# Neurological Factors in Violent Behavior (The Dyscontrol Syndrome)

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The social, economic, and cultural contributions to intrafamilial violence are so many, and the role of personality disorders and mental illness is so obvious, that the part played by brain damage and metabolic disorders is often overlooked. This is unfortunate because the most dangerous symptom of organic disease—unpredictable attacks of uncontrollable rage in response to seemingly trivial provocation—can usually be prevented by appropriate medication, whereas it is resistant to psychotherapy (Goldstein and Huber, 1974).

Explosive rage is part of the dyscontrol syndrome as defined by Mark and Ervin, 1970. It is one of the causes of wife and child battery, motiveless homicide, unprovoked assault on friends or strangers, sexual assault, dangerously aggressive driving, and senseless destruction of property. Even when the violence is only verbal, it can disrupt marriages and spoil careers, and has a harmful effect on children who are exposed to it in the home.

Outbursts of violence also occur in schizophrenia, manic depressive psychosis, and the personality disorders, as an "acting out" of emotional turmoil, and it is only too easy to ascribe all such attacks to functional psychiatric disorders when in reality some of them are the result of structural or metabolic disease.

Before considering the dyscontrol syndrome in detail, it may be mentioned that neurological disorders can disrupt domestic tranquility in other ways. Difficulty in thinking and communicating hinders the solution of interpersonal problems; this hindrance leads to frustration and anger—and it is easier to hit than to talk. Moreover, many brain-damaged individuals lack perception and miss the significance of the cues which are so essential to smooth social relationships—facial expressions, intonations of voice, and gestures. They often fail to perceive the frustration and anger of others, and sometimes they do not realize their own anger. A few display a perilous lack of a sense of fear. Moreover, as a result of diminished capacity for abstract thought, they often fail to foresee the ultimate results of their impulsive actions.

Organic neurological disorders in the victim also play a part in intrafamilial violence. Whereas a brain-damaged child in a favorable family environment often excites almost excessive devotion from the parents, it is also, however, possible for an aggressive, destructive, hyperkinetic child to evoke antipathy and invite retaliation. This danger also applies to some epileptic children with behavioral problems and to children with learning disabilities caused by minimal brain damage. Needless to say, the interaction between a brain-damaged child and a parent who suffers from emotional dyscontrol can have dire results. Brain damage in the parent may incite violence at the hands of the spouse or adolescent children, especially in social strata where aggression is condoned. A mildly retarded wife or husband who cannot cope successfully with the complexities of life may incur the wrath of the normal spouse who does not realize the true state of affairs. A wife and mother who is aggressive because of unrecognized brain damage will sometimes provoke other members of the family to physical retaliation. There is also the problem of the masochistic or sadistic individual who deliberately or subconsciously prods a

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brain-damaged partner into violent behavior.

### The Organic Dyscontrol Syndrome

This account is based on 70 cases seen in private neurological practice over a period of five years. Ages ranged from 14 to 68; forty were under thirty. There were 25 females and 45 males. Sixty-seven were white and three were black. The majority came from middle and upper socio-economic classes.

| DIAGNOSIS | No. |
|---|---|
| Epilepsy (temporal lobe) | 20 |
| Epilepsy (Jacksonian) | 1 |
| Minimal Brain Dysfunction | 20 |
| Head injury (after age 15) | 12 |
| Brain tumor (post-operative) | 5 |
| Brain tumor (pre-operative) | 1 |
| Encephalitis in infancy or childhood | 2 |
| Multiple sclerosis | 3 |
| Stroke | 2 |
| Arrested hydrocephalus | 1 |
| Alzheimer's disease | 1 |
| Organic brain syndrome, cause not established | 2 |
| | 70 |

More than half were referred for symptoms other than emotional dyscontrol or violence, the existence of which was a carefully preserved family secret which was uncovered only by direct questioning. These cases will be discussed elsewhere.

Kaplan (1899) described what he called the explosive diasthesis in these words: "Following the most trivial and most impersonal causes, there is the effect of rage with its motor accompaniments. There may be the most grotesque gesticulations, excessive movements of the face, and a quick sharp explosiveness of speech; there may be cursing and outbreaks of violence which are often directed towards things; there may or may not be amnesia for these events afterwards. These outbursts may terminate in an epileptic fit. There is an excess in the reaction, with inadequate adaptation to the situation which is so remote from a well considered and purposeful act that it approaches a pure psychic reflex."

Some patients report that explosive rage differs in quality as well as in degree from the ordinary anger aroused by adequate provocation. "Something comes over me, and takes charge of my brain." The intensity of the fury bears little relationship to conventional anger, and there seems to be a total transformation of the personality during these attacks. Occasionally the term dyscontrol cannot be applied, because although the patient feels a catastrophic sense of fury, he or she is able to control it. A young man who developed mild temporal lobe seizures and some intellectual blunting as a result of a head injury reported that when faced with a difficult intellectual task or an irritating social situation, he would experience a sense of rage, but was able to contain it—sometimes by abruptly walking out of the room. Kinnier Wilson (1940) provides an example of the same phenomenon in a young woman who had survived encephalitis lethargica. "She used suddenly to become conscious of a rising surge within her, a seemingly physical wave which flooded her brain and caused her to clench her fists, set her jaws, and glare in frenzy at her mother; 'Had my mother said anything then to cross me, I would have killed her'." Her attacks were followed by remorse.

The explosion may be preceded by a premonitary sense of mounting tension, or a

sense of helplessness and depression, but in other cases, the onset is abrupt and without any warning. The attack can last minutes to hours and is followed in most cases by remorse. A frequent comment is, "How could I do a thing like that?" So great is the remorse that suicide or attempted suicide is not at all uncommon (Mark and Ervin, 1970).

In most cases, the patient remembers what he did or said; in others there is partial or complete amnesia, either because the patient suppresses the memory or because the rage was an ictal (*i.e.*, epileptic) event.

The violence which accompanies the rage may be verbal or physical; in the former, unwonted obscenity and profanity are common. Physical forms of violence often have a primitive quality – biting, gouging, spitting, and so on. An attractive young woman of 25 severely bit a policeman who had reprimanded her for a parking offense. A man who had had temporal lobe seizures in his youth, attacked an elderly woman (whom he liked), and having demolished her skull with a hammer, he stabbed her several times with a kitchen knife and then tried to set fire to the body. Another patient was so incensed by the driver of a car who cut in front of him that he started screaming with rage and slammed his fist through the windscreen. Ervin and Mark (1970) point out that patients who suffer from episodic dyscontrol are frequently guilty of dangerously aggressive driving and have had repeated convictions for traffic violations or a record of serious accidents. Three patients in the present series said that they were afraid to drive.

