, a mass murderer (described in Moyer, 1976).

The Encephalitides

Encephalitis lethargica became a worldwide epidemic by 1924. A part of the symptomatology frequently included a radical change in personality, characterized by a loss of impulse control, violent tempers and destructiveness, intractable, malicious spite and wanton cruelty, particularly in children from three to ten years of age. Primitive aggressive and sexual impulses were immediately carried into action, with subsequent serious and even murderous attacks on others, but occasionally upon themselves leading to gruesome self-mutilation (Von Economo, 1931; Greenbaum & Lurie, 1948; Brill, 1959; Slater & Roth, 1959). “It is as if the nervous organization subserving social behavior had become disorganized, leaving him at the mercy of the untamed instinctive drives” Slater & Roth observed.

After the acute phase of the disease process, about 54 % of the patients who showed mental symptoms continued to manifest them (Wilson, 1940). The brain areas chiefly affected by this disease are the basal ganglia, hypothalamus and the periaqueductal grey matter of the brain stem (Von Economo, 1931; Hill, 1954). Subacute and chronic forms of viral encephalitis tend to affect the medial portions of the temporal lobes, which are associated with the limbic system (Glaser & Pincus, 1969; Himmelhoch et al., 1970). The basal ganglia and the substantia nigra are also damaged, which accounts for the parkinsonian symptoms (Brill 1959). Moyer (1976) concludes: “Such a diffuse pattern of neuron necrosis tells us little about the specific neural mechanisms that underlie aggression in man. It should be noted, however, that the temporal lobe, the hypothalamus, and the mesencephalon are involved and have been shown to be important in the neural circuitry of aggression in lower animals. The characteristic behavior patterns could result from either the activation of the hostility systems by the inflammatory process or the loss of neurons in the neural systems that function to suppress activity in the aggression systems once it is started. The impulsive, compulsive aspect of the behavior appears to make the latter interpretation plausible”.

Rabies (Hydrophobia)

The classical symptoms of rabies (derived from Latin ‘rage’) include a.o. bizarre behavior changes, violent rages involving irrational assaults, extreme irritability, and – in former times – ultimately death. The viral infection, transmitted to the victim in the saliva of the rabid animal eventually affects the whole brain, with damage most extensive in the temporal lobe. Negri bodies containing the rabies virus are most concentrated in the cell bodies of the hippocampus, providing the definitive postmortem diagnosis (Papez, 1937; Lyght, 1966; Moyer, 1976).

Infantile Cerebral Injury

After craniocerebral injuries, patients, especially young men, often go through a phase of hyperactivity and aggressiveness, for instance, McLaurin & Heimer (1965) reported as 'restless and combative' 7 of 12 patients with contusions of the temporal lobe. Head injuries caused by falls or automobile accidents frequently result in loss of consciousness. As the individual regains consciousness he goes through a period of uncontrolled violence and aggression toward those around him (Mark & Ervin, 1970). The child's personality may show a complete reversal, characteristically emotional instability with aggressiveness, cruelty, and lack of impulse control (Strecker & Ebagh, 1924; Kasanin, 1929; Blau, 1937; Tuerk et al., 1975).

Szawczyk (1974) related infantile cerebral injury to incidence of manslaughter in a sample of 10 males 14-15 yrs-old. Physical sequelae, he concluded, were confounded by prevalence of broken homes, alcohol abuse, and self-worth conflicts. Sutorová & Trávníková (1976) compared 43 children (8-12 yrs-old) who had suffered brain concussion with a control group of healthy siblings. In 90 % of the concussed subjects, posttraumatic changes were shown in affectivity (lability of mood) and sociability (aggressive or evasive tendencies).

Comparing medical histories of 65 delinquent children referred to a court clinic with nonreferred delinquents, Lewis & Shanok (1979a,b) and Lewis et al. (1979a,b;

1983) concluded that early CNS trauma (perinatal difficulties), parental psychopathology, and social deprivation may be responsible for serious, often violent, delinquency.

Van Törne (1974) presents evidence of brain damage in self-mutilating children and adolescents.

Post-traumatic Personality Changes

Feuchtwanger (1923) studied patients with frontal gunshot wounds and described changes in mood and attitude including irritability. Patients with orbital lesions were said to have normal intelligence but severe personality changes; they were aggressive, demanding, and were prone to criminal offences (Benson & Geschwind, 1975).

In patients with moderate acute brain damage (e.g. concussion), occasionally episodes of aggressive behavior occur in which crimes of violence may be committed. This stage of clouded consciousness may take the form of an acute delirium, particularly in the old and alcoholic and in cases complicated by severe loss of blood and secondary infection. The patient experiences terrifying visual hallucinations and delusions. The picture as a whole is labile, fear, excitement and aggressiveness giving way with rapidity to a dazed, dreamy state or to a bland euphoria.

A pattern of post-traumatic personality change is that towards a querulous, morose mood with episodes of aggression and explosive anger. Features of the two types of change, the euphoric-disinhibited, and the morose aggressive, may of course be combined in the same patient (Slater & Roth, 1969).

If cerebral contusion is all extensive, it is likely to leave behind a change in personality, by which neurasthenic, hysterical and paranoid reactions are favored, and one is likely to see inadequate control of mood variations, both endogenous and psychogenically precipitated, tendencies towards hypochondriasis, irritability and aggressiveness, and a general lack of initiative and energy (Slater & Roth, 1969).

Minimal Brain Damage / Minimal Brain Dysfunction (MBD) in Children

MBD is apparently a catch-all diagnosis for a variety of minor physiological and neurological disorders of the CNS. The emotionality of MBD children shows four major types of dysfunction: increased lability, altered reactivity, increased aggressiveness, and dysphoria (Wender, 1971). The most common symptoms of MBD may be described as inappropriate poorly controlled behavior, shortened attention span, and intellectual deficit. It may also involve aggressive acts, tantrums, sexual displays, and verbal outbursts directed at others (Pincus & Glaser, 1966; Pincus & Tucker, 1974).

Wender (1971) indicates that, although many parents characterize their MBD children as 'angry', they usually describe irritability rather than hostility. He also emphasizes that when sadism or preoccupation with violence and death are present, it is not characteristic of MBD but suggests more serious pathology, notably borderline schizophrenic process.

Prechtl (1960) has described a choreiform syndrome in children who had suffered from perinatal hypoxia, and also a hypokinetic syndrome associated with hypotonia, drowsiness and apathy, alternating with irritability, which he also attributes to minimal brain damage.

Prechtl's syndrome appears to be clinically related to the hyperkinetic syndrome first described by Kramer & Pollnow (1932). This is a condition of persisting motor unrest which makes its appearance between the ages of 2 and 4 years. The child is often very aggressive, especially to his brothers and sisters, who may be brutally attacked without provocation. Parents complain of the cold unaffectionate character of the child. Epilepsy is a complication in about half of the cases, and EEG abnormalities, often localized in the temporal region, are relatively common (Ingram, 1956; Slater & Roth, 1969).

Hyperactivity may be the most prominent symptom. It is still a debated issue if and in what way hyperkinesis and aggression are related. Nagaraja (1976) studied 50 children in India referred for hyperactive behavior. She found hyperactivity to be associated with aggression, distractibility, destructiveness, excitability, and mental and emotional retardation.

Also de Sousa & de Sousa (1977) describe hyperkinesia as including aggressiveness. There is,

however, evidence of hyperactivity and aggression to be independent dimensions in hyperkinetic/MBD boys (Loney et al., 1978; Langhorne & Loney, 1979; Loney, 1980; Milich et al., 1982; cf. also Prinz et al., 1981; Stewart et al., 1981).

Elliott (1982) reports neurological findings in 286 15-80 yrs-old patients with a history of recurrent attacks of uncontrollable rage occurring with little or no provocation and dating from early childhood or from a physical brain insult at a later date. Objective evidence of developmental or acquired brain defects was found in 94 %. The most common abnormality was minimal brain dysfunction (41 %). Complex partial seizures of an epileptic nature occurred at some time in 30 %.

Lesions without Personality Transformation

In view of the limbic, especially hypothalamic and hippocampal involvement in virtually all syndromes in which 'aggression' is a prominent symptom, it may come as a surprise to find that a number of pathological states preferentially involve the hippocampus, frequently without seizures and without transformations in personality. Haymaker et al. (1958) and Goldstein (1974) mention: posticteric encephalopathy (sequel of kernicterus), herpes encephalitis, Wernicke syndrome, and Boeck sarcoid.

Neuroendocrinological Disorders

The Premenstrual Syndrome (PS). Typically the premenstruum is likely to be a recurrent phase of increased depression and irritability for many women. Direct and indirect observable phenomena of the PS include fluid retention and weight gain; the exacerbation of various allergic phenomena, notably asthma and urticaria; exacerbation of epileptic phenomena; changes in carbohydrate metabolism in the direction of hypoglycemia; aches and pains; negative affect, notably depression, anxiety, feelings of hostility and increased irritability during the late luteal phase of the menstrual cycle.

Several studies have documented the disproportionately high rate of occurrence of various types of disturbance during the premenstrual and menstrual phases. These have included: psychiatric hospital admissions; suicide attempts; neurotic and psychotic depressions; assaultive behavior in hospitalized psychiatric patients; accidents; and crimes of violence; as well as questionnaire studies of mood swings, irritability, tension, depression, etc., during the menstrual cycle (Greene & Dalton, 1953; Morton et al., 1953; Thorghele, 1957; Pennington, 1957; Dalton, 1959 et seq.; Shainess, 1961; Ribeiro, 1962; Coppen & Kessel, 1963; Southam et al., 1965; Sutherland & Stewart, 1965; Hamburg, 1966 et seq.; Mandell & Mandell, 1967; Janowsky et al., 1967; Moos, 1968; Hamburg et al., 1968; Iven & Bardwick, 1968; Gottschalk, 1969; Jacobs & Charles, 1970; Glass et al., 1971; Ellis & Austin, 1971; Jordheim, 1972; Parlee, 1973; Sachar, 1976; Moyer, 1976; Thiessen, 1976; Steiner & Carroll, 1977; d'Orban & Dalton, 1980; Woods et al., 1982).

Most of the described phenomena occur or are exacerbated during the midluteal and premenstrual phase. For example, d'Orban & Dalton (1980), to mention only one recent study, investigated 50 women (15-48 yrs-old) charged with crimes of violence and determined that 44 % of them committed their offense during the paramenstruum.

Parlee (1973) criticized the poor quality of most studies in this field, particularly those that suggest irritability, impulsivity, or criminality to be a mark of the premenstruum. Be that as it may, the existence of the premenstrual syndrome is beyond doubt.

Many theories of hormonal etiology of premenstrual mood disorder have appeared over the years. Moyer (1976) and Sachar (1976) list several attempts at explanation of the underlying physiology. These usually focus on estrogens and progesterone from the point of view of (1) absolute amounts of one hormone or the other; (2) relative amounts, estrogen/progesterone balance; (3) idiosyncratic sensitivity to estrogens; and (4) withdrawal reaction regarding either hormone.

The most forceful present proponent of the estrogen dominance theory is Dalton (1964), who argues from her successful experience in treating hundreds of premenstrual sufferers with progesterone injections.

Another hypothesis states that the cyclic increase of aldosterone, leading to increase of sodium and water retention, results in secondary neuronal irritability and consequent psychic symptoms. An explanation explored by Moyer (1976) relates to the tendency for a cyclic hypoglycemic reaction. There is evidence which implicates low blood sugar as a causal factor in hostility and crime (Vide infra). Furthermore, low blood sugar tends to intensify allergic reactions, a frequent component of which is an increase in aggressive tendencies (Randolph, 1962).

Women with dysphoric premenstrual symptoms have also been shown to have high prolactin levels especially in the premenstruum. Carroll & Steiner (1978) hypothesized that high prolactin levels associated with low progesterone levels may cause the symptoms of anxiety or irritable hostility.

The physiology of the premenstruum is neither a necessary nor a sufficient cause of any of the symptoms but rather must be regarded as a contributing cause and as one of a possible group of 'triggering' factors acting on an underlying predisposition. It must always be understood that a correlation between a biological phenomenon and a behavioral one does not necessarily imply an etiologic relationship in either direction. Moyer (1976) concludes his review of the PM as follows:

“Although the evidence certainly indicates an increase in feelings of irritability and hostility in some women during the premenstrual period, there is no good physiological explanation of the cause as yet. It must be recognized that the menstrual process is a phenomenon loaded with psychological meaning in most cultures. It therefore seems unlikely that the psychological changes associated with the period just prior to the onset of the menses are

exclusively or even primarily of physiological origin”.

Androgens and Aggression. Studies trying to relate aggressive behavior to circulating androgens, particularly testosterone (Persky et al., 1968, 1971; Kreuz & Rose, 1972; Rudd et al., 1968; Meyer-Bahlburg et al., 1973; 1974; Ehrenkranz et al., 1974; Brown & Davis, 1975; Rada et al., 1976; Kedenburg, 1977; Scaramella & Brown, 1978) are suggestive but as yet inconclusive. The association between (self-reports of) aggression and blood or urinary testosterone levels is not particularly robust in human males. Exogenous androgen administration may, however, enhance aggressive tendencies (e.g. Sands, 1954), while reduction of circulating androgen level, either by means of castration or by means of ‘chemical castration’ (antiandrogens, estrogens) may reduce some forms of aggressive behavior in men (see Moyer, 1975, and references therein).

The role of androgens in animal agonistic behavior is relatively well established. The limited data on hormonal correlates of aggressiveness in humans are consistent with the animal findings that the male hormones tend to heighten or potentiate aggressiveness while female hormones tend to decrease the probability of aggressive behavior.

