Case Report

Hazard of Video Games in Patients With Light-Sensitive Epilepsy

Neil R. Dahlquist, MD; James F. Mellinger, MD; Donald W. Klass, MD

VIDEO games have become an epidemic form of amusement. Recently, two reports appeared from England of patients with epilepsy in whom playing video games precipitated seizures[1,2] (*Time*, July 19, 1982, p 67). To emphasize the particular hazard of video games for susceptible persons with light-sensitive epilepsy, we herein document the first record, to our knowledge, of a similar case in the United States.

Report of a Case

A 15-year-old boy played video games for one year before his first seizure. He had no medical problems until March 7, 1982. While the boy played an electronic game called "Combat," a friend noted that he acted as if he were in a daze and that his hand was twitching. When he returned home shortly thereafter, his mother noticed that he had lost his glasses, that he was "belligerent for the first time in his life," and that his mentation was slow. Later that month, his parents heard a crash while the boy was taking a shower, and they found him on the floor in an extremely agitated and belligerent state. They also noted that his right arm twitched briefly and, afterwards, that he was transiently confused.

On April 19, while playing "Pac-Man" with his father, the boy had a generalized tonic-clonic seizure that lasted several minutes. He was taken to a local hospital, where he was considered to be in a postictal state. This lasted about two hours; otherwise, results of neurological examination were normal. A computed tomographic scan with contrast medium was normal. An EEG obtained while the patient was awake was reported to show a burst of spike and wave activity during photic stimulation at a flash frequency of 15 Hz. While he slept, an EEG showed a solitary burst of diffuse spike and wave activity. Phenytoin sodium was prescribed in doses of 300 mg/day.

Later, the boy had three more generalized seizures, always associated with bright, early-morning sunlight. One seizure occurred while he was riding in a convertible down a tree-lined boulevard in flickering sunlight. The second seizure occurred while the patient was seated facing the early-morning sunlight streaming through a screened porch, and he had the third seizure while he was facing the sun behind a paned glass window.

The patient came to the Mayo Clinic on Aug 19. At that time, results of a neurological examination were normal. An EEG during wakefulness and during hyperventilation was normal; however, photic stimulation at 20 Hz consistently elicited diffuse bursts of high-amplitude, bilaterally synchronous spike and slow-wave complexes, which were sometimes accompanied by generalized myoclonus. The photoparoxysmal responses often persisted for 3 or 4 s after termination of the photic stimulus. During photic-induced paroxysms, the patient consistently failed to respond to auditory signals, and repetitive motor activity was interrupted (Figure). No paroxysmal discharges occurred during sleep, but frequent generalized spike and slow-wave complexes and myoclonus were recorded for a short time after arousal from sleep.

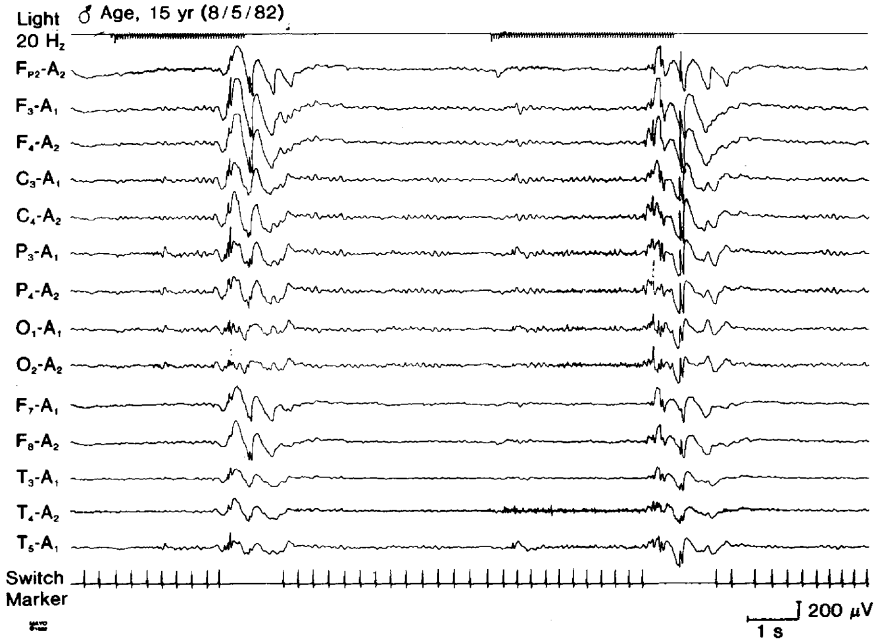
The next day, an EEG contained no paroxysmal abnormalities during photic stimulation, while the patient viewed several different geometric patterns, or while he played two games of "Pac-Man." The frequency of flickering of the "Pac-Man" images was measured at about 15 Hz.

The patient was advised to taper the dosage of phenytoin and to begin to take 250 mg of valproic acid four times daily. In the one month since that time, he has avoided playing electronic games and has had no further seizures.

Comment

Our patient is similar to the patient of Daneshmend and Campbell,[2] who was a teenager, had seizures provoked by playing a video game, and exhibited photoparoxysmal responses in the EEG at flash frequencies between 15 and 21 Hz. We consider seizures induced by playing video games similar to television-induced seizures, which have been well recognized in epileptic patients who are sensitive to flickering lights or geometric patterns. In some patients, susceptibility to seizures induced by watching television or playing video games may be influenced by associated nonvisual factors, such as hyperventilation and prior deprivation of sleep. Spontaneous fluctuations in threshold for photosensitivity are also well known.[3] Thus, it is not surprising that our patient was extremely photosensitive on the first day of testing but was not sensitive to flickering lights, geometric pattern, or the video game on the following day. Furthermore, time of day probably had an influence. On the second day, he was tested in the afternoon. His history suggested that

Electroencephalogram shows photoparoxysmal responses to light at flash frequency of 20 Hz and arrest of motor activity (marker in lowermost channel) during paroxysms.