Impulsive sexual misbehavior in the home or outside is less common than temper dyscontrol in organic cases, perhaps because libidinous activities tend to be reduced by organic lesions of the brain. This is especially evident in temporal lobe epilepsy (Walker and Blumer, 1975). However, there are exceptions. Mohan and his colleagues (1975) describe an example, and quote others, of hyper-sexuality in disease of the limbic system.

Sexual offenses are not necessarily accompanied by anger; a literary example is that of Jacques in Zola's *La Bete Humaine*, a psychomotor epileptic who was not always able to control his urge to kill women to whom he was sexually attracted. It is tempting to speculate that Zola got the idea from the writings of his contemporary, Falret, who in 1861 described epileptics who were the helpless victims of violent impulses.

Many suffer from pathological intoxication in the sense that a small amount of alcohol triggers either rage or drunkenness. (Marihuana, on the other hand, rapidly dissipates rage, according to two personal cases who tried it. Tinklenberg and Woodrow (1974) report that sophisticated drug addicts agree that marihuana is the drug which is least likely to lead to aggression.)

As Kaplan pointed out, an attack of rage may terminate in a seizure, but this occurrence is rare. A young woman who suffered from infrequent temporal lobe seizures reported such an event. She was trying to get a tablet of aspirin out of a bottle when the cap stuck. She found herself screaming with rage and actually "saw red." She then fell to the ground and was seen having a generalized convulsion. Such chromatopsia is an occasional feature of seizures arising in the temporal lobe.

In addition to poor impulse control, the patient may exhibit symptoms and signs appropriate to the underlying condition, whatever it may be. While this condition is obvious in some cases, there are many in whom defects are not disclosed by conventional neurological examination because the signs are "soft" and are revealed only by diligent inquiry or by psychometric tests designed to identify organic disorders. This limitation applies particularly to the cognitive, affective, and somatic disturbances of the syndrome of minimal brain dysfunction (Pincus and Glaser, 1966; Anderson, 1972; Gubbay, 1975). There is often an early history of such defects, which the patient may have outgrown. Indeed the striking thing about many of these patients is that they appear so normal when encountered on the witness stand or on a social occasion. Many of the studies of violence have been conducted on prison populations or in mental institutions, or in neurosurgical institutes to which the most serious cases gravitate, and these reports emphasize the prevalence of a low I.Q. and an adverse domestic environment in early

life—parental dissension and violence, lack of ordinary affection, poverty, and alcoholism. None of these features was conspicuous in the present series.

Only one was mentally retarded, and three individuals with temporal lobe epilepsy exhibited schizophrenia-like symptoms, as described below. The remainder were neither psychotic nor chronically malevolent and were earning a living, running a home, or going to school. Yet members of this group of otherwise pleasant people had been responsible for all the disasters listed in the second paragraph of this paper.

**Prevalence of the Organic Dyscontrol Syndrome**

There is relatively little information on this point. Homicide excepted, there are no statistics as to the prevalence of aggression in the family circle, but it is generally agreed that the cases which come to official notice represent only the tip of the iceberg. This was the opinion of a British Government Committee studying wife battery (1975) which expressed its concern about the problem in the United Kingdom to the extent of advising that refuges for battered wives and their children should be provided to the tune of one refuge per 10,000 population. It is estimated that there are over one million cases of child battery in the United States every year, but nothing is known as to what proportion of these cases are due to organic disorders.

There are many reasons why intrafamilial violence is under-reported. In the first place, a violent temper is often regarded as a quirk of personality rather than a matter for medical concern, especially in strata of society in which violence is so common that it excites little comment. Secondly, few people are willing to admit to an uncontrollable temper, whether from a sense of shame, a fear of committment, or a fear of legal penalties, and the family often helps in the coverup. Euphemisms are the rule. A man who admitted that he had a "short fuse" actually assaulted his wife and children on several occasions and had broken furniture during his rages. To uncover this kind of thing it is necessary for the physician to ask the right questions: "Do you have difficulty in controlling your temper? Have you been charged with traffic violations or dangerous driving? Are you especially sensitive to alcohol?" It is also necessary to inquire, preferably at a later interview, about the more delicate question of inability to control inappropriate sexual impulses.

A third cause of under-reporting is that the violent patient is unpopular with physicians. We try to avoid both the patient and the subject, or we seek a legal remedy as a means of evading the issue, or we attribute the violence to cultural factors. Like the patient, we use euphemisms such as "irritability," "explosive personality," "the hyper-responsive syndrome," and so on.

Uninhibited conduct is a way of life in the lower socio-economic strata of some subcultures, where it excites little comment. In *The Courage of His Convictions* (Parker and Allerton, 1967), a habitual criminal states that "Violence is, in a way, like bad language, something that a person like me is brought up with, something I got used to very early as part of the daily scene of childhood, as you might say. I don't at all recoil from the idea. And I don't have an inborn dislike of the thing, like you do. As long as I can remember I have seen violence in use all around me—my mother hitting the children, my brothers and sisters all whacking their mother or other children, the man downstairs bashing his wife , and so on." This was in England. The same unhappy situation was brought out by Davis (1963), who writes about the American scene. "The lower classes not uncommonly teach their children and adolescents to strike out with their fists or knives, and to be certain to hit first. Boys and girls engage in free-for-all family encounters." Clearly it is easier to identify the dyscontrol syndrome when the attacks constitute a break in the life style of the patient than to recognize it in strata of society in which violence is so common. It is precisely in these strata of society that the organic dyscontrol syndrome is likely to be most common because of the prevalence of

conditions which give rise to it—head injuries, epilepsy, the syndrome of minimal brain dysfunction and a low IQ.

### The Pathophysiology of the Organic Dyscontrol Syndrome

Clinical and experimental evidence indicate that explosive rage often results from disorders which affect the limbic system, a phylogenetically ancient portion of the brain which is interposed between the diencephalon and the neocortex. There has been much discussion as to the anatomical limits of the system, but the portions of it which are pertinent to the study of rage are the amygdala and the hippocampus in the temporal lobe, the hypothalamus, the cingulate gyri and cingulum, the septum pellucidum and septal area, and related portions of the thalamus, basal ganglia, orbital region of the frontal lobe and mid-brain. This system is intimately concerned, not only with the expression of emotion, but also with the neural control of visceral functions and chemical homeostasis (Isaacson, 1974; MacLean, 1956).

An early hint of the link between the limbic system and rage was unwittingly furnished by Boerhaave (1715), who spoke of patients "gnashing their teeth and snarling like a dog" when suffering from rabies, which attacks the hippocampus and the brain stem. In his description of this disorder Gowers (1892) spoke of the patient being "exhausted by attacks of fury."