Besides the gross hormonal differences between the sexes, there is suggestive evidence that individual differences in the levels of certain hormones may also contribute to individual behavioral differences. It is possible that in some individuals higher levels of testosterone under certain social circumstances might facilitate more intense states of anger and hostility or might catalyze the transition from the affective state of hostility and anger into the actual physical behavior and violent act (Rada et al., 1976; Moyer, 1976). Meyer-Bahlburg (1981) concludes in his recent review that “it seems likely that androgens play only a limited role among many other factors in the development of aggressive behavior since this has been found to be so on the level of subhuman primates... Yet, developmental effects of androgens on aggression seem highly likely”.

Other Hormonal Disorders. In Cushing’s disease, in which there is excessive secretion of adrenocortical hormones and very high circulating concentrations of these hormones, patients are often described as being easily aroused into anger. With successful treatment of the condition, this behavior tends to diminish (Hamburg, 1971).

High levels of circulating thyroid hormone, either exogenously administered or endogenously produced as in Graves’ disease (hyperthyroidism), are associated with a strong tendency to irritability of temper. Robertson (1875) already referred to the instability and difficulties in interpersonal relations attributed to the patient’s irritability. Graves and Basedow both referred to the nervous features of the disease which bears both their names. Among these are excessive movements and emotional excitability, quarrelsomeness, impatience, fury, and liability to explosive rage (Rome & Robinson, 1959; Sachar, 1975). With diminishing levels of thyroid hormone, irritability tends to diminish also (Hamburg, 1971).

Miscellaneous Brain Trauma

There are a variety of disorders that involve generalized or diffuse damage to the CNS, including cerebral arteriosclerosis, senile dementia, Korsakoff's syndrome, Huntington's chorea, and normal pressure hydrocephalus. These dysfunctions frequently present a common symptomatology referred to as chronic brain syndrome, which is characterized by memory deficit, orientation loss, and affective disturbances. There are wide fluctuations of mood and a general emotional instability, but the affective pattern is dominated by anger, rage, and increased irritability (Lyght, 1966). In senile dementia acute deliria may occur with severe restlessness, auditory and visual hallucinations, and paranoid suspicions which may lead to a violent assault (Slater & Roth, 1969).

Huntington's Chorea. There is some evidence that the premorbid personality of a person destined to develop Huntington's chorea tends to be more abnormal than that of individuals in the same family who are not carrying the responsible dominant gene. Thus alcoholism and criminality have been frequently recorded as having preceded the appearance of the characteristic neurological and psychiatric symptoms by a considerable interval. Other precursors are impulsive and unpredictable behavior, outbursts of explosive rage and violence, gross callousness, and sexual promiscuity (Minski & Guttman, 1938; Slater & Roth, 1969). The patient may become irritable, moody, ill-tempered, and show a morose and truculent discontent. He is oversensitive to slights and may express ideas of reference or paranoid delusions. In the choreic patient, against a background of slow, muddled thought, ill-directed attention and loss of initiative, there are often to be seen attacks of acute restlessness and irritability, and the patient if disturbed or interfered with is inclined to be spiteful, quarrelsome and violent (Slater & Roth, 1969).

Cerebral Arteriosclerosis. In association with general deficiency phenomena, there appear increased irritability and emotional instability. Such a state of irritability, which may open the scene as a prodromic manifestation, has been described long ago, not only in the slow progressive type of psychosis but also in cases where an apoplectic attack marks the onset of the psychosis. Irritability, emotionalism, explosive outbursts of weeping or laughter, or other sensory and even auditory hallucinations may indeed precede apoplectic attacks by weeks or months. Transitory ideas of persecution or of jealousy, but rarely paranoid states, may be encountered (Ferraro, 1959).

Trypanosomiasis. African sleeping sickness caused by *Trypanosoma gambiense* has long been known to involve the CNS producing sleepiness and simple dementia. Early in the illness changes of personality and behavior, especially in children and young people, are often the first sign of the infection; they may take the form of irritability, aggressiveness, querulousness, and be accompanied by intellectual deterioration. Paranoid features are frequent (Slater & Roth, 1959).

Malaria. Malignant tertian malaria may start with an attack which is predominantly cerebral in localization. Delirium and drowsiness followed by coma are the main symptoms. In the epileptiform twilight state excitation and violent behavior may occur. In these cases the formation of ‘malaria granulomata’ have been observed (Slater & Roth, 1969).

Typhus Fever. An infection with a characteristic cerebral pathology is the louse-borne typhus fever, of which widespread epidemics occurred in certain theaters during the last war. Subjective symptoms early in the disease are headache, irritability, giddiness and insomnia. After a short period of drowsiness, delirium with very vivid dream-like hallucinations commences, lasting over the whole period of raised temperature. States of panic with violence and restlessness, ideas of persecution and fantastic delusions are frequent. Neurological findings have been related to vascular cerebral lesions (Slater & Roth, 1969).

Neurosyphilitic Conditions: General Paralysis. In this condition, a disturbance of affect may set in relatively early. With the advance of the illness, irritability, loss of memory, and slovenliness become more obvious. Some patients commit sudden acts of violence. About 3 % of the cases of general paralysis present the schizophrenic form. Some patients in this group present paranoid delusions as the only outstanding clinical manifestation. A laborer brought a revolver to the factory and shot a fellow worker, imagining that the latter held a grudge against him (Bruetsch, 1959).

Neurosyphilitic Conditions: Juvenile Meningovascular Neurosyphilis. Whenever congenital syphilis involves the CNS, it may cause arrest or deterioration of the intellectual development of the child. Behavior disorders are occasionally observed including attacks of rage and impulsive acts. Irritability, restlessness, and depressive phases are often present (Bruetsch, 1959).

Gilles de la Tourette Syndrome. This is a neuropsychiatric disease with a childhood onset below age 16, characterized by chronic involuntary movements, obsessions, compulsions, utterances, echolalia, coprolalia, and aggressive behavior (Yaryura-Tobias, 1979).

Alcohol Hallucinosis and Amphetamine/Cocaine. Psychosis. Attacks on imagined persecutors, resulting from hallucinations, delusions and paranoid ideation, on the basis of chronic alcohol, cocaine or amphetamine abuse have been described. In his terror and panic, the patient is very dangerous, and trivial causes may lead to catastrophes of violence or murder (Thompson, 1959; Slater & Roth, 1969). Irritability has been described as a prodromal symptom in delirium tremens, as well as in chronic alcoholic mental deterioration (Thompson, 1959). Irritability is also a symptom of the deficiency syndrome after alcohol

withdrawal (Scholz, 1962).

Lead Encephalopathy. Children from poor environments with low magnesium and iron levels resulting from malnutrition, who live in the lead contaminated, atmosphere of urban centers may suffer from lead encephalopathy. Among juvenile delinquents over 60 % suffer from this condition according, to Aleksandrowicz (1976). Significant correlations between lead levels in hair samples and behavior problems including aggression in children (6-14 yrs) have been found by Marlowe & Errera (1982).

Vitamin B deficiency may also cause paranoia and violence (Zucker et al., 1981).

Klüver-Bucy syndrome (hypoaggressiveness, hypersexuality, orality) in man has been described by Shraberg & Weisberg (1978).

Hypoglycemia

Hypoglycemia (low blood sugar) may be caused by hyperinsulism, a therapeutic overdose of insulin, Addison's disease (hypoadrenalcorticalism), hypothyroidism, several pituitary dysfunctions, and by idiopathic blood sugar deficit. The symptoms of hypoglycemia are highly varied and are primarily psychological or neurological. Negative emotions predominate, including anxiety, depression, impulsivity, irritability and aggressiveness. The patient may be morose, asocial, sullen, and generally misanthropic. He is often rude and profane and the aggressive reaction may develop into a full-blown rage in which the individual becomes violent and destructive, attacking both objects and people, at times with fatal results (Aldersberg & Dolger, 1938-9; Greenwood, 1935; Rud, 1937; Ziskind & Bailey, 1937; Billig & Spaulding, 1947; Podolsky, 1964).

Wilder (1947) has summarized a wide variety of crimes comitted during a hypoglycemic state. See also Moyer (1976), Buckley (1979), Morton et al. (1953), Gloor (1967), Lyght (1966), Frederichs & Goodman (1969).

When the hypoglycemia is severe, the individual may become confused and disoriented and develop a fugue state in which he wanders aimlessly around the streets engaging in irrational and sometimes violent behavior. He may show complete amnesia for the period of fugue. Several irrational murders have been committed and attempted by hypoglycemic patients and in some cases the subject has been judged not guilty by reason of temporary insanity (Kepler & Moersch, 1937; Anderson, 1940; Hill & Sargant, 1943; Clapham, 1965; Schwadron, 1965). Many clinical studies have shown that a drop in glucose level results in an aggressive episode that can be promptly terminated by sugar intake (Moyer, 1976).

The reasons for increased aggressive tendencies during hypoglycemia are not yet clear.

According to Ervin (1969), hypoglycemia is a well-known provocation for epileptic foci, and the limbic system appears particularly sensitive to it. Thus patients with minimal deep temporal lobe damage may only have episodes of aggressive dyscontrol under conditions of hypoglycemia. There is considerable evidence that low blood glucose levels are associated with disruptions in the EEG pattern (Fabrykant & Pacella, 1948). Moyer (1976) hypothesized: "If there is a deficiency of glucose, the brain loses its fuel supply and is less able to extract oxygen from the blood. One result is a loss of function of some of the neural systems. If, as in the case of alcohol, the systems first affected are the ones related to neural inhibition, the hyperaggressiveness associated with hypoglycemia might be expected to occur".

Bolton (1971 et seq.) found evidence that the exceptionally high level of hostility and aggressiveness of the Qolla Indians of the Peruvian Andes may be related to their chronic hypoglycemia.

Virkkunen (1982) and Virkkunen & Huttunen (1982) found evidence for abnormal glucose tolerance among violent offenders. Offenders with antisocial personality revealed that blood glucose concentrations, having risen usually to a high level, fell to clinically significant hypoglycemia from which the return to the original basal values were slow. Those with an intermittent explosive disorder had much the same kind of abnormal curve as the antisocial personality group, but the return from reactive hypoglycemia to the original basal values was rapid.

The Allergic Tension-Fatigue Syndrome

The term 'allergic tension-fatigue syndrome' was introduced in 1954 to describe the allergic behavior pattern (Speer, 1954). It is important to note that behavior disturbances are only one of many possible allergic reactions and that all individuals with allergies do not show a behavioral alteration (Moyer, 1976). The most common descriptive term used in connection with this syndrome is 'irritability', or any of its many equivalents such as irascibility, cantankerousness, antagonism, combativeness, peevishness, etc. (Kahn, 1927; Rowe, 1930; Pounders, 1948; Davison, 1952; Schaffer, 1953; Speer, 1954, 1958, 1970; Coca, 1959; Randolph, 1959, 1962; Crook et al., 1961; Fredericks & Goodman, 1969; Mandell, 1969; Campbell, 1970).

The intensity of the symptoms may vary from a mild irritable reaction in which the individual is a little more easily annoyed than usual to a psychotic aggressive reaction. Many of the symptoms in allergies of the nervous system are also characteristic of the syndrome of MBD. Schneider (1945) has indicated that allergy is one of the most important factors, frequently unrecognized, in the production of the hyperkinetic child syndrome. Allergens that produce the syndrome are highly varied, while the sensitivity of the individual varies idiosyncratically.

The basic physiological cause of the irritable allergic reaction is not yet clear. It has been suggested that allergens have a direct effect on the nervous system, that cerebral edema causes the mental symptoms, and that circumscribed angioedema of the brain is involved in

the symptom formation. Moyer (1976) concludes his review of the syndrome: "It can be inferred from the preceding data that individuals with allergy-induced aggressive behavior may have angioedema in any one of several portions of the brain through which the neural system for irritable aggression courses. The pressure of the swelling may sensitize or activate those neural systems, or may deactivate some of the systems that have an inhibitory function, as appears to be the case with specific localized brain tumors".

Metabolic Disorders

"Certain metabolic disorders that diffusely involve the nervous system, many of them inborn errors of metabolism, are associated with aggressive personality types. The 'usual' temperament of children with Sanfillipo syndrome, Spielmeyer-Vogt syndrome, and phenylketonuria is aggressive and antagonistic as compared with children of the same age and similar mental states with Down syndrome, cretinism, and Dawson encephalitis (subacute sclerosing panencephalitis)" (Goldstein, 1974).

Spielmeyer-Vogt and Sanfillipo syndromes are diffuse neuronal storage disorders. In phenylketonuria synapse formation may be at fault. It is not exactly known how the personality is affected. Problems of frustration tolerance and automutilation in juvenile neuronal-ceroid-lipofuscinosis (Batten-Spielmeyer-Vogt disease) have been described by Steinhausen (1978).

Phenylketonuria (PKU). PKU is a disorder of metabolism inherited through a single autosomal recessive gene. The primary affect in homozygotes has been traced to a deficiency of the enzyme phenylalanine hydroxylase. The metabolic abnormalities lead to abnormal development of the CNS during its development and result in the characteristic severe mental retardation associated with the syndrome. In addition to the severe mental retardation, patients frequently show other aspects of abnormal behavior including seizures, clumsiness, anxiety, hyperactivity and irritability. Wright & Tarjan (1957) in their study of PKU indicated that none of the patients, "could be described as friendly, placid or happy". Uncontrollable temper tantrums were common and 10 % of the patients were destructive and had noisy psychotic episodes. The uncontrollable temper tantrums and irritability are frequently cited as the reasons for admitting such patients to institutions (Nash, 1975). The manner in which the metabolic disorder produces these effects is not known.

Down Syndrome. The disorder in Down syndrome ('mongoloid idiocy') concerns nucleic acids, anatomically evidenced in the karyotype as a trisomy. Children thus affected typically have gentle, placid personalities. However, during the third decade of life or shortly thereafter, many of them develop seizures and show the insidious onset of personality change, generally with the appearance of aggressive behavior (Haberland, 1969; Owens et al., 1971). A number of morphologic studies have disclosed the lesions of Alzheimer disease (neurofibrillary degeneration, senile plaques, and granulovacuolar degeneration) to be present

at this time.

