sleep was usually light and not disturbed by these jerks. Except for migraine, he was in excellent health. Examination findings were normal. An EEG and CT scan were normal.

After he took 1 mg of clonazepam at night, the sleep myoclonus completely disappeared, and he was able to enjoy uninterrupted sleep. An all-night polygraphic recording done while he was taking clonazepam and symptom-free showed no myoclonic activity. His sleep pattern was normal. After taking clonazepam for six months, he noticed a few myoclonic jerks in the early morning hours. The dosage was not increased. After ten months of treatment, he was feeling somewhat depressed and the drug therapy was discontinued. This resulted in the return of the myoclonic activity.

COMMENT

Myoclonic activity during early stages of sleep is a seemingly normal phenomenon in many persons. It usually occurs when dropping off to sleep, often involves the distal extremities, and is unassociated with EEG changes.[3] Sometimes the activity occurs as part of an arousal response and may be associated with a K complex in the EEG.[4] These movements are most evident in stage I sleep and rapidly reduce in frequency as a person attains deeper stages.

Symonds[1] in 1953 described five patients with excessive amounts of myoclonic activity during sleep. Three complained of disturbed sleep. He suggested that these myoclonic movements were different from the common "nocturnal jerks" and coined the term "nocturnal myoclonus." He suggested that this was in some way related to epilepsy. Others, however, did not find any evidence favoring this relationship.[5] Detailed polygraphic study of 16 patients with this disorder was reported by Guilleminault et al in 1975.[2] These authors found "repetitive myoclonic movements of the lower extremities" that consisted of a "rapid partial flexion of the knee and hip lasting for several seconds." Each myoclonic period usually lasted for 10 to 30 minutes, occurred several times during the night, often awakened the patient, and was primarily observed during the slow-wave (III and IV) of sleep. In this respect, they were different from the usual myoclonus in stage I sleep. Treatment with a combination of hydroxytryptophan and carbidopa was not effective.

It is unlikely that sedation caused by clonazepam was responsible for its beneficial effect in our patients, as no other sedative hypnotics had been helpful. Our patients required only very small doses of the medication to control their symptoms. The effect appears to be very specific; the nocturnal myoclonus could not be observed in the second patient during polygraphic recording while he was taking the drug even though he had a classical history before treatment.

Nonproprietary Names and Trademarks of Drugs

Carbamazepine—*Tegretol.*
Carbidopa—*Lodosyn.*
Clonazepam—*Clonopin.*

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Violent Automatism in a Partial Complex Seizure

Report of a Case

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• We describe a patient who had a violent automatism that occurred during a partial complex seizure. The initial spike wave activity was recorded from nasopharyngeal leads that were lost as the patient began his vigorous, violent activity. This activity included nondirected, automatic, stereotyped behavior with physical assaults on objects in his path.
(*Arch Neurol* 37:120-122, 1980)

We describe the nature and extent of a violent automatism as a manifestation of a partial complex seizure (PCS). The term "partial complex seizure" is the international classification of "psychomotor seizure" or "temporal lobe seizure."[1] The term "violent" implies the use of extreme physical force often accompanied by destructiveness or intense vehement feelings, but not necessarily suggesting a specific directedness or aggressiveness (*Webster's Dictionary*).

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REPORT OF A CASE

A 29-year-old, right-handed man had a history of psychomotor seizures since age 9. The patient was born of a normal pregnancy, labor, and delivery, and developed normally until age 13 months, when he had meningitis. The father was alcoholic and physically abusive. Teachers reported hyperactivity, short attention, and difficulty with peer relations when the patient was 7. The parents were divorced at this time. The first seizure occurred when the patient was 9 years old. He was found staring and unresponsive and then ran outside, but could not explain his behavior afterwards. Because the patient struck a hospital staff member during a seizure, he was trans-

ferred to a psychiatric hospital (1956 to 1961), where he stayed until age 14. His mother said that she could not manage him because of a "constant bad temper" and that he would "rip up the house" during his seizures. After two months, the patient was hospitalized again (1962 to 1972). Records show normal general and neurologic examination results. The patient was described as having "paranoid trends, tangential thinking and unpredictable behavior." The records further noted that the patient became angry easily and attacked patients and hospital employees on several occasions, but whether this happened during seizures is not known. Seizures were controlled with phenobarbital, primidone, phenytoin sodium, thioridazine, and he was discharged at age 25. The patient recalled numerous difficulties with the hospital staff and related that they would beat him as well as other patients. Since discharge, he has had several small jobs and is living alone.