In 1892 Goltz reported that removal of portions of the cerebral hemispheres in dogs produced a phenomenon which later came to be called sham rage. These animals are liable to periodic attacks of rage, and also snarl and bite in response to minor provocation.

Other workers confirmed these studies, and by carrying out a series of systematic ablations in cats, Bard (1928) found that the posterior hypothalamus had to be intact if sham rage was to appear. Many years later Sano (1966) showed that in man explosive rage could be abolished by bilateral lesions in the posterior hypothalamus.

Although the literature on this subject speaks of the "decorticate" animal, the term is misleading because in most cases a good deal more than the cortex was removed. Indeed, Bard and Mountcastle found that in cats, removal of the neocortex only, leaving the limbic cortex intact, produced placidity. "Even with the most painful stimulus, these animals could not be made to show the slightest signs of anger, nor did they show any of the autonomic responses; no pupilary dilatation, no increase in cardiac rate,no increase in cardiac output, no sweating, not even a rise of blood sugar. These passive animals had all of the hypothalamus and the whole limbic system intact and showed not the slightest reaction of aggression or rage" (Bard, 1974). This observation is in accord with clinical experience that explosive rage is far more common in disorders involving the limbic system than it is in diseases of the neocortex.

Experiments with animals and man show that the limbic system contains within itself both excitatory and suppressive mechanisms. Electrical stimulation of the central and medial portions of the amygdala complex usually induces rage, whereas the animal is pacified by stimulation of the lateral portion of the nucleus. Bilateral amygdalotomy *usually* reduces the ferocity of Rhesus monkeys, the lynx and other animals. In man, this operation *usually* abolishes the explosive rage of the dyscontrol syndrome. Narabayashi reports more consistent improvement when the lesion is made in the medial portion of the lateral nucleus of the amygdala.

In the cat, destruction of the ventro-medial nucleus of the hypothalamus produces—after a delay of weeks—a chronically savage animal. Possible reasons for this delay are discussed by Glusman (1974). A more prolonged delay is sometimes seen in the development of explosive rage in man, following the removal of brain tumors involving the limbic system, after head injuries, and after encephalitis.

In man, electrical stimulation of limbic structures can produce both pleasant and unpleasant sensations; of the latter, a sense of fear and apprehension is the most common.

Anger is rarely induced in the operating room (Gloor, 1975). Yet as we all know, a sudden fright can provoke anger, so that this discrepancy may be due to the social setting of the experiment. There have been a few notable exceptions. In a case described by Heath, Monroe, and Mickle (1955), stimulation in the region of the amygdala produced fear on some occasions and anger on others, and Ervin and Mark (1970) provide a well-documented example of violent attack behavior triggered by electrical stimulation in the region of the amygdala. Again, stimulation of the central grey matter of the brainstem in Gibbon monkeys, carried out in the laboratory, caused attacks on other animals, but this did not occur when the stimulation was carried out by radio waves in animals running free in their natural habitat—illustrating the effect of the social setting (Delgado, 1969, p. 132). In a human case, stimulation of the medial portion of the amygdala produced the prodromata of attack behavior without the attack itself on some days, but if the patient had been disturbed by a prior argument of the family, electrical stimulation at the same point triggered an attack of uncontrolled violence (Mark, Sweet, and Ervin, 1975).

Clearly, the limbic system does not act in a vacuum as an autonomous center for aggressive behavior, but interacts with other portions of the brain. This is illustrated at the electrophysiological level by the fact that stimulation of midline structures of the cerebellum can inhibit sham rage, and stimulation of the caudate nucleus has a pacifying effect. Nevertheless, the explosive rage of the organic dyscontrol syndrome is usually the product of a damaged limbic system, and it has been controlled in a large number of children and adults by stereotaxic operation on the amygdala, posteromedial hypothalamus, the cingulate gyri and underlying cingulate bundle, and the anterior thalamus, and also by unilateral temporal lobectomy, bilateral temporal lobotomy, and orbito-frontal tractotomy (Hitchcock *et al.*, 1972).

Predatory aggression in animals (for instance, the lion in search of a meal) involves other circuits which need not concern us here. In man, predatory violence is cold and calculated, and is carried out for profit. It is properly viewed as originating in the neocortex. In the context of intrafamilial violence, it usually takes the form of murder for financial gain, or to secure freedom from an unwanted spouse; exceptionally it is merciful in intent. Sometimes the distinction between predatory and affective aggression is blurred, as in the case of an individual who plans a crime of violence in cold blood and carries it out under the influence of alcohol or drugs which are taken to produce "Dutch courage."

### Etiology of the Organic Dyscontrol Syndrome

This condition can occur at any age, but it is most common in adolescence and early adult life. In general, aggression declines with advancing age, but it can still develop for the first time in old age when the brain is assailed by organic disease such as a stroke or Alzheimer's disease.

Males are more affected than females, at all ages. Aggressive behavior is sometimes associated with high androgen levels in the plasma, and in most animals, male castration, which lowers androgen levels, has a taming effect. This is illustrated by the difference between "the raging bull and the peaceful steer" (Moyer, 1971). However, Bremer (1959) found that in man castration did not inhibit aggression except in relation to sexual crime, but this observation ignores the possible psychodynamic effects of castration. Men with an extra male chromosome—the XYY type—are often, but not always, more aggressive and more given to crimes of violence than men with a normal chromosome pattern.

The family background is important. An uncontrollable temper sometimes runs in families, involving several generations and affecting about half of the progeny of a violent parent (Davenport, 1915). The writer has encountered one pedigree in which typical explosive rage appeared in three generations, affecting precisely half the sibship in each generation. There was no evidence of neurological disease, epilepsy, or overt mental

illness in this cultivated family. Hill and Watterson (1942) report that aggressive behavior was found nearly three times as frequently among first degree relatives of aggressive psychopaths as amongst those of "inadequate" psychopaths, but they did not distinguish between explosive rage and other types of aggression. There is a high incidence of EEG abnormalities in the families of patients showing episodic aggressive behavior (Mitsuda, 1967). A genetic factor is also evident in temporal lobe epilepsy (Bray, 1964), although structural lesions are present in the majority of cases—sclerosis of Ammon's horn, porencephalic cysts, hamartomas, benign glial tumors, and so on (Mathieson, 1975). It may be that it is the genetic factor which determines whether such lesions will produce epilepsy and/or the dyscontrol syndrome. Thus, sclerosis of the hippocampus is the most common cause of temporal lobe seizures (Falconer *et al.*, 1964). It is usually due to the hypoxia produced by severe febrile convulsions, and it is possible that while the convulsive response to pyrexia is genetically determined, the subsequent development of temporal lobe seizures is due to sclerosis of Ammon's horn produced by the original convulsions.