“It is of interest, in light of the personality changes, that the earliest example of neurofibrillary degeneration appears in the hippocampal cortex and that granulovacuolar degeneration occurs shortly thereafter in these same areas and remains largely confined to the hippocampus. Senile plaque formation is observed at still a later time, but it too is generally severe in the cortex of the temporal lobes. The cingulate gyrus shows notable atrophy underscoring the involvement of the limbic system’ (Goldstein, 1974). The neuropathological lesions appear to be directly related to the aggressive behavior shown by patients with Down syndrome (Nash, 1975).

The Leach-Nyhan Syndrome. Hyperuricemia, or X-linked uricaciduria, a type of mental retardation associated with an elevation of the blood uric acid, has been first described by Lesch & Nyhan (1964). The Lesch-Nyhan syndrome includes spastic cerebral palsy, choreoathetosis, bizarre, intense and compulsive self-mutilation, and aggressive behavior (Lesch & Nyhan, 1964; Nyhan et al., 1969; Nyhan, 1968, 1970, 1976; Dizmang & Cheatham, 1970; Nash, 1975; Moyer, 1976; Fernald, 1976; Christie et al., 1982). Genetically the condition is considered to be a disease of the male transmitted as an X-linked recessive character (Dizmang & Cheatham, 1970). The condition is familial (Bakwin & Bakwin, 1966).

The self-mutilating behavior is correlated with an abnormality of purine metabolism. The behavior can be induced in rabbits by administration of theophylline, in mice by clonidine, and in rats by large doses of caffeine. It is conceivable that internal accumulation of CNS-stimulants such as the environmental purines (in coffee, tea, etc.) may have some bearing on individual differences in human aggressiveness (Hamburg, 1971).

The metabolic defect responsible for the Lesch-Nyhan syndrome is a gross deficiency of the enzyme hypoxanthine-guanine phosphoribosyl transferase (HGPRT). The mechanism by which the deficiency in HGPRT is related to the compulsive self-destructive and aggressive behavior is not known (Nash, 1975).

Other Findings. Yaryura-Tobias & Neziroglu (1975, 1978) describe a hypothalamic dysfunction related to a serotonergic imbalance in female patients. Symptomatology includes aggressive behavior, self-mutilation, abnormal EEGs (dysrhythmias), and altered glucose tolerance curves. The authors propose a disturbance in tryptophan metabolism, caused by an increase of insulin output.

Changes in the pattern of catecholamine secretion correlate with convictions for violence in maximum security hospital detainees (Woodman et al., 1978; Woodman, 1979). Subjects convicted of sexual offenses showed significantly greater cAMP (cyclic adenosine monophosphate) excretion than other patients and normal controls (Woodman & Hinton, 1978). In addition, violent maximum security patients had significantly lower adrenalin, significantly higher noradrenaline, and very significantly higher N/A ratios than nonviolent

controls in urine samples (Woodman et al., 1977).

Sandler et al. (1978) found that plasma concentrations of free and conjugated phenylacetic acid, the major metabolite of phenylethylamine, were higher in ten prisoners serving long terms of imprisonment for violent crimes than in pair-matched non-violent control prisoners. The investigators believe that phenylethylamine overproduction may represent a compensatory response by the body in its attempt to curb aggressive tendencies present as a result of some as yet unknown functional derangement.

Brown et al. (1982) examined the life history of aggression and suicidal behavior (regarded as a specific aggression-related behavior) in 12 17-32 yr-old males with borderline personality disorders. Histories of aggressive behaviors and of suicide attempts were significantly associated with each other, and each was significantly associated with lower 5-HIAA levels (5-hydroxyindoleacetic acid, a serotonin metabolite).

Virkkunen (1979) reported that lower serum cholesterol levels were characteristic of men with antisocial personality. This finding, however, could not be confirmed by Stewart & Stewart (1981).

Conclusions

(1) In a fairly recent review article on 'Brain Research and Violent Behavior', Goldstein (1974) reported that there is no unanimity of opinion concerning the morphophysiological substrates that underlie abnormal aggressive behavior in animals or man.

There was, however, general agreement that derangements of behavior probably have morphophysiological substrates that involve, in some differential and selective fashion, predominantly brain structures rostral to the rhombencephalon. In general terms, portions of the neuraxis caudal to the midbrain may be regarded as being concerned largely with neural mechanisms that maintain and support essential biological functions (i.e., respiration, cardiovascular regulation), and control, regulate, and supply effector mechanisms. Portions of the CNS rostral to the rhombencephalon appear to contain neural substrates concerned with goal-directed behavior and the motivational and emotional concomitants that make such behavior possible. Stated broadly and simply, it is believed that impulses generated in sensory systems, the cerebral cortex, and still undetermined neural structures may activate triggering mechanisms that in turn excite visceral and somatic systems whose activities in concert provide the physiologic expression of aggressive behavior. This conclusion is still valid.

(2) There is also general agreement that the limbic lobe and limbic system occupy central positions in the neural mechanism that governs behavior and emotion. In man, as well as in infrahuman organisms, lesions in these areas may lead to pathological changes in motivational, emotional and behavioral systems, particularly those associated with fear and aggression (fight/flight), and/or may lead to long-term changes in personality make-up in the direction of dysphoric states, hostility and morose moods. Pathologies on an organic basis

may also lead to psychopathological symptom formation, e.g. paranoid ideation.

In general, the conclusion reached by Moyer (1976) can still be regarded as valid. He stated: “There are a variety of pathologies of the brain resulting in changes that increase the probability of activity in neural substrates for aggression, with a consequent lowering of the threshold for hostile thoughts and behavior. Subcortical tumors in various portions of the limbic system result in a gradual change in the individual’s personality, with irritability as one of the predominant symptoms... Several disorders, including miscellaneous brain trauma, rabies, and encephalitis, result in damage to particular portions of the limbic system with a consequent personality alteration in which inadequate control of aggressive impulses predominates. An individual’s behavior may range from a moderate loss of impulse control to pathological aggressive and even homicidal tendencies... In certain individuals with temporal lobe epilepsy the spontaneous activation of the neural systems for aggression results in periodic aggressive episodes. There is some reason to believe that the neural substrates underlying the convulsive process are not the same as those responsible for the aggressive outbursts. Surgery that excises a portion of the subcortex may result in an alleviation of the hostility without affecting the convulsions or vice versa. Some of the pharmaceutical agents that are useful in the control of the seizures may exacerbate the aggressive behavior, and medication to reduce the hostility may increase the seizure tendency”.

(3) Phenomenologically, two distinct types or patterns of ‘aggression’ are discernible in the clinical context: (a) paroxysmal rage outbursts of an impulsive, relatively uncontrollable nature, evidently related to (lesions of) an ‘offensive aggression’ circuitry in the limbic system. Lion (1974) and Lion & Penna (1974) report that the most common form of deviant human aggression occurs in the class of personality disorders labelled variously as Explosive, Passive-aggressive, or Antisocial. The bulk of prisoners in the USA are made up of individuals with this diagnosis. They demonstrate recurring and labile hostile episodes, rage attacks, temper outbursts; some have serious problems of impulse control and others, though able to plan and premeditate criminal activity, are bothered by paroxysmal aggressive and impulsive acts which severely hamper their actions and lives.

(b) The other pattern manifests assaultiveness, combativeness and hostility due to delusional beliefs, hallucinations, and paranoid ideation. The eventual violence used is essentially self-defensive and panicky – the preemptive strike of a ‘cornered cat’ – preceded and accompanied by overwhelming emotions of fear and terror (Indeed, fear might well be the most forceful precipitant of human violence, pathological or not). It is related to psychopathological symptom formation on the basis of more diffuse lesions or substrate changes of the CNS. “This class of aggressive patients is much different from the personality disordered-group – the patients’ aggressiveness stems from not poor impulse control, but from disordered and aberrant thinking” (Lion & Penna, 1974).

(4) Neuropathologically males are, as in so many areas of life, by far the more vulnerable sex.

(5) Within the context of human political violence (war, civil war, state terror, terrorism), criminal and familial-domestic violence, ‘pathological aggression’, whether on an organic

basis or not, is but a minor problem.

REFERENCES

- AIRD R. & T. YAMAMOTO (1966) Behavior disorders of childhood. EEG & Clin. Neurophysiol., 21, 148-55.
- ALDERSBERG D. & H. DOLGER (1938-9) Medico-legal problems of hypoglycemic reactions in diabetes. Annals Internal Medicine, 12, 1804-5.
- ALEKSANDROWICZ J. (1976) Si vis salutem para pacem. Peace Res. 81 3, 67-9.
- ALPERS B. (1937) Relation of the hypothalamus to disorders of personality: report of a case. Arch. Neurol. Psychiat. 361, 291-503.
- ALSTRÖM C. (1950) A study of epilepsy in its clinical, social and genetic aspects. Acta Psychiat. Neurol. Scand., Suppl. 63.
- ANDERSON E. (1940) Psychiatric complications of hypoglycemia in children. Lancet, 2, 329-31.
- ANDY O. & M. JURKO (1972) Hyperresponsive syndrome. in: Hitchcock et al. (Eds).
- ARAI N.; Y. SHIBATA; A. AKAHENE & O. MIYAZAWA (1966) Electroencephalographic analysis of behavior problem children. Acta Criminologica et Medicina Legalis Japonica, 32, 143-52.
- ARCHER J. (1976) The organization of aggression and fear in vertebrates. in: Bateson & Klopfer (Eds).
- ARTHURS R. & E. CAHOON (1964) A clinical and electroencephalographic survey of psychopathic personality. Amer. J. Psychiat. 120, 875-7.
- ASSAEL M.; R. KOHN-RAZ & S. ALPERN (1967) Development analysis of EEG abnormalities in juvenile delinquents. Dis. Nerv. System 28, 49-54.
- BACH-Y-RITA G.; J. LION & F. ERVIN (1970) Pathological intoxication: clinical and electroencephalographic studies. Amer. J. Psychiat. 127, 698-703.
- BACH-Y-RITA G.; J. LION; C. CLIMENT & F. ERVIN (1971) Episodic dyscontrol: a study of 130 violent patients. Amer. J. Psychiat. 127, 11, 1473-8.
- BAKWIN H. & R. BAKWIN (1966) Clinical management of behavior disorders in children. Saunders, Philadelphia.

- BANAY R. (1944) Pathologic reaction to alcohol. Vol. I: Review of the literature and original case reports. *Quart. J. Studies on Alcohol* 4, 580-605.
- BARASH O. (1977) *Sociobiology and behavior*. Elsevier, Amsterdam.
- BASTIAANS J. (1969) The role of aggression in the genesis of psychosomatic disease. *J. Psychosom. Res.* 13, 307-14.
- BATESON P. & P. KLOPFER (Eds) (1976) *Perspectives in Ethology*. Vol. 2. Plenum, NY.
- BAYRAKAL S. (1965) The significance of electroencephalographic abnormality in behavior-problem children. *Canad. Psychiat. Ass.* 3., 10, 367-92.
- BEHAR O. & M. STEWART (1982) Aggressive conduct disorder of children: the clinical history and direct observations. *Acts Psychiat. Scand.* 65, 3, 210-20.
- BENDER L. (1953) *Aggression, hostility, and anxiety in children*. Thomas, Springfield.
- BENSON D. & N. GESCHWIND (1975) Psychiatric conditions associated with focal lesions of the central nervous system. in: *American Handbook of Psychiatry*, Vol. 4. Basic Books, NY.
- BILLIG H. & C. SPAULDING (1947) Hyperinsulism of menses. *Industrial Med.* 16, 336-9.
- BLACKBURN R. (1975) Aggression and the EEG: A quantitative analysis. *J. Abnorm. Psychol.* 64, 4, 358-65.
- BLAU A. (1937) Mental changes following head trauma in children. *Arch. Neurol. & Psychiat.* 35, 723-69.
- BLUMER O. (1974) Organic personality disorders. in: Lion (Ed).
- BLUMER O. (1975) Temporal lobe epilepsy and its psychiatric significance. in: F. Benson & O. Blumer (Eds.) *Psychiatric aspects of neurologic disorders*. Grune & Stratton, NY.
- BLUMER O. (1976) Epilepsy and violence. in: Madden & Lion (Eds).
- BOLTON R. (1971) Aggression in Qolla society. *Doct. Diss. Univ. Cornell, Ithaca NY.*
- BOLTON R. (1973) Aggression and hypoglycemia among the Qolla: a study in psychobiological anthropology. *Ethnol.* 12, 227-57.
- BOLTON R. (1976) Hostility in fantasy: a further test of the hypoglycemia-aggression hypothesis. *Aggress. Behav.* 2, 4, 257-74.