The patient's seizures have tended to occur in clusters, eight to ten at a time, every one to two months. The seizures are rarely related to any specific events, ie, sleep loss, alcohol use, or intense emotion. The patient describes an aura of a warm feeling in the abdomen, rising to the left side of the chest and occasionally associated with headache. He feels that by concentrating and shaking his head he may abort the seizure. He has no memory of the seizures, which have lasted no longer than a few minutes. The patient reports anecdotally that during seizures he has "destroyed" an x-ray room, thrown a bowling ball at another person, and assaulted a passenger on a bus. The patient's mother reports that he suddenly screams, unprovoked, and becomes destructive, upsetting furniture and pushing things off tables. The patient has attacked persons, but to the best of our knowledge, this has occurred only in association with attempts to constrain him during his violent activity. After a seizure, he is confused and usually sleeps, but shows concern as to whether anyone was hurt. In spite of the patient's interictal irritability, there has been no history of violent physical behavior except during seizures.

Six stereotyped seizures were videotaped during hospitalizations. The patient would scream loudly, then become quiet for a few seconds—manifesting a frightened appearance—scream again, and then become active. On those occasions when soft restraints were used during EEG recording, he would flail with his hands over his head as if pushing something away in a frightened manner (thus dislodging the electrodes). Then he would strike the side rail of the bed repeatedly. When unrestrained in his hospital room, he would jump up and run around the room. On one occasion, he grabbed the drapes next to his bed and kicked out from the wall on them. On another occasion he pulled the nurse's call button out of the wall, ran into the bathroom and swung it around his head so it was crashing against the walls. In all observations, he was locked by himself in a closed room, having no opportunity to direct aggression against people, but he would throw and tip over objects. Between one and two minutes after seizure onset, he would suddenly cease his violent behavior and appear quite confused, gradually regaining awareness.

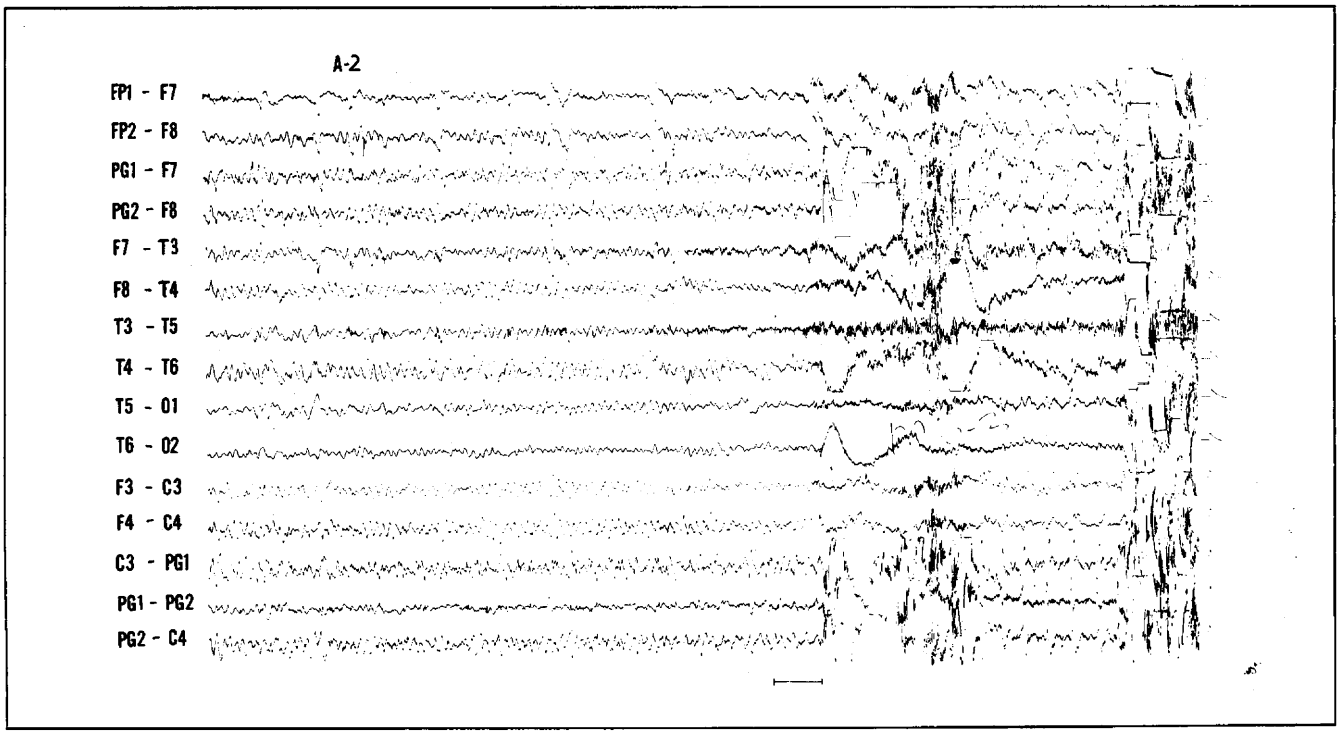
Findings on general and neurologic examinations, skull roentgenograms, and computerized tomography were within normal limits. Neuropsychological test results are reported elsewhere.[2]