Most observers agree that some—but not all—of the children reared in an atmosphere of uncontrollable temper, parental dissension or separation, and emotional deprivation become violent themselves, and it is not always easy to decide whether the effect is due to heredity, emotional trauma, bad example, or a mixture of all three. Nevertheless, many children brought up in this atmosphere do *not* become violent.

Patients with the organic dyscontrol syndrome fall into two groups. In the first, there is a history of temper tantrums in infancy and childhood which has persisted as more formidable explosions of rage in adolescence and adult life. In the second group, formerly normal individuals become subject to explosive rage as a sequel to a brain insult or metabolic disorder.

In the first group, the emotional dyscontrol dates from early life and can often be traced to prenatal, natal, or postnatal events. These include birth trauma, fetal anoxia, infantile convulsions, head injury, encephalopathy, complicating infectious diseases, and encephalitis. These infants are difficult to rear, often reach the milestones of development late, and are marked in childhood by seizures or the protean manifestation of minimal brain dysfunction including hyperkinesis. Explosive rage also occurs in more severely affected individuals with cerebral palsy and mental retardation. It is often difficult to identify the precise cause of the disability because infants with congenital defects are often premature and are therefore particularly liable to birth injury. A prolonged and difficult labor at term is not uncommon in the history of infants who subsequently develop epilepsy, minor brain dysfunction, or cerebral palsy. Psychoanalysts have sought a link between a violent birth and a violent child; an alternative explanation is that violent birth damages the brain in a structural sense.

In the second group, explosive rage appears in a previously normal subject as a result of head trauma or other cerebral insult.

### Head Trauma

After cerebral concussion the patient often goes through an aggressive and combative stage which may last minutes, hours, days, or weeks. This is followed by headaches, lightheadedness, lack of energy, and irritability; in the more severely affected cases, there is intellectual loss and personality disorder with or without traumatic epilepsy. A change of personality is particularly prominent in some children following severe injuries; they display a total change of character, often without significant intellectual loss, a situation reminiscent of what happened to some children following epidemic encephalitis. As Blau (1936) described it, "A previously normal child becomes asocial, unmanageable, and unyielding to any form of training. Hyperkinesis is an outstanding symptom and is shown by marked restlessness and hyperactivity. They become disobedient and disrespectful

towards their parents in marked contrast to the other siblings, and frequently run away from home for long periods. Emotional upsets, temper tantrums, and marked irritability are frequent. Usually there is marked compulsiveness and even an explosive manner in their activities. Common antisocial trends include unrestrained aggressiveness, destructiveness, quarrelsomeness, cruelty to younger children and animals, lying and stealing. Their whole personality is essentially egocentric and self-interested, with total disregard for the welfare of others.... They are disruptive in classrooms so that suspension is unavoidable. As they grow older their school grades and accomplishments become poorer and they may be considered mentally retarded."

Head injuries in adults are less devastating, but character disorders and pathological rage are not rare. Hooper and his colleagues (1945) found 12 cases of explosive rage in 2,000 men who had suffered serious head injury in World War II.

In the author's experience, explosive rage sometimes develops two or three years after the initial trauma. The contre coup lesions of closed head injuries show a predilection for the tip and orbital surface of the frontal lobe and the anterior portion of the temporal lobe, and Crompton (1971) has found pathological evidence of damage to the anterior hypothalamus. In other words, portions of the limbic system which are known to be concerned with the production of affective aggression are particularly vulnerable to severe closed head injuries; multiple minor head injuries can have a cumulative effect, as in the "punch drunk syndrome" in which explosive rage is not uncommon.

### Minimal Brain Dysfunction

This label is a convenient one for a wide variety of mild cognitive, motor, and sensory defects which may be developmental or due to prenatal, natal, or postnatal insults to a formerly normal brain (Pincus and Glaser, 1966; Anderson, 1972; Gubbay, 1975). Defects are noted in infancy and childhood and in many cases they lessen, or disappear entirely, thanks to the plasticity of the youthful brain. Others such as pure dyslexia and right-left disorientation may persist throughout life; this is usually taken to mean that they are developmental in origin. Thus, pure dyslexia can be hereditary. Symptoms to be looked for in the early history of patients with the dyscontrol syndrome include specific learning defects, constructional apraxia, difficulty with geographical relationships, left-right disorientation, poor attention span, incapacity for abstract thought and introspection, circumstantiality, imperception, excessive synkinesis, clumsiness, mild choreiform movements, impairment of stereognosis and graphesthesia, hyperkinesis, and visuo-motor defects.

Children with dyslexia and other forms of minor brain dysfunction are prone to develop neurotic reactions, including aggressive behavior. Critchly (1970), writing about the dyslexic child, says that "It is a commonplace observation that once a dyslexic child is diagnosed as being the victim of a genuine inherited disability and is not an ordinary stupid, lazy, or neurotic youngster, its self respect is immediately enhanced and any bad behavior they have shown improves without intervention on the part of child psychiatry." This is often true, but not always. Some patients, notably those who suffered a natal or postnatal brain insult, have temper tantrums in their earliest years, and these may persist through adolescence and adult life even if the patient has outgrown his other handicaps. The fact that in some of these individuals, explosive rage does not respond to psychotherapy but does respond to medication suggests that the dyscontrol is organic in origin rather than simply a response to frustration.

### Epilepsy

It is widely believed that temporal lobe epileptics are more prone to behavioral disorders than are patients with centrencephalic epilepsy. The reported incidence varies

with the source of the cases (Stevens, 1975). As is to be expected, it is higher in epileptics who have been committed to institutions than in those who can live at home. It is also high in those admitted to neurosurgical clinics because they are a selected group of intractable cases. Rodin (1973) reported pathological aggression in only 4.8% of 700 cases from the Michigan Epilepsy Center. Currie and his colleagues (1970) at the London Hospital found it in 7% of 666 patients with temporal lobe epilepsy of mixed etiology and type. Bingley (1958), who drew his material from the neurological and neurosurgical services of a general hospital in Sweden, found aggressive behavior in 17% of 90 cases of temporal lobe epilepsy. Gastaut (1955) reported paroxysmal rages in 50% of temporal lobe epileptics. Falconer found pathological aggressiveness, occurring in outbursts in otherwise well-adjusted individuals, in 38% of 50 cases of temporal lobe epilepsy with a predominately unilateral spike focus; another 14% had a milder or more persistent aggressiveness associated with a paranoid outlook. This distinction between explosive rage in an otherwise adjusted personality, and aggression resulting from a a paranoid outlook, is not always made in the literature; moreover, it is often difficult to know whether the term "aggressive behavior" refers to affective or predatory aggression.