- BOWMAN K. & E. JELLINEK (1941) Alcoholic mental disorders. *Quart. J. Studies on Alcohol* 2, 312-
- BREWER C. (1971) Homicide during a psychomotor seizure. *Med. J. Austr.* 1, 857-9.
- BRILL H. (1959) Postencephalic psychiatric conditions. in: *American handbook of psychiatry*. Vol. 2, Basic Books, NY.
- BROWN C. et al. (1979) Human aggression and its relationship to cerebrospinal fluid, 5-hydroxyindoleacetic acid, 3-methoxy-4-hydroxyphenylglycol, and homovanillic acid. in: Sandler (Ed).
- BROWN C. et al. (1982) Aggression, suicide, and serotonin: relationships of CSF amine metabolites. *Amer. J. Psychiat.* 139, 6, 741-6.
- BROWN W. & C. DAVIS (1975) Serum testosterone and irritability in man. *Psychosom. Med.* 37, 87-
- BROWNE-MAYERS A. & L. STRAUB (1953) Metrazol activation of electroencephalograms in psychiatric patients. *J. Nerv. Ment. Dis.* 117, 151-
- BRUETSCH W. (1959) Neurosyphilitic conditions: general paralysis, general paresis, dementia paralytica, chronic brain syndrome associated with syphilitic meningoencephalitis. in: *American Handbook of Psychiatry*, Vol. 2, Basic Books, NY.
- BUCKLEY R. (1979) Hypoglycemia, temporal lobe disturbance and aggressive behavior. *J. Orthomolecular Psychiat.* 8, 3, 188-92.
- BUIKHUISEN W. (1982) Aggressive behavior and cognitive disorders. *Internat. J. Law & Psychiat.* 5, 2, 205-17.
- CAMPBELL M. (1970) Allergy and behavior: neurologic and psychic syndromes. in: F. Speer (Ed), *Allergy of the nervous system*. Thomas, Springfield.
- CARROLL B. & M. STEINER (1978) The psychobiology of premenstrual dysphoria: the role of prolactin. *Psychoneuroendocrinol.* 3, 2, 171-80.
- CARVALHAL RIBAS J. et al. (1974) Behavior disorders with predominance of aggressiveness, irritability, impulsiveness, and instability: clinical electroencephalographic study of 100 cases. *Arquivos de Neuro-Psiquiatria* 32, 3, 187-94.
- CHRISTIANI K.; R. SIEBERT & B. VÖLKER (1977) Electroencephalographic examinations in emotionally disturbed children and adolescents. *Fortschritte der Neurologie, Psychiatrie und ihre Grenzgebiete* 45, 4, 321-30.

- CHRISTIE R. et al. (1982) Lesch-Nyhan disease: clinical experience with nineteen patients. *Developm. Med. & Child Neurol.*, 24, 3, 293-306.
- CLAPHAM B. (1965) An interesting case of hypoglycemia. *Medico-Legal J.* 33, 72-3.
- COCA A. (1959) *The pulse test.* Lyle Stuart, NY.
- COHN R. & J. NARDINI (1958) The correlation of bilateral occipital slow activity in the human EEG with certain disorders of behavior. *Amer. J. Psychiat.*, 115, 44-54.
- COLEMAN L. (1974) Perspectives on the medical research of violence. Paper Ann. Conv. Orthopsychiat. Ass.
- COPPEN A. & N. KESSEL (1963) Menstruation and personality. *Brit. J. Psychiat.* 109, 711-21.
- CRAIG W. (1928) Why do animals fight? *Internat. J. Ethics* 31, 246-78.
- CROOK W. et al. (1961) Systemic manifestations due to allergy: report of fifty patients and a review of the literature on the subject (sometimes referred to as allergic toxemia and the allergic tension-fatigue syndrome). *Pediatrics* 27, 790-99.
- CURRIE S. et al. (1971) Clinical course and prognosis of temporal lobe epilepsy: a survey of 666 patients. *Brain* 94, 173-90.
- CUSHING H. (1929) *The pituitary body and hypothalamus.* Thomas, Springfield.
- DALTON K. (1959) Menstruation and acute psychiatric illness. *Brit. Med. J.*, 1, 148-9.
- DALTON K. (1960) Schoolgirls' misbehavior and menstruation. *Brit. Med. J.*, 2, 1647-9.
- DALTON K. (1961) Menstruation and crime. *Brit. Med. J.*, 2, 1752-3.
- DALTON K. (1964) *The premenstrual syndrome.* Thomas, Springfield.
- DALTON K. (1980) Clinical aggression in premenstrual syndrome. Paper ISRA Conf. Haren, 1980.
- DAVISON H. (1952) Allergy of the nervous system. *Quart. Rev. Allergy & Applied Immunol.* 6, 157-88.
- DENNEN J. van der (1977) *Neuropathology, psychopathology, and psychosomatics in relation to aggression and anger.* 3 vols. Polemologisch Inst., Univ. Groningen.

DENNEN J. van der (1980) Problems in the concepts and definitions of aggression, violence, and some related terms. *Polemologisch Inst., Univ. Groningen, Publ.* 1980/14.

DENNEN J. van der & P. van der MOLEN (1981) Violent aggression as a social unskill; Notes on the psychopathology of everyday-life. Paper ISRA Conf. Strasbourg, 1981.

DENNEN J. van der & P. van der MOLEN (1981) Striving, playing and learning: a novel conceptual model of coping skills. Paper ISRA Conf., Strasbourg, 1981.

DENNERSTEIN L. & G. BURROWS (1979) Affect and the menstrual cycle. 3. Affective Disorders 1, 2, 77-92.

DETRE T. & R. FELDMAN (1963) Behavior disorders associated with seizure states: pharmacological and psychosocial management. in: G. Glaser (Ed) EEG and behavior. Basic Books, NY.

DETRE T.; O. KUPFER & S. TAUB (1975) The nosology of violence. in: Fields & Sweet (Eds).

DINNEN A. (1971) Homicide during a psychomotor seizure. *Med. J. Austr.* I, 1353.

DIZMANG L. & C. CHEATHAM (1970) The Lesch-Nyhan syndrome. *Amer. J. Psychiat.* 127, 5, 671-7.

DOWZENKO A. et al. (1975) Clinical characteristics of epileptics admitted during one year to the hospital for nervous and mental diseases in Pruszkow. *Psychiatria Polska* 9, 4, 389-98.

DURHAM W. (1976) Resource competition and human aggression. Part I: A review of primitive war. *Quart. Rev. Biol.*, 51, 385-415.

ECONOMO C. von (1931) Encephalitis lethargica: its sequelae and treatment. Oxford Univ. Press, London.

EEG-OLOFSSON O. (1970) The development of the electroencephalogram in normal children and adolescents from the age of one through 21 years. *Acta Paediat. Scand., Suppl.* 208.

EHRENKRANZ J.; E. BLISS & M. SHEARD (1974) Plasma testosterone: correlation with aggressive behavior and dominance in man. *Psychosom. Med.* 36, 5, 469-75.

EIBL-EIBESFELDT I. (1974) Phylogenetic adaptations as determinants of aggressive behavior in man. in: J. de Wit & W. Hartup (Eds), Determinants and origins of aggressive behavior. Mouton, The Hague.

ELLINGSON R. (1954) Incidence of EEG abnormality among patients with mental disorders

of apparently non-organic origin: critical review. *Amer. J. Psychiat.* 111, 263-75.

ELLIOTT F. (1976) Neurological factors in violent behavior (The dyscontrol syndrome). *Bull. Amer. Acad. Psychiat. & the Law* 4,4, 297-315.

ELLIOTT F. (1982) Neurological findings in adult minimal brain dysfunction and the dyscontrol syndrome. *J. Nerv. Ment. Dis.* 170, II, 680-7.

ELLIS D. & P. AUSTIN (1971) Menstruation and aggressive behavior in a correctional center for women. *J. Criminal Law, Criminol. & Police Sci.* 62, 388-95.

ERVIN F. (1967) Brain disorders IV. Associated with convulsions (epilepsy). in: A. Freedman & M. Kaplan (Eds), *Comprehensive textbook of psychiatry*. Williams & Wilkins, Baltimore.

ERVIN F. (1969) The biology of individual violence: an overview. in: D. Mulvihill, M. Tumin & L. Curtis (Eds) *Crimes of violence*. U.S. Government Printing Office, Washington DC.

ERVIN F.; A. EPSTEIN & H. KING (1955) Behavior of epileptic and non-epileptic patients with 'temporal spikes'. *Arch. Neurol. Psychiat.* 749 488-97.

ERVIN F.; V. MARK & W. SWEET (1969) Focal cerebral disease, temporal lobe epilepsy and violent behavior. *Transact. Amer. Neurol. Assoc.* 94, 253-6.

ERVIN F. ; J. DELGADO; V. MARK & W. SWEET (1969) Rage: a para-epileptic phenomenon? *Epilepsia* 10, 415-20.

FABRYKANT M. & B. PABELLA (1948) Association of spontaneous hypoglycemia with hypocalcemia and electrocerebral dysfunction. *Arch. Internal Med.* 81, 164-202.

FALCONER D. & D. TAYLOR (1968) Surgical treatment of drug resistant temporal lobe epilepsy due to mesial temporal sclerosis: etiology and significance. *Arch. Neurol.* 19, 353-61.

FALCONER M.; D. HILL; A MEYER & J. WILSON (1958) Clinical, radiological, and EEG correlations with pathological changes in temporal lobe epilepsy and their significance in surgical treatment. in: M. Baldwin & P. Bailey (Eds), *Temporal lobe epilepsy*. Thomas, Springfield.

FALCONER M. (1967) Some functions of the temporal lobes with special regard to affective behavior in epileptic patients. *J. Psychosom. Res.* 9, 25-

FENTON G. & E. UDWIN (1965) Homicide, temporal lobe epilepsy and depression: a case report. *Brit. J. Psychiat.* 111, 304-6.

- FERNALD C. (1976) The Lesch-Nyhan syndrome: cerebral palsy, mental retardation, and self-mutilation. *J. Pediatric Psychol.* 1, 3, 51-5.
- FERRARO A. (1959) Psychoses with cerebral arteriosclerosis. in: *American Handbook of Psychiatry*, Vol. 2, Basic Books, NY.
- FEUCHTWANGER E. (1923) *Die Funktionen des Stirnhirns*. Springer, Berlin.
- FIELDS W. & W. SWEET (Eds) (1975) *Neural bases of violence and aggression*. W.H.Green, St. Louis.
- FLOR H. (undated) *Psychosis and temporal lobe epilepsy*. M.D.Thesis. Univ. Edinburgh.
- FLOR H. (1972) Ictal and interictal psychiatric manifestations in epilepsy: specific or non-specific? A critical review of some of the evidence. *Epilepsia* 13, 773-83.
- FLOR H. (1973) Psychiatric syndromes considered as manifestations of lateralized temporal-limbic dysfunction. in: Laitinen & Livingston (Eds).
- FLOR H. (1974) Psychosis, neurosis and epilepsy. *Brit. J. Psychiat.* 124, 144-50.
- FREDERICHS C. & H. GOODMAN (1969) *Low blood sugar and you*. Constellation Internat., NY.
- FREUD S. (1928) Dostoevsky and parricide. in: *Coll. Papers Vol. V*, 1950.
- FROMM E. (1973) *The anatomy of human destructiveness*. Holt, Rinehart & Winston, NY.
- GALTUNG J. (1964) A structural theory of aggression. *J. Peace Res.* 1, 95-119.
- GASTAUT H. (1953) So-called 'psychomotor' and 'temporal' epilepsy. *Epilepsia*, 2, 59-75.
- GASTAUT H. (1954) *The epilepsies: their electro-clinical correlates*. Thomas, Springfield.
- GASTAUT H. & Y. GASTAUT (1951) *Corrélations électroencéphalographiques et cliniques à propos de 100 cas d'épilepsie dite 'psychomotrice' avec foyers sur la région temporale du scalp*. *Rev. Oto-Neuro-Ophtal.* 23, 257-62.
- GASTAUT H.; C. MORIN & N. LESÈVRE (1955) *Etude du comportement des épileptiques psychomoteurs dans l'intervalle de leurs crises; les troubles de l'activité globale et de la sociabilité*. *Ann. Med. Psychol.* 113, 1-27.
- GASTAUT H.; H. TERZIAN; R. NAQUET & K. LUSCHNAT (1952) *Corrélations entre les 'automatismes' des crises temporales et les phénomènes électroencéphalographiques qui les*

accompagnent. *Rev. Neurol (Paris)*, 86, 678-82.

GESCHWIND N. (1975) The clinical setting of aggression in temporal lobe epilepsy. in: Fields & Sweet (Eds).

GIBBENS T. (1956) Sane and insane homicide. *J. Criminal Law, Criminol. & Police Sci.* 49, 110-15.

GIBBENS T.; D. POND & D. STAFFORD-CLARK (1959) Follow-up study of criminal psychopaths. *J. Ment. Sci.* 105, 108-15.

GIBBS F. (1951) Ictal and non-ictal psychiatric disorders in temporal lobe epilepsy. *J. Nerv. Ment. Dis.* 113, 522-8.

GIBBS F. (1955) Subjective complaints and behavior disturbances associated with 14/6 per second positive spikes. *EEG & Clin. Neurophysiol.* 7, 315-6.

GIBBS F. (1956) Clinical correlates of 14 and 6 per second positive spikes. *EEG & Clin. Neurophysiol.* 8, 149.

GIBBS E. & F. GIBBS (1951) Electroencephalographic evidence of thalamic and hypothalamic epilepsy. *Neurol.* 1, 136-44.

GIBBS F. & E. GIBBS (1963) Fourteen and six per second positive spikes. *EEG & Clin. Neurophysiol.* 15, 4.

GIBBS F.; E. GIBBS & W. LENNOX (1937) Epilepsy: a paroxysmal cerebral dysrhythmia. *Brain* 60, 377-88.

GIBBS E.; GIBBS F. & B. FUSTER (1948) Psychomotor epilepsy. *Arch. Neurol. Psychiat.* 60, 331-9.

GIGLI G.; S. MAZZA; P. BERGONZI & G. MENNUNI (1981) Epilessia, aggressività e criminalità. *Archivio di Psicologia, Neurologia & Psichiatria* 42, 2, 273-84.

GIRGIS M. (1977) Social implications of the dyscontrol syndrome: neuropsychiatric correlates. *Austr. & N. Zealand J. Psychiat.* 11, 4, 245-9.

GIRGIS M. & L. KILOH (Eds)(1960) *Limbic epilepsy and the dyscontrol syndrome.* Elsevier, Amsterdam.

GLASER G. (Ed) (1963) *EEG and behavior.* Basic Books, NY.