From the Department of Neurology, Mayo Clinic and Mayo Foundation, Rochester, Minn.

Reprint requests to Mayo Clinic, Rochester, MN 55905 (Dr Klass).

he was more likely to have seizures in the morning, and the previous EEG contained spontaneous epileptiform abnormality only after the patient was aroused from sleep. Treatment with valproic acid was advised because the drug has been effective in patients with light-sensitive epilepsy.[4] Physicians should be aware, however, that playing video games may be hazardous for such patients.

References

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Brief Reports

Severe Metabolic Complications in a Cross-Country Runner With Sickle Cell Trait

Kathy J. Helzlsouer, MD; Frederick G. Hayden, MD; Alan D. Rogol, MD

• A 17-year-old black male with sickle cell trait collapsed after completion of a 5,000-m cross-country race. He manifested CNS dysfunction, disseminated intravascular coagulation, rhabdomyolysis, renal insufficiency, and deep venous thrombosis. Sickle cell crisis may have been etiologic in the pathogenesis of this constellation of abnormalities.

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THE prevalence of sickle cell trait among American blacks is approximately 7% to 9%. Although population studies have not demonstrated a difference in mortality rates when comparing adults with AS hemoglobin and a matched group with AA hemoglobin, certain abnormalities (including hematuria, bacteriuria, hyposthenuria, and splenic infarction during high-altitude exposure) occur with increased frequency in the population with sickle cell trait.[1]

This report describes severe CNS dysfunction, disseminated intravascular coagulation, rhabdomyolysis, and renal insufficiency that occurred after a cross-country race in a young endurance-trained athlete with sickle cell trait. The role of sickle cell trait and the mechanisms predisposing the patient to the development of a possible sickle cell crisis are discussed.

Report of a Case

A 17-year-old black male with sickle cell trait collapsed at the end of a 5,000-m cross-country race, after having placed 11th of more than 200 runners (time, 16 minutes 28 s). The elevation of the course was 145 m; the ambient temperature, 16 °C; and dew point, 5 °C. When initially evaluated by race physicians, he was unresponsive to deep pain and was apneic and bradycardic. He was resuscitated with mouth-to-mouth ventilation and transported to the University of Virginia Hospital, Charlottesville.

One year before admission, the patient had collapsed after another cross-country race. He manifested abdominal pain, nausea and vomiting, dyspnea, and leg muscle cramps and was admitted to another hospital. Laboratory data are presented in the Table. Hemoglobin electrophoresis disclosed Hb S values of 42% and Hb A of 58%. His condition improved with intravenous (IV) hydration and ampicillin trihydrate therapy and he was discharged after a one-week hospitalization. He was advised to discontinue cross-country racing. However, he continued to run competitively without symptoms during the ensuing year. On the evening before the present admission, he took a decongestant containing phenylpropanolamine hydrochloride and chlorpheniramine maleate because of symptoms of nasal congestion.

On admission to the University of Virginia Hospital, approximately 45 minutes after his collapse, he was awake but disoriented to place and time and amnestic for the events leading to his hospitalization. He complained of severe leg cramps. Examination disclosed the following values: BP, 160/90 mm Hg while supine and 138/80 mm Hg while sitting; pulse rate, 148 beats per minute and regular; respirations, 30/min; and rectal temperature, 36.7 °C. Pertinent physical findings included normal results of a lung examination, hypoactive bowel sounds with a nontender abdomen and no organomegaly, bloody stool on rectal examination, and diffuse muscle contractions in the lower extremities with marked tenderness to palpation. The admission laboratory data are presented in the Table. Analysis of urine obtained by catheterization disclosed the following values: specific gravity, 1.014 g/mL; pH, 9; proteinuria (4+); five to ten RBCs per high-power field (HPF) and one to five WBCs per HPF. Results of a qualitative test for myoglobin (toluidine blue O) was positive after precipitation of hemoglobin with 80% saturated ammonium sulfate.

Treatment initially consisted of hydration with bicarbonate-containing IV fluids. Within several hours of admission, hematemesis, bloody diarrhea, and hypoxemia developed (Table). The peripheral blood smear demonstrated fragmented and burr cells, and findings from laboratory studies were consistent with disseminated intravascular coagulation (DIC). A ventilation-perfusion lung scan did not show evidence of pulmonary emboli. The DIC and hypoxemia resolved within 48 hours without anticoagulant therapy. Rhabdomyolysis was evident on admission and was associated with renal insufficiency (Table). Asymptomatic hypocalcemia and hyperphosphatemia occurred within 24 hours of admission. The serum creatinine concentration peaked on the third day, with a decrease in creatinine clearance to 19 mL/min. Renal function gradually improved and asymptomatic hypercalcemia occurred with persistence of the hyperphosphatemia. On the sixth hospital day, deep venous thrombosis in the left leg was documented by venogram and treated with IV heparin. The month-long hospitalization was further complicated by a sensorimotor neuropathy resulting from compression of the left peroneal and tibial nerves.

At discharge, the patient continued to show mild renal insufficiency with substantial potassium and magnesium wasting, requiring oral supplementation; mild elevation of the serum transaminase concentrations; and the persistent sensorimotor neuropathy. He was given treatment

From the Departments of Internal Medicine (Drs Helzlsouer and Hayden), Pathology (Dr Hayden), Pediatrics (Dr Rogol), and Pharmacology (Dr Rogol), the University of Virginia School of Medicine, Charlottesville.

Reprint requests to Department of Pediatrics, Box 386, University of Virginia Hospital, Charlottesville, VA 22908 (Dr Rogol).