In the present series, seizures were present or had been present at some time in the patient's life in 39 cases. Of these none had the bilateral synchronous discharges of centrencephalic epilepsy. Thirty-two had spikes or slowing originating in the temporal lobe. Five showed epileptic discharges which did not appear to originate in the temporal lobe, and two had bilateral temporo-frontal theta waves. The EEG was abnormal in an additional thirteen patients who had never had a seizure. Seven of these displayed epileptic temporal lobe discharges, four had bilateral temporo-frontal theta activity, one had right frontal slowing following the removal of a memingioma, and one had unilateral fronto-parietal slowing in association with Jacksonian seizures.

The prevalence of abnormal EEG's in this group is surprisingly high (70%) considering the fact that no chemical activation procedures were employed and that sleep records with nasopharyngeal electrodes were used in only one-third of the cases.

In epileptics, episodic rage occurs under three circumstances. It can occur when attempts are made to restrain the patient during post-epileptic automatism. Secondly, patients with temporal lobe lesions, epileptic or otherwise, often have inter-ictal explosions of rage on trivial provocation. Thirdly, there is ictal rage. Many authors believe that rage can be part of a seizure, but Gloor (1975) believes that it is a very uncommon manifestation of an epileptic discharge, though he agrees that the association of aggressive behavior and temporal lobe epilepsy is a real one—conclusions based on the hundreds of cases of epilepsy examined at the Montreal Neurological Institute. The fact that ictal rage is rarely uncovered by electrical stimulation of the brain in conscious patients emphasizes the role of the social setting in experimental work, to which attention has already been directed. In his analysis of 100 cases of temporal lobe epilepsy, Williams (1969) identified 17 cases of ictal aggression. In the present series four cases were subject to attacks of violence which came on abruptly out of a clear sky, lasted a few minutes, and were followed by amnesia, exhaustion and headache.

Interictal rage is not uncommon. In some the attacks of violence are preceded by a period of mounting tension which may be obvious to others if not to the patient. In others, however, the onset is abrupt and without warning, and it is difficult to know whether they should be regarded as ictal or interictal. The presence or absence of amnesia is not decisive, since many patients with temporal lobe seizures can describe what happened during the attack. The attacks occur in response to seemingly minor provocation. The word "seemingly" is used advisedly because provocation which appears trivial to the observer may have psychodynamic significance for the patient. Thus, a temporal lobe epileptic attempted to strangle his wife because a remark she made reminded him of his mother, whom he had hated. The wife was a surrogate victim. The patient usually remembers most of what he did or said during these attacks, unless it is

advantageous to forget it, and remorse usually follows. Occasionally the patient insists that his behavior is appropriate.

Precisely similar episodes occur in people who have never had a seizure, but whose EEG's display evidence of a seizure disorder. It is important to recognize these cases for what they are, because the dyscontrol usually responds favorably to anti-convulsant medication. They have to be distinguished from a larger group of individuals who are given to recurrent violence, who are not epileptic, and whose EEG's display bilateral paroxysmal slow waves, predominantly in the anterior temporal and frontal regions. This theta (5-7 HZ) activity is frequently to be seen in the resting record, but the number of positive results is greatly increased by activation techniques, notably by using alpha chloralose (Monroe, 1970). Williams (1969) studied the EEG's of a large group of patients who were habitually aggressive. After those who were mentally retarded or who had epilepsy or who had had a major head injury were removed from the series, the EEG was abnormal in 57%, whereas an abnormal EEG was found in only 12% of apparently normal people who had committed a solitary major violent crime—the same as in the population at large. Bilateral theta activity was the most common finding in the aggressive group. Williams concludes that "the distribution and type of the EEG abnormalities suggest that the primary disturbance of function responsible for them is in the diencephalic and mesencephalic components of the reticular activating or 'limbic' mechanisms which have their densest projections to the anterior temporal and frontal cortex." Theta activity is found in apparently normal children, and becomes progressively less prevalent in early adult life; it may well represent a maturational lag (Monroe, 1970).

It must be emphasized that ictal aggression plays a negligible role in criminal violence. The notion that a premeditated crime carried out for gain or revenge can be attributed to ictal discharges or post-ictal automatism is inconsistent with all that is known about epilepsy. Ictal, post-ictal, and interictal aggression are poorly organized, senseless, and out of character.

Explosive rage is only one of a number of disturbances which may be associated with temporal lobe epilepsy. Intermittent symptoms include depersonalization, depression, free-floating anxiety, and hallucinations. These patients, unlike most psychotics, usually describe their experiences objectively and accurately.

More persistent symptoms include personality disorders and a schizophrenia-like syndrome. In an important paper, Slater, Beard, and Glithero (1963) reported—as had others before them—the development of acute psychotic episodes closely resembling schizophrenia many years after the onset of temporal lobe seizures. The mental symptoms often appear at a time when the frequency and severity of the seizures is waning. The mode of onset can be acute, sub-acute, or insidious, and the disorder tends to be episodic. Psychiatric examination shows a high incidence of personality changes with an organic complexion, but these patients differ from true schizophrenics in that the personality usually remains warm and outgoing. As Rodin (1975) has pointed out, "The temporal lobe patient can be brought to tears or be made to laugh gaily within a matter of minutes by a skillful interviewer. . . . This cannot be done with patients with what one calls primary schizophrenia." Moreover they are not chronically irritable or malevolent, and the attacks of rage constitute a break in the life-style of the patient. In the patients described by Slater and his colleagues, pneumoencephalography disclosed the presence of an atrophic process of the brain in 37 out of 56 cases.

A personal case illustrates some of these features. An intelligent woman of 30, with a warm and pleasant personality, developed temporal lobe attacks. In only two of these did she lose consciousness. Most of the attacks consisted of a momentary dreamy state during which external objects appeared unfamiliar. Over the same period she became subject to attacks of what she called insane rage. She was unaware of any external or internal reason for her anger. She might go down to the cellar and start breaking things or scream at her children or her husband for no reason at all. These attacks disturbed her greatly, and she had been under psychotherapy for 8 years, without benefit. She complained of episodes

of depersonalization. Sometimes she felt that "my head is in one position, my mind in another, and my body in a third." Occasionally when she would reach for an object it would seem to move slightly. She also suffered from geographical and left-right disorientation. She achieved good grades in school except in mathematics; even the simplest calculation was, and is, beyond her capacity. She suffers from pathological intolerance for alcohol which was not present in her earlier years. An electroencephalogram three years after the onset of the seizures showed epileptic spikes in all leads, especially in the left temporal area. More recent tests show diffuse slowing without any focal or paroxysmal activity. The seizures, the attacks of explosive rage, and her schizoid symptoms disappeared entirely when she was given 400 mgms. of Dilantin daily, but a new symptom developed. She had been aware for most of her life that she was devoid of an ordinary sense of fear, such as other people have. It did not disturb her to go out alone at night in a bad neighborhood. Under Dilantin, she developed a normal sense of fear and apprehension under such circumstances. The therapeutic response in this case supports Symonds' suggestion (Hill, Pond, Symonds, 1962) that the schizophrenic features encountered in some temporal lobe epileptics are the result of interference with thought and perception by persistent subclinical seizure activity in the temporal lobe. The same inference may be drawn from the remarkable decrease of both seizures and personality disorders, including aggression, which follows hemispherectomy in children with infantile hemiplegia (White, 1961).