GLASER G. (1967) Limbic epilepsy in childhood. *J. Nerv. Ment. Dis.* 144, 391-7.

GLASER G. (1975) Epilepsy: neuropsychological aspects. in: American Handbook of Psychiatry, Vol. 4. Basic Books, NY.

GLASER G. & D. PINCUS (1969) Limbic encephalitis. *J. Nerv. Ment. Dis.* 149, 59-

GLASER G.; R. NEWMAN & R. SCHAFER (1963) Interictal psychosis in psychomotor-temporal lobe epilepsy: an EEG psychological study. in: Glaser (Ed).

GLASS G.; G. HENINGER; M. LANSKY & K. TALAN. Psychiatric emergency related to the menstrual cycle. *Amer. J. Psychiat.* 128, 705-11.

GLOOR P. (1960) Amygdala. in: J. Field, H. Magoun & V. Hall (Eds) *Handbook of Physiology*. Vol. II: Neurophysiology. Williams & Wilkins, Baltimore.

GLOOR P. (1967) Discussion following Kaada in: C. Clemente & D. Lindsley (Eds), *Aggression and defense: neural mechanisms and social patterns*. Univ. California Press, Los Angeles.

GLOOR P. (1972) Temporal lobe epilepsy: its possible contribution to the understanding of the functional significance of the amygdala and of its interaction with neocortical-temporal mechanisms. in: B. Eleftheriou (Ed), *The neurobiology of the amygdala*. Plenum, NY.

GOLDSTEIN M. (1974) Brain research and violent behavior. *Arch. Neurol.* 30, 1, 1-35.

GONZALEZ DE RIVERA J. (1981) Psychopathology of epilepsy. *Psiquis: Revista de Psiquiatria, Psicología y Psicosomática* 2, 2, 64-80.

GOTTSCHALK L. (1959) The measurement of hostile aggression through the content analysis of speech some biological and interpersonal aspects. in: S. Garattini & E. Sigg (Eds), *Aggressive behaviour*. Wiley, NY.

GOTTSCHALK L.; S. KAPLAN; G. GLESER & C. WINGET (1961) Variations in the magnitude of anxiety and hostility with phases of the menstrual cycle. *Psychosom. Med.* 23, 448.

GRAHAM K. (1980) Theories of intoxicated aggression. *Canad. J. Behav. Sci.* 12, 2, 141-58.

GRAHAM P. & M. RUTTER (1958) Organic brain dysfunction and child psychiatric disorders. *Brit. Med. J.* 3, 695-700.

GREEN J. (1960) The hippocampus. in: J. Field, H. Magoun & V. Hall (Eds), *Handbook of Physiology*. Vol. II: Neurophysiology. Williams & Wilkins, Baltimore.

GREEN J. (1951) Association of behavior disorder with an electroencephalographic focus in children without seizures. *Neurol.* 11, 337-44.

- GREEN J. & H. STEELMAN (Eds) (1956) *Epileptic seizures*. Williams & Wilkins, Baltimore.
- GREENBAUM J. & L. LURIE (1948) Encephalitis as a causative factor in behavior disorders of children. *JAMA* 136, 923-30.
- GREENE R. & K. DALTON (1953) The premenstrual syndrome. *Brit. Med. J.* 1, 1007-14.
- GREENWOOD J. (1935) Hypoglycemia as a cause of mental symptoms. *Pennsylvania Med. J.* 39, 12-16.
- GROEN J. (1957) Psychosomatic disturbances as a form of substituted behavior. *J. Psychosom. Res.* 2, 85-
- GROEN J. (1972) The study of human aggression. *Psychotherapy & Psychosom.* 20, 5, 312-5.
- GROSS H. & E. KALTENBÄCK (1975) Epilepsy and delinquent aggressive behavior. *Nervenarzt* 46, 8, 472-4.
- GROSS M. (1971) Violence associated with organic brain disease. in: J. Fawcett (Ed), *Dynamics of violence*. Amer. Med. Assoc., Chicago.
- GROSS M. & W. WILSON (1964) Behavior disorders of children with cerebral dysrhythmia. *Arch. Gen. Psychiat.* 11, 610-19.
- GUNN J. (1978) Epileptic homicide: a case report. *Brit. J. Psychiat.* 132, 510-13.
- GUNN J. & J. BONN (1971) Criminality and violence in epileptic prisoners. *Brit. J. Psychiat.* 118, 337-43.
- GUNN J. & G. FENTON (1969) Epilepsy in prisons: a diagnostic survey. *Brit. Med. J.* 4, 326-9.
- GUNN J. & G. FENTON (1971) Epilepsy, automatism and crime. *Lancet* 1, 1173-6.
- HAAS A. de (1963) Epilepsy in criminality. *Brit. J. Criminol.* 3, 248-57.
- HABERLAND C. (1969) Alzheimer's disease in Down syndrome: clinical-neuropathological observations. *Acta Neurol. Belg.* 69, 369-80.
- HAMBURG D. (1966) Effects of progesterone on behavior. in: R. Levine (Ed), *Endocrines and the central nervous system*. Williams & Wilkins, Baltimore.

- HAMBURG D. (1971) Recent research on hormonal factors relevant to human aggressiveness. *Internat. Soc. Sci.* 3. 23, 1, 36-47.
- HAMBURG D. (1977) Developments in research on the psychobiology of aggression. in: J. Tanner (Ed), *Developments in psychiatric research*. Hodder & Stoughton, London.
- HAMBURG D. & M. TRUDEAU (Eds) (1981) *Biobehavioral aspects of aggression*. Alan R. Liss, NY.
- HAMBURG D.; R. MOOS & I. YALOM (1968) Studies of distress in the menstrual cycle and the postpartum period. in: R. Michael (Ed), *Endocrinology and human behaviour*. Oxford Univ. Press, London.
- HARRIS R. (1978) Relationship between EEG abnormality and aggressive and anti-social behaviour a critical appraisal. in: L. Hersov, M. Berger & P. Schaffer (Eds), *Aggression and anti-social behaviour in childhood and adolescence*. Pergamon, Oxford.
- HARTY J.; E. GIBBS & F. GIBBS (1942) Electroencephalographic study of 275 candidates for military service. *J. Nerv. Ment. Dis.* 96, 435-
- HAYMAKER W. et al. (1958) Occurrences of lesions in the temporal lobe in the absence of convulsive seizures. in: M. Baldwin & P. Bailey (Eds), *Temporal lobe epilepsy*. Thomas, Springfield.
- HELLER A. (1979) *On instincts*. Van Gorcum, Assen.
- HENRY C. (1963) Positive spike discharges in the EEG and behavior abnormality. in: Glaser (Ed).
- HILL D. (1944) Cerebral dysrhythmia: its significance in aggressive behavior. *Proc. Roy. Soc. Med.* 37, 317-30.
- HILL D. (1952) EEG in episodic psychotic and psychopathic behaviour: a classification of data. *EEG & Clin. Neurophysiol.* 4, 419-42.
- HILL D. (1953) Psychiatric disorders of epilepsy. *Med. Press* 20, 473.
- HILL D. & D. POND (1952) Reflections on one hundred capital cases submitted to electroencephalography. *J. Ment. Sci.* 98, 23-43.
- HILL D. & D. WATTERSON (1942) Electro-encephalographic studies of psychopathic personalities. *J. Neurol. Psychiat.* 5, 47-65.
- HIMMELHOCH J; J. PINCUS; G. TUCKER & T. DETRE (1970) Subacute encephalitis: behavioral and neurological aspects. *Brit. J. Psychiat.* 116, 531-8.

- HITCHCOCK E.; L. LAITINEN & K. VAERNET (Eds) (1972) *Psychosurgery*. Thomas, Springfield.
- HOLLOWAY R. (1968) Human aggression: the need for a species-specific framework. in: M. Fried, M. Harris & R. Murphy (Eds), *War; the anthropology of armed conflict and aggression*. National History Press, NY.
- HOLT R. (1970) On the interpersonal and intrapersonal consequences of expressing or not expressing anger. *J. Consult. Clin. Psychol.* 35, 1, 8-12.
- HORN K. (1972) Zur Sozialpsychologie von Kommunikation und Interaktion unter der Bedingung von Konflikt und Aggression. *Jahrbuch für Friedens- und Konfliktforschung*, Bd II.
- HORN K. (1972) Gibt es einen Aggressionstrieb? *Psyche* 26, 10, 799-817.
- HUGHES J. (1965) A review of the positive spike phenomenon. in: W. Wilson (Ed), *Applications of electroencephalography in psychiatry*. Duke Univ. Press, Durham, NC.
- HUGHES J.; E. MEANS & B. STELL (1965) A controlled study on the behavior disorders associated with the positive spike phenomenon. *EEG & Clin. Neurophysiol.* 18, 349-53.
- HUGHES J.; R. SCHLAGENHAUFF; M. CURTIN & V. BROWN (1961) Electroclinical correlation in temporal lobe epilepsy with emphasis on inter-areal analysis of the temporal lobe. *EEG & Clin. Neurophysiol.* 13, 333-
- INGRAM T. (1956) A characteristic form of over-active behavior in brain-damaged children. *J. Ment. Sci.* 102, 550-
- IVEY M. & J. BARDWICK (1968) Patterns of affective fluctuation in the menstrual cycle. *Psychosom. Med.* 30, 3, 336-45.
- JACKSON J. (1958) Temporary mental disorders after epileptic paroxysms. in: J. Taylor (Ed), *Selected writings of John Hughlings Jackson*. Basic Books, NY.
- JACOBS T. & E. CHARLES (1970) correlation of psychiatric symptomatology and the menstrual cycle in an outpatient population. *Amer. J. Psychiat.* 126, 148-52.
- JANOWSKY D.; GORNEY & A. MANDELL (1967) The menstrual cycle: psychiatric and ovarian-adrenocortical hormone correlates. Case study and literature review. *Arch. Gen. Psychiat.* 17, 459-69.
- JASPER J.; B. PERTUISET & H. FLANIGAN (1951) EEG and cortical electrograms in patients with temporal lobe seizures. *Arch. Neurol. Psychiat.* 65, 272-90.

- JENKINS R. & B. PACELLA (1943) Electroencephalographic studies of delinquent boys. *Amer. J. Orthopsychiat.* 13, 107-20.
- JONES E.; B. BAGCHI & R. WAGGONER (1955) Focal abnormalities of the electroencephalogram in juvenile behavior disorders. *J. Nerv. Ment. Dis.* 122, 28-35.
- JORDHEIM D. (1972) The premenstrual syndrome clinical trials of treatment with a progestogen combined with a diuretic compared with both a progestogen alone and with a placebo. *Acta Obstet. Gynecol. Scand.* 51, 77-80.
- KAHN I. (1927) Pollen toxemia in children. *JAMA*, 88, 241-2.
- KASANIN J. (1929) Personality changes in children following cerebral trauma. *J. Nerv. Ment. Dis.* 69, 385-406.
- KEATING L. (1961) Epilepsy and behavior disorder in schoolchildren. *J. Ment. Sci.* 107, 161-80.
- KEDENBURG H. (1977) Androgen and aggressive behavior in man. *Dissert. Abstracts* 38, 2-A, 885-6.
- KEDENBURG H. (1979) Testosterone and human aggressiveness Analysis. *J. Human Evol.* 8, 3, 407-10.
- KENNARD M. (1949) Significance of abnormal EEGs in disorders of behavior. *EEG & Clin. Neurophysiol.* 1, 118-9.
- KENNARD M.; A. SCHWARTZMAN & T. MILLER (1958) Sleep, consciousness, and the alpha electroencephalographic rhythm. *Amer. Med. Assoc. Arch. Neurol. & Psychiat.* 79, 328.
- KEPLER E. & F. MOERSCH (1937) The psychiatric manifestations of hypoglycemia. *Amer. J. Psychiat.* 14, 89-110.
- KIDD M. (1973) An EEG study of delinquent adolescents with reference to recidivism and murder. *Folia Psychiat. et Neurol.* 27, 77-84.
- KILLEFER F. & W. STERN (1970) Chronic effects of hypothalamic injury: report of a case of near total hypothalamic destruction resulting from removal of a craniopharyngioma. *Arch. Neurol.* 22, 419-29.
- KILOH L. & J. OSSELTON (1966) *Clinical Electroencephalography.* Butterworth, London.
- KIM S. (1976) The Lorenzian theory of aggression and peace research: a critique. *J. Peace Res.* 13, 4, 253-75.

- KLETSCCHKA H. (1966) Violent behavior associated with brain tumor. *Minnesota Med.* 49, 1853-5.
- KLIGMAN D. & D. GOLDBERG (1975) Temporal lobe epilepsy and aggression: problems in clinical research. *J. Nerv. Ment. Dis.* 160, 5, 324-41.
- KNOTT J. & J. GOTTLIEB (1943) Electroencephalogram in psychopathic personality. *Psychosom. Med.* 5, 139-42.
- KRAMER F. & H. POLLNOW (1932) über eine hyperkinetische Erkrankung im Kindesalter. *Monatschrift Psychiat. Neurol.* 82, 1-
- KRESCNER M.; M BENDER & I. STRAUS (1936) Mental symptoms in cases of tumor of the temporal lobe. *Arch. Neurol. Psychiat.* 35, 572-96.
- KREUZ L. & R. ROSE (1972) Assessment of aggressive behavior and plasma testosterone in a young criminal population. *Psychosom. Med.* 34, 39 321-32.
- KRYNICKI V. (1978) Cerebral dysfunction in repetitively assaultive adolescents. *J. Nerv. Ment. Dis.* 166, 59-67.
- LAITINEN L. & K. LIVINGSTON (Eds) (1973) *Surgical approaches in psychiatry.* Medical & Technical Publ. Co., Lancaster.
- LANGHORNE J. & J. LONEY (1979) A four-fold model for subgrouping the hyperkinetic/MBD syndrome. *Child Psychiat. & Human Developm.* 9, 3, 153-9.
- LESCH M. & W. NYHAN (1964) A familial disorder of uric acid metabolism and central nervous system function. *Amer. J. Med.* 36, 561-70.
- LEVY S. (1959) Postencephalitic behavior disorder a forgotten entity. *Amer. J. Psychiat.* 115, 1062.
- LEVY S. (1966) The hyperkinetic child: its diagnosis and treatment. *Internat. J. Neuropsychiat.* 2, 330-36.
- LEVY S. & M. KENNARD (1953) Study of electroencephalogram as related to personality structure in group of inmates of state penitentiary. *Amer. J. Psychiat.* 109, 832-9.
- LEWIS D. (1976) Delinquency, psychomotor epileptic symptoms, and paranoid ideation: a triad. *Amer. J. Psychiat.* 133, 12, 1395-8.
- LEWIS D. & S. SHANOK (1979) Medical histories of psychiatrically referred delinquent children; an epidemiological study. *Amer. J. Psychiat.* 136, 2, 231-3.