Surface EEG recording demonstrated mild transient abnormal diffuse 4-7 CPS activity during the waking state. No spike and wave discharges were present during the awake or sleep record with surface recording alone. With nasopharyngeal recordings, there were fairly frequent interictal spike and wave discharges from the left nasopharyngeal lead (medial temporal region) (Figure). An EEG recording during the clinical seizure demonstrated an increased frequency of left nasopharyngeal spike and wave discharges starting approximately 20 to 30 s before his first scream. Then there would be repetitive 5 to 7 cycles per s sharp wave and low-voltage 20 to 30 cycles per s from the left nasopharyngeal area. By this time, muscle and movement artifact would obscure the record and the patient would pull out the electrodes (Figure).

Subsequent to the study of the patient's seizure disorder, the patient was stabilized with carbamazepine therapy and was seizure-free for six months. During a single seizure at that time, the patient partially disrobed in a public place, but there was no report of violent behavior.

COMMENT

There is controversy in analysis of aggression or violence appearing in patients with PCS.[3] Clarification of the issue has been attempted by



Initially, interictal spike waves are demonstrated at PG1. At onset of seizure, spike discharges become repetitive, phase-reversing at PG1. Artifact is seen because of patient's scream and gross movement.

distinguishing the ictal (or peri-ictal) from the interictal clinical manifestations of PCS.[4,5] Among those authors focusing on interictal behavior, there is a spectrum of opinions regarding the occurrence of violence in seizure patients.[5-14]

There also are conflicting reports regarding the occurrence and nature of violence during an ictus. After photographing the seizures of 150 patients, Rodin[8] noted "42 had ictal psychomotor automatisms and 15 had postictal psychomotor attacks. There was no instance of ictal or postical aggression." Knox[15] found only one case in 434 unselected cases of epilepsy that had peri-ictal aggressiveness. Mirsky and Harman[16] considered ictal rage "either a curiosity or a nonexistent phenomenon." Malamud[17] reported five cases of patients with violent behavior who had tumors of the limbic system. Gunn and Fenton[18] described two patients diagnosed as temporal lobe epilepsy who appeared to have violent behavior during a peri-ictal period. Currie et al[19] analyzed 666 cases of PCS and found 122 patients with emotional components to their seizures. Of these, "rage attacks occurred in 16 patients (five of these were adult), six had panic attacks, and five had violent outbursts." Of 199 patients with well-observed seizures (various types), King and Ajmone Marsan[20] reported nine with peri-ictal behavior that they could define as violent (ie, pushing, flailing, and throwing objects), but there was only one episode in which destruction of objects occurred during what appeared to be an automatic state. Of note, directed, complex, aggressive behavior has not been documented during an ictal event.

It is our opinion that the violent episodes described in this patient represent ictal events. The destructive behavior was self-limited, always one to two minutes in duration, stereotyped, and nondirected. During each seizure in which nasopharyngeal leads were used for EEG recording, the seizure was preceded by repetitive spike discharges from the left medial temporal region. Because the patient then pulled out the electrodes, we cannot be certain exactly how much of the behavior was ictal and how much was postictal. Most likely, the active phase with violent behavior is ictal, and the confused, more relaxed phase afterward is postictal.

The clarity of the "violent" nature of the behavior in this case must be stressed. The behavior was destructive, though automatic and stereotyped, in that the patient used his fists for pounding and his hands for pulling. Physical attack on objects was clearly manifest. Most importantly, the ictal behavior was not goal-oriented and thus, more primitive than could be considered socially aggressive.[21] This case represents the most violent degree of behavior documented in a patient during a PCS at the UCLA Neuropsychiatric Institute during the last 15 years of special attention to medically refractory seizures.

The question of why this patient is violent during ictal events should be raised. Hypothalamic isolation in cats,[22] experimentally induced temporal lobe epilepsy in rats,[23] and electrical brain stimulation in animals[24-27] and man[11,28] has caused aggressiveness and rage and attack behaviors; however, it has been suggested that stimulus-bound behavior is probably determined by personality[29] and modified by experience.[30-32] Epstein and Ervin[33] have suggested that the content of highly organized seizures can be understood as having psychodynamic meaning. This patient's ictal behavior could be seen as being determined by a violent social background including abusiveness by his father, his peer group, and the hospital attendants.

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Nonproprietary Names and Trademarks of Drugs

Carbamazepine—*Tegretol.*
Thioridazine—*Mellaril.*

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