### Brain Tumors

Tumors involving the limbic system and colloid cysts of the third ventricle are apt to produce apathy even before intracranial pressure has risen (Elliott, 1969). They can also give rise to angry aggressive behavior. This occurred in 6% of 250 cases of temporal lobe glioma studied by Bingley (1958) and in four out of 17 cases reported by Malamud (1967). It has been seen in a dermoid cyst of the third ventricle (Alpers, 1937) and in hypothalamic tumors, sub-frontal meningiomas, tumors involving the cingulate gyri, tumors and cysts of the septum pallucidum, and glioma of the optic chiasma. A personal case: an intellectually precocious boy of 14 with undescended testes and an infantile penis gave a two-month history of episodic rage in which he became violent. Between attacks he was frightened and remorseful. There were no abnormal signs on neurological examination or pneumoencephalography, but an EMI scan showed a fatty tumor lying between the hypothalamus and the pituitary fossa—one of the few sites in which intracranial lipomata are found.

Explosive rage sometimes occurs for the first time after the removal of a cerebral tumor. This occurred in 5 cases in the present series—a glioma of the temporal lobe, 3 parasaggital meningiomas and a sphenoid ridge meningioma.

### Infections

Viral and bacterial invasion of the brain are occasional causes of explosive rage and aggressive behavior, which usually appear *after* the acute phase is over; it can also occur in the acute stage of cerebral malaria and rabies. Gower (1892) speaks of patients with rabies who are "exhausted by attacks of fury," and it is of interest to note that the Negri bodies of this disease are most numerous in the hippocampus. Episodes of rage and destructiveness can also occur in the early stage of general paresis and are sometimes seen following severe bacterial meningitis and cerebral abscess.

Viral encephalitis has provided some of the most dramatic examples of post-infective episodic rage. It occasionally occurs as a sequel to herpes simplex encephalitis, which has a predilection for the temporal lobes. Herpes encephalitis can also produce extreme placidity, the reverse of hyper-irritability. Corsellis and his colleagues have reported on

the anatomical findings in three patients who died, after prolonged survival, from this disease. During life they presented a Kluver-Bucy syndrome—extreme placidity, inability to retain memories for even a minute, and a marked oral tendency. At autopsy they were found to have almost complete destruction of the anterior portion of both temporal lobes, including the amygdala (Corsellis, Janota, and Hierons, 1975). The epidemic of encephalitis lethargica which occurred in the second and third decades of this century provided many examples of the dyscontrol syndrome. This disease could change a previously pleasant child into a cruel, aggressive psychopath, given to lying, stealing, and violent behavior (Wilson, 1940).

### Cerebral Vascular Disease

A stroke or subarachnoid hemorrhage occasionally causes explosive rage either as the patient emerges from the coma or as a delayed phenomenon. In the present series it occurred in a man who had a subarachnoid hemorrhage accompanied by left hemiparesis, and in another man who had multiple transient ischemic attacks involving both the carotid and the vertebral basilar systems. The writer has also seen the reverse situation. A physician had been prone to severe temper outbursts throughout his life. He often assaulted his wife. He was hypertensive and diabetic and developed a mild hemiparesis as a result of a cerebral infarct. This incident increased his violence. One evening, however, he complained of unaccustomed dizziness and appeared a little confused and the next morning his wife noticed a striking change in his temperament. He had suddenly become benign and remained so for a year until his death from a myocardial infarction. During this period he never lost his temper and was kind and considerate; he was also rather irresponsible and facetious. Autopsy revealed so many small infarcts in both hemispheres and in the brain stem that it was impossible to draw any conclusions as to which was responsible for his change of behavior.

### Miscellaneous Neurological Diseases

Paroxysmal rage is sometimes seen in pre-senile and senile dementia. It is not uncommon with or without schizophrenic-like symptoms, in Huntington's chorea, a disease in which there is degeneration of the caudate nucleus and putamen. These structures are not usually considered to be part of the limbic system, but electrical stimulation of the caudate nucleus has a pacifying effect on Rhesus monkeys. Episodic dyscontrol has also been reported in normal pressure hydrocephalus, and in the present series it was the sole symptom of arrested internal hydrocephalus in a young man. Multiple sclerosis of late onset appeared to be responsible for the development of temporal lobe seizures and the dyscontrol syndrome in two personal cases; this disease was present in 1% of 666 cases of temporal lobe epilepsy studied by Currie and his colleagues (1971). However, the association between multiple sclerosis and seizures is sometimes fortuitous, post-mortem examination revealing the presence of some other disease in addition to the multiple sclerosis.

In the Down syndrome (mongolism), children generally are passive and happy, but if they survive to the third or fourth decade, some develop a pre-senile dementia akin to Alzheimer's disease, together with aggressive behavior and seizures. In these cases neurofibrillary degeneration is especially marked in the hippocampal gyrus, the cortex of the temporal lobe, and the cingulate gyrus (Jervis, 1948). Aggressive behavior is also characteristic of certain rare metabolic disorders involving the nervous system—the San Filippo syndrome, phenylketonuria, and the Spielmeyer-Vogt disease.

Diseases which involve the hippocampus do not *necessarily* produce either seizures or disordered behavior. The same inconsistency is seen in tumors involving the limbic system, and all the evidence derived from experimental procedures in animals and

psychosurgical operations in man points to the fact that both the production of rage and its control depend on the precise position of lesions within the limbic system. Even within the amygdala complex alone, the exact locus of the lesion may determine whether rage or placidity is to be the result. Moreover, in man heredity appears to pay a part in determining whether a given lesion will or will not give rise to seizures, and the same may be true of episodic rage.

### Endocrine and Metabolic Disorders

Of the metabolic disorders that can trigger explosive rage, hypoglycemia is the most common, whether it be functional, iatrogenic, or due to an insulinoma.