- LEWIS D. & S. SHANOK (1979) A comparison of the medical histories of incarcerated delinquent children and a matched sample of non-delinquent children. *Child Psychiat. & Human Developm.* 9, 4, 210-14.
- LEWIS D.; S. SHANOK & D. BALLA (1979) Perinatal difficulties, head and face trauma, and child abuse in the medical histories of seriously delinquent children. *Amer. J. Psychiat.* 136, 4-A, 419-23.
- LEWIS D.; S. SHANOK & D. BALLA (1979) Parental criminality and medical histories of delinquent children. *Amer. J. Psychiat.* 136, 3, 268-92.
- LEWIS D. et al. (1962) Psychomotor epilepsy and violence in a group of incarcerated adolescent boys. *Amer. J. Psychiat.* 139, 7, 882-7.
- LEWIS D. et al. (1983) Homicidally aggressive young children: neuropsychiatric and experiential correlates. *Amer. J. Psychiat.* 140, 2, 148-53.
- LEWIS J. (1975) Violence and epilepsy. *JAMA* 232, 11, 1165-7.
- LIBERT M. (1980) A propos des aspects psychiatriques d'un cas de maladie de Unverricht-Lundborg. *Annales Médico-Psychologiques* 138, 2, 197-201.
- LIDDELL D. (1953) Observations on epileptic automatism in a mental hospital population. *J. Ment. Sci.* 99, 732-48.
- LION J. (1972) *Evaluation and management of the violent patient.* Thomas, Springfield.
- LION J. (Ed) (1974) *Personality disorders: diagnosis and management.* Williams & Wilkins, Baltimore.
- LION J. (1975) The development of a violence clinic. in: Pasternak (Ed).
- LION J. & M. PENNA (1974) The study of human aggression. in: Whalen (Ed).
- LION J. & M. PENNA (1976) Some scientific, clinical. and ethical issues in the treatment of aggressive patients. in: Madden & Lion (Eds).
- LION J.; G. BACH-Y-RITA & F. ERVIN (1968) The self-referred violent patient. *JAMA* 205, 503-5.
- LION J.; G. BACH-Y-RITA & F. ERVIN (1969) Violent patients in the emergency room. *Amer. J. Psychiat.* 125, 12, 1706-11.
- LION J.; D. MADDEN & R. CHRISTOPHER (1976) A violence clinic: three years'

- experience. *Amer. J. Psychiat.* 133, 4, 432-5.
- LION J. et al. (1974) A violence clinic. *Maryland State Med. J.* 23, 45-8.
- LISHMAN W. (1966) Psychiatric disability after head injury: the significance of brain damage. *Proc. Roy. Soc. Med.* 59, 261.
- LIVINGSTON S. (1964) Epilepsy and murder. *JAMA* 188, 2, 172-81.
- LOMBROSO C. et al. (1966) Ctenoids in healthy youths: controlled study of 14- and 6-per second positive spiking. *Neurol.* 16, 1152-8.
- LONEY J. (1980) Hyperkinesia comes of age: what do we know and where should we go? *Amer. J. Orthopsychiat.* 50, 1, 28-42.
- LONEY J.; J. LANGHORNE & C. PATERNITE (1978) An empirical basis for subgrouping the hyperkinetic/miminal brain dysfunction syndrome. *J. Abnorm. Psychol.* 87, 4, 431-41.
- LOOMIS S. (1965) EEG abnormalities as a correlate of behavior in adolescent male delinquents. *Amer. J. Psychiat.* 121, 1003-6.
- LYGHT C.(Ed) (1966) *The Merck manual of diagnosis and therapy.* Merck, West Point, Pa.
- MacDONALD J. (1961) *The murderer and his victim.* Thomas, Springfield.
- MADDEN D. & J. LION (Eds) (1976) *Rage-Hate-Assault and other forms of violence.* Spectrum, NY.
- MALAMUD N. (1967) Psychiatric disorder with intracranial tumors of limbic system. *Arch. Neurol.* 17, 113-23.
- MALETZKY B. (1973) The episodic dyscontrol syndrome. *Dis. Nerv. Syst.* 34, 178-85.
- MALETZKY B. (1976) The diagnosis of pathological intoxication. *J. Studies on Alcohol* 37, 9, 1215-28.
- MALETZKY B. & J. KLOTTER (1974) Episodic dyscontrol: a controlled replication. *Dis. Nerv. Syst.* 35, 4, 175-9.
- MANDELL A. & M. MANDELL (1967) Suicide and the menstrual cycle. *JAMA* 200, 792.
- MANDELL M. (1969) Cerebral reactions in allergic patients. Case histories and provocative test results. Paper 25th Conf. Amer. Coll. Allergists, Washington DC.
- MARGERISON J. & J. CORSELLIS (1966) Epilepsy and the temporal lobes. *Brain* 89,

499-530.

MARINACCI A. (1956) Electroencephalography in alcoholism. in: G. Thompson (Ed), Alcoholism. Thomas, Springfield.

MARINACCI A. (1963) A special type of temporal lobe psychomotor seizures following ingestion of alcohol. Bull. Los Angeles Neurol. Soc. 28, 241-50.

MARK V. (1978) Sociobiological theories of abnormal aggression. in: I. Kutash et al. (Eds) Violence; perspectives on murder and aggression. Jossey-Bass, San Francisco.

MARK V. & F. ERVIN (1970) Violence and the brain. Harper & Row, NY.

MARK V. & W. SWEET (1974) The role of limbic brain dysfunction in aggression. in: S. Frazier (Ed), Aggression. Williams and Wilkins, Baltimore.

MARK V. et al. (1967) Brain disease and violent behavior. Paper Soc. Advancement Behav. Therapy, Washington DC.

MARLOWE M. & J. ERRERA (1982) Low lead levels and behavior problems in children. Behav. Disorders 7, 3, 153-72.

MARTINIUS J. & P. STRUNK (1979) Homicide of an aggressive adolescent boy with right temporal lesion: case study. Z. f. Kinder- und Jugendpsychiatrie 7, 3, 199-207.

MAUDSLEY H. (1874) Responsibility in mental disease. Appleton, NY.

McCLELLAND D. et al. (1953) The achievement motive. Appleton, NY.

McLAURIN R & F. HEIMER (1965) The syndrome of temporal lobe contusion. J. Neurosurg. 23, 296-304.

MECIR J. (1976) Homicidal behavior of persons older than nineteen years. Ceskoslovenska Psychiatrie 72, 6, 374-83.

MECIR J. & SIROTKOVA K. (1980) Catamnestic observation of children with behavior disorders of impulsive and aggressive nature. Ceskoslovenska Psychiat. 76, 5, 289-98.

MEGARGEE E. (1966) Undercontrolled and overcontrolled personality types in extreme antisocial aggression. Psychol. Monogr. 80, 611, whole issue.

MEGARGEE E. (1970) The prediction of violence with psychological tests. in: C. Spielberger (Ed) Current topics in clinical and community Psychology, Vol. I, Academic Press, NY.

- MEGARGEE E. (1972) *The psychology of violence and aggression*. General Learning Press, Morristown, NJ.
- MELLO N. & J. MENDELSON (1975) Alcoholism: a biobehavioral disorder. in: *American Handbook of Psychiatry*. Vol. 4, Basic Books, NY.
- MENDELSON J. & N. MELLO (1974) Alcohol, aggression and androgens. in: S. Frazier (Ed), *Aggression*, Williams & Wilkins, Baltimore.
- MENNINGER K. (1963) *The vital balance*. Viking Press, NY.
- MEYER-BAHLBURG H. (1981) Androgens and human aggression. in: P. Brain & D. Benton (Eds), *The biology of aggression*. Sythoff & Noordhoff, Alphen a.d. Rijn.
- MEYER-BAHLBURG H. et al. (1974) Aggressiveness and testosterone measures in man. *Psychosom. Med.* 36, 3, 269-74.
- MILICH R; J. LONEY & S. LANDAU (1982) Independent dimensions of hyperactivity and aggression: a validation with playroom observation data. *J. Abnorm. Psychol.* 91, 3, 183-98.
- MILSTEIN V. & J. SMALL (1971) Psychological correlates of 14 and 6 positive spikes, 6/sec. spike wave, and small sharp spike transients. *Clin. EEG* 2, 206-12.
- MINKOWSKA F. (1937) Heredity of epilepsy and schizophrenia. *Arch. Klaus Stift. Vererbungsforschung* 12, 33-
- MINSKI L. & E. GUTTMANN (1938) Huntington's chorea: a study of thirty-four families. *J. Ment. Sci.* 84, 21-
- MIRSKY A. & N. HARMAN (1974) On aggressive behavior and brain disease some questions and possible relationships derived from the study of men and monkeys. in: Whalen (Ed).
- ITSUDA H. (Ed) (1967) *Clinical genetics in psychiatry: problems in nosological classification*. Igaku Shoin, Tokyo.
- MNUKHIN S. & E. DINABURG (1965) Epileptiform manifestations in early right sided and left sided lesions of the brain in children. *Zh. Nevropat. Psikiat. Korsakov* 65, 1073.
- MONROE R. (1970) *Episodic behavioral disorders: a psychodynamic and neurophysiological analysis*. Harvard Univ. Press, Cambridge, MA.
- MONROE R. (1974) Maturational lag in central nervous system development as a partial explanation of episodic violent behavior. in: K. de Wit & W. Hartup (Eds), *Determinants and origins of aggressive behavior*. Mouton, The Hague.

- MONROE R. (1975) Drugs in the management of episodic behavioral disorders. in: Fields & Sweet (Eds).
- MONROE R. (1978) Brain dysfunction in aggressive criminals. Heath, Lexington, MA.
- MOOS R. (1968) The development of a menstrual distress questionnaire. *Psychosom. Med.* 30, 853-67.
- MOOS R. et al. (1969) Fluctuations in symptoms and moods during the menstrual cycle. *J. Psychosom. Res.* 13, 37-44.
- MORENO B. (1981) Etologia, psicofisiologia y violencia. *Revista Colombiana de Psiquiatria* 10, 1, 8-24.
- MORTON J. (1950) Premenstrual tension. *Amer. J. Obstet. & Gynecol.* 60, 343-52.
- MORTON J. et al. (1953) A clinical study of premenstrual tension. *Amer. J. Obstet. & Gynecol.* 65, 1182-91.
- MOYER K. (1976) The psychobiology of aggression. Harper & Row, NY.
- MULDER D. & D. DALY (1952) Psychiatric symptoms associated with lesions of temporal lobe. *JAMA* 150, 173-6.
- MULLAN J. (1971) Violent behavior: a case report presented at the central neurosurgical society. Chicago.
- MURDOCH B. (1972) Electroencephalograms, aggression and emotional maturity in psychopathic and non-psychopathic prisoners. *Psychologia Africana* 14, 216-31.
- MUSAPH H. (1959) Aggression and symptom formation in dermatology. *J. Psychosom. Res.* 13, 257-64.
- MUSAPH H. & P. METTROP (1972) The role of aggression in human pathology. Karger, Basel.
- NAGARAYA J. (1976) The hyperactive child. *Child Psychiat. Quart.* 9, 2, 1-4.
- NASH D. (1975) Genetic aspects of contemporary violence. in: C. Wilber (Ed), *Contemporary violence*. Thomas, Springfield.
- NUFFIELD E. (1961) Neurophysiology and behaviour disorders in epileptic children. *J. Ment. Sci.* 107, 438-58.

- NUFFIELD E. (1961) Electro-clinical correlations in childhood epilepsy. *Epilepsia* 2, 178-96.
- NYHAN W. (1968) Summary of clinical features. in: *Seminars on the Lesch-Nyhan syndrome*. Fed. Proc. 27, 1034-41.
- NYHAN W. (1970) Human purine metabolism and behavior. *Engineer. & Sci.* 33, 45-9.
- NYHAN W. (1976) Behavior in the Lesch-Nyhan syndrome. *J. Autism & Childhood Schizophrenia* 6, 3, 235-52.
- NYHAN W. et al. (1969) A new disorder of purine metabolism with behavioral manifestations. *Pediatrics* 74, 20-27.
- d'ORBAN P. & J. DALTON (1980) Violent crime and the menstrual cycle. *Psychol. Med.* 10, 2, 353-9.
- OUNSTED C. (1955) Hyperkinetic syndrome in epileptic children. *Lancet* 2, 303-11.
- OUNSTED C. (1969) Aggression and epilepsy: rage in children with temporal lobe epilepsy. *J. Psychosom. Res.* 13, 237-42.
- OUNSTED C.; J. LINDSAY & R. NORMAN (1966) *Biological factors in temporal lobe epilepsy*. Heineman, London.
- OWENS D.; J. DAWSON & S. LOSIN (1971) Alzheimer's disease in Down's syndrome. *Amer. J. Ment. Defic.* 75, 606-12.
- PAILLAS J. (1958) Aspects cliniques de l'épilepsie temporale. in: M. Baldwin & P. Bailey (Eds) *Temporal lobe epilepsy*. Thomas, Springfield.
- PAPEZ J. (1937) A proposed mechanism of emotion. *Arch. Neurol. Psychiat.* 38, 725-43.
- PARLEE M. (1973) The premenstrual syndrome. *Psychol. Bull.* 60, 6, 454-65.
- PASTERNAK S. (1974) The explosive, antisocial, and passive-aggressive personalities. in: Lion (Ed).
- PASTERNAK S. (1975) (Ed) *Violence and victims*. Spectrum, NY.
- PENFIELD W. & H. JASPER (1954) *Epilepsy and the functional anatomy of the human brain*. Little, Brown, Boston.
- PENNINGTON V. (1957) Meprobamate (Miltown) in premenstrual tension. *JAMA* 164, 638-40.
- PERSKY H.; M. ZUCKERMAN & G. CURTIS (1968) Endocrine function in emotionally

disturbed and normal men. *J. Nerv. Ment. Dis.* 146, 488-97.