Wilder (1940 and 1947) has assembled a formidable bibliography of violent behavior triggered by hypoglycemia, including references to many instances of intrafamilial strife. Hill and Sargant (1943) described a case of matricide caused by hypoglycemia in a man who had suffered brain damage at birth or in infancy. A modest fall of blood sugar induced by intravenous tolbutamide activates epileptic discharges in the EEG in some patients with seizures (Green, 1963, Pampiglioni, 1973). Since hypoglycemia is usually marked by sweating, *early* flushing of the face and a sense of weakness, rather than by violence, the question arises as to whether the occurrence of explosive rage under these circumstances should suggest the presence of epilepsy or structural disease, as in Hill and Sargant's case. By way of analogy, hypoglycemia can produce hemiparesis, monoparesis, or aphasia when it occurs in individuals with carotid stenosis.

The fact that rage does not appear during the hypoglycemia induced by a five-hour glucose tolerance test, even in people who suffer from hypoglycemic anger outside the laboratory, emphasizes the role of the social setting. In the same way patients who are liable to pathological rage after drinking a small quantity of alcohol in social life do not develop symptoms from the same amount of alcohol when it is given to them in the laboratory by mouth or intravenously.

Angry aggressive behavior is seen from time to time in Cushing's disease and in the Cushing syndrome. It can also occur acutely along with other symptoms of a toxic psychosis, as a result of hypocalcemia induced by parathyroidectomy.

Premenstrual tension in women provides another example of the effects of metabolic and chemical factors on the rage threshold, a circumstance which is familiar to many. Morton and his colleagues (1953) found from a study of the records of women prisoners that 62% of their violent crimes were committed during the premenstrual week and 2% at the end of menstruation. This trend is confirmed by Dalton (1961). The premenstrual syndrome is accompanied by depression, irritability, and feelings of futility and paranoia; presumably in individuals with inadequate controls, these feelings generate aggressive behavior. More information is needed as to whether premenstrual tension can evoke episodic rage, in the absence of psychological or neurological disorders.

### Diagnostic Aids

The study of patients suffering from the dyscontrol syndrome requires a complete medical history which must go all the way back to the womb. Precise details are desirable as to prenatal, natal, and postnatal events, and subsequent illnesses and injuries. Evidence of postnatal hypoxia and infantile convulsions is important, because they are a frequent precursor of temporal lobe epilepsy. It must also be remembered that repeated minor head injuries have a cumulative effect on the brain, both in childhood and in adult life. A careful historical search must be conducted to detect failure to reach the physical and mental milestones of development at an appropriate age, and a search must also be made for evidence of minimal brain dysfunction in childhood.

The family history must be scrutinized for evidence of epilepsy; heredity plays a part

not only in the centrencephalic form of *grand mal* and *petit mal*, but also in temporal lobe epilepsy, despite the fact that a structural lesion is present in a great majority of the latter. Bray and Wiser (1964) found that 30% of their patients with temporal lobe spikes and sharp waves, who had had seizures, had a family history of epilepsy. Other things to be looked for include mental illness, personality disorders, an ungovernable temper, alcoholism, intrafamilial strife, abandonment by a parent, lack of affection in infancy, and brutal treatment in infancy and childhood.

Psychiatric examination is desirable not only to throw light on intrafamilial psychodynamics, but also to assess the patient's personality before the brain injury occurred. Psychological tests designed to identify physical disorders are useful when there is no overt evidence of organic disease.

A routine neurological examination itself seldom discloses anything amiss, but a search for evidence of specific learning deficiencies often proves fruitful. The history must be carefully scrutinized for evidence of epilepsy, which can assume subtle and easily ignored forms.

Laboratory investigation starts with electroencephalography. A normal result from a single test with scalp electrodes has little value. There is an appreciable increase in the yield of positive findings on the second or third test; this may be due in part to the fact that the patient has become more relaxed about the procedure. Sleep records with nasopharyngeal electrodes, which can pick up discharges from the medial aspect of the temporal lobes, are essential, and the yield is still further improved by activation techniques—hyperventilation, photic stimulation, and the use of pharmacological agents. Even when all these methods fail, and even when recordings taken from the surface of the brain prove negative, seizure activity may be found in deep subcortical structures by using depth electrodes. Most clinicians have to make a diagnosis without these sophisticated techniques; they are mentioned only to emphasize the limitations of the scalp-electrode EEG and the danger of concluding that because a scalp EEG is normal, all is well within the brain.

Plain x-rays of the skull should always include half axial views to display the base, because in patients with temporal lobe seizures, the middle fossa on one side may be smaller than the other, signifying that something has happened in early life to stunt the growth of the brain on that side.

Computerized axial tomography (the EMI scan) makes it possible to identify morphological abnormalities such as focal dilations of the ventricular system (notably the temporal horn), porencephalic cysts, severe cortical atrophy, tumors, angiomatous malformations, and internal hydrocephalus. This non-invasive outpatient procedure is proving valuable in identifying organic pathology in cases suffering from explosive rage even when pneumoencephalography and arteriography have failed to reveal anything amiss, but it is necessary to remember that small lesions such as the all-important medial temporal lobe sclerosis may escape detection.

A five-hour glucose tolerance test should be carried out if there is the slightest suggestion of hypoglycemia.

### Treatment

Explosive rage is a symptom of many disorders, psychological and physical, structural and metabolic, congenital and acquired, and in many cases it has multiple roots. The presence of organic disease, for instance, does not preclude the development of a functional psychosis, and *vice versa*. Moreover, the sense of guilt caused by the organic dyscontrol syndrome often gives rise to a train of secondary emotional disorders, while the brain damage which is causing the attacks of rage can itself produce disorders of thought, emotion, and behavior. These circumstances give rise to problems in the management and treatment of the patient. Should treatment be in the hands of the

psychiatrist or the neurologist? Rodin (1975), from his great experience of epileptics, says, "I have tried in a number of these difficult treatment problems to restrict my role to the management of the seizure disorder itself and to have the psychiatrist take care of the mental and emotional problems. It has never been successful. Direction in the total management was lacking and a characteristic question was: 'Is this a symptom I should be talking to you about, or to Dr. X?' The neurologist has been played against the psychiatrist in the same way as children tend to play one parent against the other to obtain their gratifications. Unless the neurologist and the psychiatrist consult constantly with each other after each patient's visit, they cannot achieve anything worthwhile." The same point has been made by Monroe (1970). This problem also arises in dealing with the dyscontrol syndrome in non-epileptic subjects. There is no easy solution. It is essential for one doctor to take charge, and there should be agreement, not only as to the general strategy of treatment, but also as to the identity and dose of medications to be used. Even this *detente* often fails to bridge the gap between psychodynamics and neurology. As one bewildered patient put it, "After ten years of psychiatric treatment I have come to believe that my troubles are due to a sexual hangup. Now *you* tell me that it is all due to minor brain damage. Whom am I to believe?"