PERSKY H.; K. SMITH & G. BASU (1970) Relationship of hostility and testosterone production rate in normal young men. *Psychosom. Med.* 32, 5, 553-

PERSKY H.; K. SMITH & G. BASU (1971) Relation of psychologic measures of aggression and hostility to testosterone production in man. *Psychosom. Med.* 33, 265-77.

PINCUS J. & G. GLASER (1966) The syndrome of 'minimal brain damage' in childhood. *New England J. Med.* 27, 275-

PINCUS J. & G. TUCKER (1974) *Behavioral neurology.* Oxford Univ. Press, NY.

PIONKOWSKI J.; I. WOJDYSLAWSKA & E. PATURA (1975) Pathological intoxication with alcohol. *Psychiatria Polska* 9, 2, 161-6.

PODOLSKY E. (1955) The diseased brain and homicide. *J. Nerv. Ment. Dis.* 124, 621-4.

PODOLSKY E. (1962) The epileptic murderer. *Medico-Legal J.* 30, 176-9.

PODOLSKY E. (1964) The chemistry of murder. *Pakistan Med.* 3. 15, 9-14.

PODOLSKY E. (1965) Organic brain conditions and homicide. *Corrective Psychiat. & J. Soc. Therapy* 11, 91-4.

POND D. (1961) Psychiatric aspects of epileptic and brain damaged children. *Brit. Med. J.* 2, 1376-87; 1454-9.

POND D. (1963) The development of normal rhythms. In: D. Hill & G. Parr (Eds), *Electroencephalography.* Macmillan, NY.

POUNDERS C. (1948) The allergic child. *Southern Med. J.* 41, 142-6.

PRECHTL H. (1960) *The long term value of the neurological examination of the newborn infant.* Heinemann, London.

PRECHTL H. & C. STEMMER (1962) The choreiform syndrome in children. *Developm. Med. Child Neurol.* 4, 119-

PRINZ R.; P. CONNOR & C. WILSON (1981) Hyperactive and aggressive behaviors in childhood: intertwined dimensions. *J. Abnorm. Child Psychol.* 9, 2, 191-202.

RADA R.; D. LAWS & R. KELLNER (1976) Plasma testosterone levels in the rapist. *Psychosom. Med.* 38, 4, 257-68.

RADA R.; R. KELLNER & W. WINSLOW (1976) Plasma testosterone and aggressive

behavior. *Psychosom.* 17, 3, 138-42.

RANDOLPH T. (1947) Allergy as a causative factor of fatigue, irritability, and behavior problems of children. *J. Pediatrics* 31, 560-

RANDOLPH T. (1959) Ecologic mental illness psychiatry exteriorized. *J. Lab. & Clin. Med.* 54, 936.

RANDOLPH T (1962) Human ecology and susceptibility to the chemical environment. Thomas, Springfield.

RASA A. (1980) Ethological aspects of aggressive behavior. Paper Conf. Biology of Aggression, Bonas.

RASA A. (1982) Towards a structural concept of agonism. *Aggr. Behav.* 6, 3, 253-60.

REEVES A. & F. PLUM (1969) Hyperphagia, rage and dementia accompanying a ventromedial hypothalamic neoplasm. *Arch. Neurol. Chic.* 20, 616-24.

RENSBURG M. van; et al. (1975) Temporal lobe epilepsy due to an intracerebral Schwannoma: case report. *J. Neurol. Neurosurg. & Psychiat.* 38, 703-9.

REVITCH E. & L. SCHLESINGER (1976) Murder: evaluation, classification, and prediction. in: I. Kutash et al. (Eds) *Violence; perspectives on murder and aggression.* Jossey-Bass, San Francisco.

RIBEIRO A. (1962) Menstruation and crime. *Brit. Med. J. I,* 640-1.

ROBERTSON A. (1675) On Graves' disease with insanity. *J. Ment. Sci.* 20, 573-

ROBINS L. (1966) *Deviant children grown up.* Williams & Wilkins, Baltimore.

ROCHLIN G. (1973) *Man's aggression: the defense of the self.* Gambit, Boston.

RODIN E. (1973) Psychomotor epilepsy and aggression. *Epilepsia* 14, 91-4.

RODIN E. (1973) Psychomotor epilepsy and aggressive behavior. *Arch. Gen. Psychiat.* 28, 210-13.

RODIN E.; M. KATZ & K. LENNOX (1976) Differences between patients with temporal lobe seizures and those with other forms of epileptic attacks. *Epilepsia* 17, 3, 313-20.

ROGER A. & M. DONGIER (1950) *Corrélations électrocliniques chez épileptiques internés.* *Rev. Neurol.* 83, 593-6.

ROGINA V. & E. SERAFETINIDES (1962) Epilepsy and behavior disorder in patients with generalized spike and wave complexes. EEG & Clin. Neurophysiol. 14, 3.

ROME H. & D. ROBINSON (1959) Psychiatric conditions associated with metabolic, endocrine, and nutritional disorders. in: American Handbook of Psychiatry. Vol. 2. Basic Books, NY.

ROTH M. (1968) Cerebral disease and mental disorders of old age as causes of antisocial behavior. in: A. de Rueck & R. Porter (Eds), The mentally abnormal offender. Little, Brown, Boston.

ROTH M. & M. HARPER (1962) Temporal lobe epilepsy and the phobic-anxiety syndrome. Compr. Psychiat. 3, 215-26.

ROTHMAN L. et al. (1976) Intracranial ectopic pituitary adenoma: case report. J. Neurosurg. 44, 96-9.

ROWE A. (1930) Allergic toxemia and migraine due to food allergy. California & Western Med. 33, 785-93.

ROWE A. (1944) Clinical allergy in the nervous system. J. Nerv. Ment. Dis. 99, 834-41.

RUD E. (1937) Spontaneous hypoglycemia with peculiar psychic disturbance. Acta Med. Scand. 91, 648-55.

RUDD B.; D. GALAL & M. CASEY (1968) Testosterone excretion rates in normal males and males with an XYY complement. J. Med. Genetics 5, 286-8.

RUMMEL R. (1977) Understanding conflict and war. Vol. 3. Sage, Beverly Hills.

SACHAR E. (1975) Psychiatric disturbances associated with endocrine disorders. in: American Handbook of Psychiatry. Vol 4, Basic Books, NY.

SACHAR E. (Ed) (1976) Hormones, behavior and psychopathology. Raven Press, NY.

SANDLER M. (Ed) (1979) Psychopharmacology of aggression. Raven Press, NY.

SANDLER M. et al. (1978) Phenylethylamine overproduction in aggressive psychopaths. Lancet 8103, 1269-70.

SANDLER M. et al. (1979) Phenylethylamine in human aggressive behavior. in: Sandler (Ed).

SANDS D. (1954) Further studies on endocrine treatment in adolescence and early adult life. J. Ment. Sci. 100, 211-19.

- SANO K. (1962) Sedative neurosurgery: with special reference to postero-medial hypothalamotomy. *Neurol. Medicochir.* 4, 112-42.
- SARTESCHI P.; E. LONGO & S. BAGLIVO (1978) Pathological aggressiveness in man: some theoretical and practical considerations. in: Valzelli (Ed).
- SAYED Z.; S. LEWIS & R. BRITAIN (1969) An electroencephalographic and psychiatric study of 32 insane murderers. *EEG Clin. Neurophysiol.* 27, 335-
- SCARAMELLA T. & W. BROWN (1978) Serum testosterone and aggressiveness in hockey players. *Psychosom. Med.* 40, 3, 262-5.
- SCHAFFER N. (1953) Personality changes induced in children by the use of certain antihistaminic drugs. *Annals of Allergy*, 11, 317-8.
- SCHNEIDER W. (1945) Psychiatric evaluation of the hyperkinetic child. *J. Pediatrics* 26, 559-70.
- SCHULZ H. (1982) Das Ausfallssyndrom nach Unterbrechung der Alkoholabhängigkeit. *Fortschritte der Neurol. Psychiat.* 50, 9, 279-96.
- SCHWAB R. et al. (1965) Treatment of intractable temporal lobe epilepsy by stereotactic amygdala lesions. *Trans. Amer. Neurol. Assoc.* 90, 12-19.
- SCHWADE E. & S. GEIGER (1953) Matricide with electroencephalographic evidence of thalamic and hypothalamic disorder. *Dis. Nerv. Syst.* 14, 18-20.
- SCHWADE E. & S. GEIGER (1956) Abnormal EEG findings in severe behavior disorder. *Dis. Nerv. Syst.* 17, 307-17.
- SCHWADE E. & S. GEIGER (1960) Severe behavior disorders with abnormal electroencephalograms. *Dis. Nerv. Syst.* 21, 616-20.
- SCHWADE E. & O. OTTO (1953) Homicide as a manifestation of thalamic or hypothalamic disorder with abnormal electroencephalographic findings. *Wisconsin Med. J.* 52, 171-4.
- SCOTT J. (1975) Violence and the disaggregated society. *Aggr. Behav.* 1, 3, 235-60.
- SERAFETINIDES E. (1965) Aggressiveness in temporal lobe epilepsies and its relation to cerebral dysfunction and environmental factors. *Epilepsia* 6, 33-42.
- SERAFETINIDES E. (1970) Psychiatric aspects of temporal lobe epilepsy. in: E. Niedmayer (Ed), *Epilepsy, modern problems in pharmacopsychiatry*. Karger, NY.
- SHAINNESS N. (1961) A reevaluation of some aspects of femininity through a study of

menstruation: a preliminary report. *Compr. Psychiat.* 2, 20-26.

SHERWIN I. (1976) Temporal lobe epilepsy: neurological and behavioral aspects. *Ann. Rev. Med.* 27, 37-47.

SHRABERG D. & L. WEISBERG (1978) The Klüver-Bucy syndrome in man. *J. Nerv. Ment. Dis.* 166, 2, 130-34.

SILVERMANN D. (1943) Clinical and electroencephalographic studies on criminal psychopaths. *Arch. Neurol. Psychiat.* 50, 18-33.

SILVERMANN D. (1944) The electroencephalogram of criminals. *Arch. Neurol. Psychiat.* 52, 38-42.

SILVERMANN D. (1949) Implication of the EEG abnormalities in the psychopathic personality. *Arch. Neurol. Psychiat.* 62, 870-3.

SIOMOPOULOS V. (1978) Psychiatric diagnosis and criminality. *Psychol. Rep.* 42, 2, 559-52.

SKELTON W. (1970) Alcohol, violent behavior, and the electroencephalogram. *Southern Med. J.* 63, 425-66.

SLATER E. & M. ROTH (1969) *Clinical psychiatry*. 3rd ed. Bailliere, Tindall & Cassell, London.

SMALL J. (1966) The organic dimensions of crime. *Arch. Gen. Psychiat.* 15, 82-9.

SMALL J. (1968) Epileptiform electroencephalographic abnormalities in mentally ill children. *J. Nerv. Ment. Dis.* 147, 341-8.

SMALL J. & I. SMALL (1964) 14 & 6 second positive spikes. *Arch. Gen. Psychiat.* 11, 645-50.

SMALL J.; V. MILSTEIN & J. STEVENS (1952) Are psychomotor epileptics different? *Amer. Med. Assoc. Arch. Neurol.* 7, 187-94.

SMALL J.; I. SMALL & N. HAYDEN (1966) Further psychiatric investigations of patients with temporal and non-temporal lobe epilepsy. *Amer. J. Psychiat.* 123, 303-10.

SOLOMON G. (1970) Psychodynamic aspects of aggression, hostility and violence. in: D. Daniels et al. (Eds), *Violence and the struggle for existence*. Little, Brown, Boston.

SOUSA A. de & D. de SOUSA (1977) Hyperkinesis. *Child Psychiat. Quart.* 10,4, 8-13.

- SPEER F. (1954) The allergic tension-fatigue syndrome. *Pediatric Clinic of North America* 1, 1029-37.
- SPEER F. (1958) The allergic tension-fatigue syndrome in children. *Internat. Arch. Allergy* 12, 207-14.
- SPEER F. (Ed) (1970) *Allergy of the nervous system*. Thomas, Springfield.
- SPELLACY F. (1977) Neuropsychological differences between violent and nonviolent adolescents. *J. Clin. Psychol.* 33, 4, 966-9.
- SPELLACY F. (1978) Neuropsychological discrimination between violent and nonviolent men. *J. Clin. Psychol.* 34, 1, 49-53.
- STAFFORD-CLARK D. & F. TAYLOR (1949) Clinical and electro-encephalographic studies of prisoners charged with murder. *J. Neurol. Neurosurg. Psychiat.* 12, 325-30.
- STEHLE H. (1960) Thalamic dysfunction involved in destructive-aggressive behavior directed against persons and property. *EEG Clin. Neurophysiol.* 12, 264-5.
- STEINER M. & B. CARROLL (1977) The psychobiology of premenstrual dysphoria: review of theories and treatments. *Psychoneuroendocrinol.* 2, 4, 321-35.
- STEINHAUSEN H. (1978) Psychopathology of juvenile neuronal ceroid-lipofuscinosis (Batten-Spielmeier-Vogt disease). *Z. f. Kinder- u. Jugendpsychiatrie* 6, 3, 255-65.
- STEVENS J. (1966) Psychiatric implications of psychomotor epilepsy. *Arch. Gen. Psychiat.* 14, 461-71.
- STEVENS J. (1972) Round-table discussion: violent behavior and the electroencephalogram. *Clin. Electroencephalography* 3, 180-214.
- STEVENS J. (1975) Interictal clinical manifestations of complex partial seizures. *Adv. Neurol.* II, 85-112.
- STEVENS J. & V. MILSTEIN (1970) Severe psychiatric disorders of childhood, electroencephalogram and clinical correlations. *Amer. J. Dis. Child.* 120, 182-92.
- STEVENS, J.; K. SACHDEV & V. MILSTEIN (1968) Behavior disorders of children and the electroencephalogram. *Arch. Neurol.* 18, 160-77.
- STEWART M. (1970) Hyperactive children. *Sci. Amer.* 222, 94-8.
- STEWART M. & S. STEWART (1981) Serum cholesterol in antisocial personality: a failure to replicate earlier findings. *Neuropsychobiol.* 7, 1, 9-11.

- STEWART M. et al. (1966) The hyperactive child syndrome. *Amer. J. Orthopsychiat.* 36, 861-7.
- STEWART M. et al. (1981) The overlap between hyperactive and unsocialized aggressive children. *J. Child Psychol. Psychiat. & Allied Disc.* 22, 1, 35-45.
- STRAUSS H. (1959) Epileptic disorders. in; *American Handbook of Psychiatry.* Vol. 2, Basic books, NY.
- STRAUSS H. et al. (1940) Studies on a group of children with psychiatric disorders I: electroencephalographic studies. *Psychosom. Med.* 2, 34-
- STRAUSS H. et al. (1952) *Diagnostic electroencephalography.* Grune & Stratton, NY.
- STRAUSS I. & M. KESCHNER (1935) Mental symptoms in cases of tumor of the frontal lobe. *Arch. Neurol. Psychiat.* 33, 986-1005.
- STRECKER E. & F. EBAUGH (1924) Neuropsychiatric sequelae of cerebral trauma in children. *Arch. Neurol. Psychiat.* 12, 443-53.
- STRUVE F. et al. (1972) Electroencephalographic correlates of suicide ideation and attempts. *Arch. Gen. Psychiat.* 27, 363-5.
- SURWILLO W. (1980) The electroencephalogram and childhood aggression. *Aggr. Behav.* 6, 1, 9-18.
- SUTHERLAND H. & I. STEWART (1965) A critical analysis of the premenstrual syndrome. *Lancet* 1, 1180-93.
- SUTOROVA D. & M. TRAVNIKOVA (1976) The effect of concussion on some personality characteristics and behavior of the child. *Psychologia a Patopsychologia Dietata* 11, 2, 143-50.
- SWEET W.; F. ERVIN & V. MARK (1969) The relationship of violent behavior to focal cerebral disease. in: S. Garattini & E. Sigg (Eds), *Aggressive behavior.* Wiley, NY.
- SWEET W. et al. (Eds) (1977) *Neurosurgical treatment in psychiatry, pain and epilepsy.* Univ. Park Press, Baltimore.
- SZEWZYK H. (1974) Manslaughter committed by juveniles with cerebral lesions contracted in early childhood. *Psychiat. Neurol. u. Mediz. Psychol.* 26, 7, 385-94.
- SZONDI, L. (1952) *Triebpathologie.* Huber, Bern.

- TATERKA J. & J. KATZ (1955) Study of correlations between electroencephalographic and psychological patterns in emotionally disturbed children. *Psychosom. Med.* 17, 62-
- TAYLOR D. (1969) Aggression and epilepsy. 3. *Psychosom. Res.* 13, 3, 229-36.
- TAYLOR J. (1979) Plasma progesterone, oestradiol 17 bêta and premenstrual symptoms. *Acta Psychiat. Scand.* 60, I, 76-86.
- TEDESCHI J.; R. SMITH & R. BROWN (1974) A reinterpretation of research on aggression. *Psychol. Bull.* 81, 9, 540-62.
- TEDESCHI J.; G. GAES & A. RIVERA (1977) Aggression and the use of coercive power. *J. Soc. Issues* 33, 1, 101-25.
- THIESSEN D. (1976) *The evolution and chemistry of aggression.* Thomas, Springfield.
- THOMPSON G. (1959) Acute and chronic alcoholic conditions. in: *American Handbook of Psychiatry.* Vol 2, Basic Books, NY.
- THOMPSON G. (1963) Electroencephalogram in acute pathological alcoholic intoxication. *Bull. Los Angeles Neurol. Soc.* 28, 217-
- TOCH H. (1969) *Violent men; an inquiry into the psychology of violence.* Aldine, Chicago.
- TÖRNE J. von (1974) On the etiology of self-mutilation in childhood and adolescence. *Z. f. Kinder- u. Jugendpsychiat.* 2, 3, 261-78.
- TORGHELE P. (1957) Premenstrual tension in psychotic women. *Lancet* 77, 163-70.
- TREFFERT D. (1964) The psychiatric patient with an EEG temporal lobe focus. *Amer. J. Psychiat.* 120, 765-71.
- TUERK K.; I. FISH & J. RANSOHOFF (1975) Head injury. in: *American Handbook of Psychiatry.* Vol. 4, Basic Books, NY.
- VALZELLI L. (1971) *Profili di psicofisiologia e neurochimica.* Manfredi, Milano.
- VALZELLI L. (Ed) (1978) *Psychopharmacology of aggression.* Karger, Basel.
- VALZELLI L. (1981) *Psychobiology of aggression and violence.* Raven Press, NY.
- VERDEAUX G. & J. VERDEAUX (1955) Etude électroencéphalographique d'un group important de délinquants primaires ou récidivistes au cours de leur détention. *Annales Médico-psychologiques* 113, 143-58.

VIRKKUNEN M. (1979) Serum cholesterol in antisocial personality. *Neuropsychobiol.* 5, 1, 27-30.

VIRKKUNEN M. (1982) Reactive hypoglycemic tendency among habitually violent offenders: a further study by means of the glucose tolerance test. *Neuropsychobiol.* 8, 1, 35-40.

VIRKKUNEN M. & M. HUTTUNEN (1982) Evidence for abnormal glucose tolerance test among violent offenders. *Neuropsychobiol.* 8, 1, 30-34.

VIRKKUNEN M.; A. NUUTILA & S HUUSKO (1976) Effect of brain injury on social adaptability: longitudinal study of frequency of criminality. *Acta Psychiat. Scand.* 53, 168-72.

VONDERAHE A. (1940) Forebrain and rage reactions. *Trans. Amer. Neurol. Assoc.* 66, 129-30.

VONDERAHE A. (1944) The anatomic substratum of emotion. *New Scholasticism* 18, 76-95.

WALKER A. (1961) Murder or epilepsy? *J. Nerv. Ment. Dis.* 133 430-7.

WALKER A & D. BLUMER (1970) Temporal lobe epilepsy and violent behavior. Lecture Johns Hopkins Hospital, Baltimore.

WALKER A. & D. BLUMER (1975) Long term effects of temporal lobe lesions on sexual behavior and aggressivity. in: Fields & Sweet (Eds).

WALLER J. & E. WHORTON (1973) Unintentional shootings, highway crashes and acts of violence: a behavior paradigm. *Accident Analysis & Prevention*, 5, 4, 351-6.

WEIL A. (1959) Ictal emotions occurring in temporal lobe dysfunction. *Arch. Neurol.* 1, 101-11.

WENDER P. (1971) *Minimal brain dysfunction in children.* Wiley, NY.

WHALEN R. (Ed) (1974) *The neuropsychology of aggression.* Plenum Press, NY.

WILDER J. (1947) Sugar metabolism in its relation to criminology. in: S. Lindner & B. Seliger (Eds), *Handbook of correctional psychology.* Philosoph. Library, NY.

WILLIAMS D. (1941) The significance of an abnormal electroencephalogram. *J. Neuropsychiat.* 4, 257.

WILLIAMS D. (1954) The electroencephalogram in affective disorders. *Proc. Roy. Soc. Med.* 47, 779.

WILLIAMS D. (1956) The structure of emotions reflected in epileptic experience. *Brain* 79, 29-67.

WILLIAMS D. (1969) Neural factors related to habitual aggression: considerations of differences between those habitual aggressives and others who have committed crimes of violence. *Brain* 92, pt 3, 503-20.

WILLIAMS D. (1969) Temporal lobe syndrome. in: P. Vinken & G. Bruyn (Eds), *Handbook of Clinical Neurology*. Vol. 29, Wiley, NY.

WILLIAMS D. (1975) Studies of persons confined for crimes of violence. in: Fields & Sweet (Eds).

WILSON E. (1975) *Sociobiology; the new synthesis*. Harvard Univ. Press, Cambridge.

WILSON E. (1978) *On human nature*. Harvard Univ. Press, Cambridge.

WILSON S. (1940) *Kinnier, Neurology*. Vol. I. Williams & Wilkins, Baltimore.

WINFIELD, D. & O. OZTURK (1959) Electroencephalographic findings in matricide. *Dis. Nerv. Syst.* 20, 1251-4.

WINKLER G. & S. KOVE (1962) Implications of electroencephalographic abnormalities in homicide cases. *Neuropsychiat.* 3, 322-30.

WINKLER G. & G. TRAIN (1959) Acts of violence with electroencephalographic changes. *Quart. Rev. Psychiat. & Neurol.* 20, 223-30.

WOERNER M. & D. KLEIN (1974) 14 and 5 per second positive spiking. Developmental and clinical correlates in psychiatric patients and their siblings. *J. Nerv. Ment. Dis.* 159, 356-61.

WOLFF H. (1969) The role of aggression in the psychopathology of illness. *J. Psychosom. Res.* 13, 315-20.

WOLFGANG M. (1958) *Patterns in criminal homicide*. Univ. Pennsylvania Press, Philadelphia.

WOODMAN D. (1979) Evidence of a permanent imbalance in catecholamine secretion in violent social deviants. *J. Psychosom. Res.* 23, 2, 155-7.

WOODMAN D. & J. HINTON (1978) Anomalies of cyclic AMP excretion in some abnormal offenders. *Biol. Psychol.* 7, 1-2, 103-8.

WOODMAN D.; J. HINTON & M. O'NEILL (1977) Relationship between violence and

catecholamines. *Percept. & Motor Skills* 45, 3 Pt 1, 702.

WOODMAN D.; J. HINTON & M. O'NEILL (1978) Plasma catecholamines, stress and aggression in maximum security patients. *Biol. Psychol.* 6, 2, 147-54.

WOODS N.; A. MOST & G. DERY (1982) Toward a construct of perimenstrual distress. *Res. Nursing & Health*, 5, 3, 128-36.

WOODS S. (1961) Adolescent violence and homicide: ego disruption and the 6 and 14 dysrhythmia. *Arch. Gen. Psychiat.* 5, 528-34.

WORTIS H. & W. MAURER (1942) 'Sham rage' in man. *Amer. J. Psychiat.* 98, 638-44.

WRIGHT S. & G. TARJAN (1957) Phenylketonuria. *Amer. Med. Assoc. J. Dis. Child* 93, 405-

WRIGHT T. (1973) Psychomotor retardation, epileptic and stuporous attacks, irritability and ataxia associated with ammonia intoxication, high blood ornithine levels and increased homocitruline in the urine. *Proc. Roy. Soc. Med.* 66, 221-2.

YARYURA-TOBIAS J. (1979) Gilles de la Tourette syndrome: interactions with other neuropsychiatric disorders. *Acta Psychiat. Scand.* 59, 1, 9-16.

YARYURA-TOBIAS J. & F. NEZIROGLU (1975) Violent behavior, brain dysrhythmia, and glucose dysfunction: a new syndrome. *J. Orthomolecular Psychiat.* 4, 3, 182-8.

YARYURA-TOBIAS J. & F. NEZIROGLU (1978) Compulsions, aggression and self-mutilation: a hypothalamic disorder? *J. Orthomolecular Psychiat.* 7, 2, 114-7.

YOSHII N.; M. SHIMOKOCHI & K. TANI (1961) The electroencephalograms in juvenile delinquents. *Folia Psychiat. Neurol. Japonica* 15, 85-91.

YOSHII N.; T. ISHIWARA & K. TANI (1963) Juvenile delinquents and their abnormal EEGs; 14 and 6 per second positive spike pattern. *Med. J. Osaka Univ.* 14, 61-6.

YOSHII N.; T. ISHIWARA & K. TANI (1964) Juvenile delinquents and their abnormal EEGs; continuous theta waves. *Folia Psychiat. Neurol. Japonica* 18, 161-7.

ZEMAN W. & F. KING (1956) Tumors of the septum pellucidum and adjacent structures with abnormal affective behavior: an anterior midline structure syndrome. *J. Nerv. Ment. Dis.* 27, 490-502.

ZIMMERMAN F. (1956) Explosive behavior anomalies in children on an epileptic basis. *NY State J. Med.* 56, 2, 2537-43.

ZISKIND F. & W. BAILEY (1937) Hyperinsulism. *J. Lab. Clin. Med.* 23, 231-40.

ZUCKER D. et al. (1981) B12 deficiency and psychiatric disorders: case report and literature review. *Biol. Psychiat.* 16, 2, 197-205.