The social, psychological, and pharmacological management of the dyscontrol syndrome has been reviewed by Moyer (1971) and discussed at some length by Monroe (1970), Lion (1972), and Lion and Monroe (1975). Their writings should be consulted to flesh out the bare bones of the subject as presented here. There can be no doubt that the lives of these patients, and of their families, can be transformed by appropriate treatment.

The patient can often be taught to recognize a premonitary dysphoria and to take evasive action by walking away from irritating confrontations, by calling his physician, or by going to a clinic. A "hot-line" similar to what is used for suicide prevention is desirable in any clinic that deals with violent patients (Lion, 1972), because whether the patient's impending rage is psychogenic or organic, or a mixture of the two, it can often be averted by a discussion with somebody outside the family circle.

In the organic type of dyscontrol it helps to explain that the attacks are largely physical in origin and will almost certainly respond to treatment. Reassurance is sorely needed, particularly by those who have vainly sought help in the past. The fact that their physician understands, and is willing to help, does much to allay the hostility and skepticism of these reluctant patients.

Alcohol must be avoided if it triggers attacks of rage.

It is prudent to point out to the patient and his family that it may take a little time to find the right drug and the correct dose. Paradoxical drug reactions are not uncommon, and the writer has seen cases in which the attacks of rage were aggravated by Dilantin, Primidone, Valium, the phenothizaines, and meprobamate. It is therefore desirable to proceed cautiously, starting with a small dose and working up to the limits of tolerance, bearing in mind the fact that if the first drug prescribed does not work at once or has undesirable side effects, these impulsive individuals are apt to go elsewhere. There are two types of intolerance: the first is indicated by the appearance of conventional toxic symptoms and signs; the second takes the form of an increase of the symptoms for which the drug is being given.

An occasional reason for therapeutic failure is that the patient does not take his medication regularly, or is not taking it at all, because he lacks insight and sees no need for treatment, or because the attacks bring him secondary gain such as avoidance of responsibility or the domination of others in the household, or because attacks of rage afford welcome relief from emotional tension. Some forget to take their medication; others who are unemployed and penurious are disinclined to spend money on expensive drugs.

The attacks can usually be reduced and sometimes abolished by pharmacological agents. These should be given on a maintenance basis when the attacks come on without

warning, but can be taken intermittently, at times of mounting tension in patients who are aware of the prodomal symptoms, as, for instance, in women whose attacks are mainly premenstrual.

If there is clinical or EEG evidence of epilepsy, anti-convulsant medication is the first choice, preference being given to the hydantoinates or carbamazepine. The hydantoinates sometimes prevent explosive rage even in the absence of clinical or EEG evidence of epilepsy (Monroe, 1970, and personal observation), and the same is true of carbamazepine (Dalby, 1975). Primidone has been less useful in the author's experience, though others recommend it (Monroe and Wise, 1965). It often reduces the seizures, but it sometimes aggravates the aggression; moreover, the dose needed to suppress seizures may cause an unacceptable degree of sedation. The same applies to barbiturates. Phenobarbitone and secobarbital, whether alone or with alcohol, are recognized by young addicts as the drugs most likely to induce aggressive behavior (Tinklenberg and Woodrow, 1974).

If anti-convulsants fail, meprobamate (which has a special effect on the amygdala and thalmus) should be added in doses of 400 mgms. b.i.d., or one of the benzodiazapines can be used, *e.g.*, Oxazapam, Valium, or Librium. Phenothiazines, so useful in the control of psychotic hostility, sometimes aggravate the organic dyscontrol syndrome. This may be because they lower the seizure threshold. They can be used in conjunction with anti-convulsants.

The amphetamines are effective in some aggressive hyperkinetic children and in some immature adults who have outgrown their hyperkinesis but remain subject to explosive rage (Ban, 1969).

Two careful studies have shown haloperidol to be effective in the treatment of children suffering from the hyperactive aggressive syndrome (Barker and Frazier, 1968; Cunningham, Pillai, and Rogers, 1968). It has also proved useful in adults suffering from the organic brain syndrome, but effective doses sometimes induce undue sedation.

The association between explosive rage and predatory aggression on the one hand, and "maleness" on the other, had led to an extensive search for androgen antagonists (Moyer, 1971). Stilbesterol has been used successfully, but its side-effects are unpopular.

In women whose attacks of explosive rage occur predominantly in the premenstrual week, small doses of meprobamate can be used to advantage. Progesterones may or may not help. Diuretics make the patient feel more comfortable in a physical sense, but do not seem to alter the rage threshold.

Propranolol, an adrenergic receptor blocking agent which in mice and rats has anti-convulsant properties and which also reduces the fighting behavior produced in rats by septal lesions (Greenblatt and Shader, 1972; Murman, Almirante and Saccani-Guelfi, 1966), has proved effective in abolishing the belligerence of patients who are emerging from coma induced by head injury or stroke, in abolishing the irritability of the post-concussional syndrome, and in preventing the less common attacks of explosive rage which may persist as a chronic relic of brain injury (Elliott, 1977). I have also found it useful, in conjunction with anti-convulsants, in preventing aggressive behavior in some individuals with psychomotor epilepsy. The value of the drug is enhanced by the fact that it does not sedate the patient in a general sense. Its central psychotropic effect appears to have nothing to do with adrenergic blocking properties, because the dextro isomer of propranolol blocks aggressive behavior in animals, though it has litttle beta blocking capacity (Bainbridge and Greenwood, 1966).

The treatment of hypoglycemias depends on the underlying cause. When rage attacks occur as a result of functional hypoglycemia, it is best treated with frequent high protein meals, reduction of carbohydrate intake, and the regular administration of Dilantin. Phenformin hydrochloride is also advised by some authorities to inhibit the secretion of insulin.

Psychosurgical treatment should be limited to severe cases of dyscontrol which have

not responded satisfactorily to conservative treatment, and they should only be carried out after careful psychological and physiological studies. The procedures include unilateral temporal lobectomy, bilateral anterior temperal lobotomy, unilateral or bilateral stereotaxic amygdalotomy, stereotaxic posteromedial hypothalamotomy, anterior and posterior cingulotomy, anterior thalamotomy, and orbito-frontal tractotomy. These techniques, and their results, are discussed in two books (Hitchcock, Laitinen, and Vaernet, 1972; Fields and Sweet, 1975). The results vary from good to indifferent, but the catastrophic disasters which sometimes resulted from frontal lobotomy are avoided by these selective procedures, and it is to be hoped that the search for still greater refinements will be pursued despite the recent irrational, emotional and ill-informed public opposition which brought such research virtually to a standstill in the United States in the early 1970's.*

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