Video Game-Related Seizures: A Report on 10 Patients and a Review of the Literature

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ABSTRACT. *Objective.* **To further describe the features, postulated pathophysiology, treatment, and outcome of seizures occurring while playing or watching video games (video game-related seizures (VGRS)).**

Design. **We evaluated retrospectively 10 patients with VGRS seen by us and reviewed 25 reported cases.**

Results. **The 35 patients ranged in age from 1 to 36 years (mean: 13.2); and 26 subjects (74%) were male. Eight individuals (29%) had prior infrequent nonfebrile seizures, 4 (11%) had febrile convulsions, and 2 (6%) had a family history of epilepsy. VGRS consisted of generalized tonic-clonic seizures in 22 of 35 individuals (63%); absences in 2 (6%); simple partial seizures in 6 (19%); complex partial seizures in 4 (11%); and other manifestations in 4. Neurologic examination and computed tomographic and magnetic resonance imaging scans were normal. Electroencephalograms demonstrated generalized or focal, interictal or ictal epileptic patterns in 11 of 21 patients (52%) and photoparoxysmal responses in 17 of 32 (53%). Eleven of 15 individuals (73%) treated with video game (VG) abstinence alone, 3 of 6 who received anticonvulsants but played VGs, and 7 of 12 treated with combined VG abstinence and anticonvulsants had no further seizures.**

Conclusions. **We postulate that a special convulsive susceptibility of selected neurons in striate, peristriate, infratemporal, and posterior parietal cortices to particular visual stimuli plays a major role in VGRS. VG abstinence is the treatment of choice of VGRS. Anticonvulsant medication is suggested only for those individuals who continue to play VGs or suffer from seizures triggered by other, unavoidable visual stimuli, or from unprovoked attacks. *Pediatrics* 1994;93:551–556; *stimulus-sensitive epilepsy, reflex epilepsy, video games, video game-related seizures, photoparoxysmal response.***

ABBREVIATIONS. VG, video game; VGRS, video game-related seizure; EEG, electroencephalogram; TV, television.

It has long been recognized that in certain individuals, including children and adolescents, epileptic seizures can be precipitated by a wide variety of stimuli,[1,2] and that visual excitations are the most common triggers of these "stimulus-sensitive" or "reflex" attacks.[1–4] The literature available to us at the time of this writing includes 25 cases of seizures reported since 1981 in individuals intent at playing video games (VGs)[5–18] or "video game-related seizures" (VGRS). Ten patients with this disorder were seen during a 3-year period in a child neurology clinic at Children's Hospital and Medical Center (W.D.G.) and two child neurology practices (S.T.G. and T.A.K.) in Seattle, WA. This experience suggests that VGRS are more frequent than commonly recognized. Because recent developments in VG technology encourage increasing numbers of persons, especially children and adolescents, to engage in these games, VGRS are a cause of growing concern both in the United States and abroad. Our work describes the features of this disorder in the patients studied by us and reviews the earlier reports by others with the hope of increasing physicians' awareness of VGRS and of their treatment.

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METHODS

Our 10 patients with VGRS, designated group I, were examined between June 1990 and July 1993. Length of follow-up ranged from 4 to 36 months. Information obtained retrospectively from clinical records and interviews included: the patient's age; gender; personal and family history; clinical pattern and frequency of seizures associated with VGs or occurring independently of them; type of VG and hardware system used; results of neurologic examination, electroencephalogram (EEG) and imaging studies; and treatment and clinical outcome. Seizures were classified as proposed by the Commission on Classification and Terminology of the International League Against Epilepsy.[19] EEGs obtained in group I patients consisted of 16-channel recordings taken during wakefulness, hyperventilation, and stroboscopic stimulation at rates of 1 to 30 Hz[20] but not during the actual playing of VGs. Similar information was derived from published reports describing VGRS in 25 patients to be referred to as group II. Results from groups I and II were analyzed both separately and jointly.

RESULTS AND LITERATURE REVIEW

Salient features of the 10 patients in group I and of the 25 cases in group II are summarized in Tables 1 and 2 and numbered 1 through 10 and 11 through 35, respectively.

Age and Gender

The ages of the patients at the time of their first VGRS ranged between 1 and 22 years (mean, 12.4; median, 13) in group I and between 4 and 36 years (mean, 13.6; median, 13) in group II. Group I included 7 males and 3 females, and group II consisted of 19 males and 6 females. There was no significant difference between the two groups in regard to age or gen-

TABLE 1. Characteristics of 10 Patients With VG-Related Seizures (Group I)*

Patient No.	Age, y/ Gender	Previous Seizures	VG Seizure Type	VG Played (VG Hardware System)	EEG PhP Responses (Flash Rate-Hz)	Treatment	Clinical Outcome
1	14/M	Yes	SP→GTC	NI (ARC)	Yes (9–30)	Abstinence	Unprovoked seizures
2	11/M	No	GTC	"Mario Bros 3" (HVC)	Yes (10–30)	Abstinence	Unprovoked seizures
3	15/F	Yes	A	NI (HVC)	Yes (10–20)	ETH and abstinence	Unprovoked seizures
4	22/M	Yes	SP→GTC	NI (HVC)	No	PB and abstinence	Seizure-free
5	15/F	No	GTC	"Mario" (HVC)	No	PHT and abstinence	Seizure-free
6	10/M	Yes	GTC	NI (HVC)	No	VPA and abstinence	TV seizures
7	14/M	No	GTC	NI (HVC)	Yes (15–29)	PHT and abstinence	Unprovoked seizures and board game seizures
8	01/F	Yes	GTC	NI (HVC)	No	VPA and avoidance	Stroboscopic seizures
9	12/M	Yes	GTC and CE	"Double Dragon" (HVC)	No	CBZ and abstinence	Unprovoked seizures
10	10/M	No	A	NI (HVC)	No	ETH and abstinence	Seizure-free

* Abbreviations: VG, video game; PhP, photoparoxysmal; Hz, Hertz; SP, simple partial; GTC, generalized tonic-clonic seizure; NI, not indicated; ARC, arcade; HVC, home video console; A, absence; ETH, ethosuximide; PB, phenobarbital; PHT, phenytoin; VPA, valproic acid; TV, television; CE, confusional episodes, unclassified; CBZ, carbamazepine.

der (Fisher's exact test $P = .5$). These groups jointly comprised 35 individuals aged 1 to 36 years (mean, 13.2; median, 13). Twenty-six (74%) were male and 9 (26%) were female.

Relevant Personal and Family History

Before their first VGRS, 6 of 10 patients in group I (Table 1) and 2 of 25 in group II (Table 2) had suffered one or a few seizures, exclusive of febrile convulsions, and three were taking anticonvulsant medications. We categorized these earlier attacks as: (1) primary generalized tonic-clonic in two individuals in group I (patients 4 and 8) and one in group II (case 17); (2) myoclonic jerks of the extremities in one person in group II (case 34); (3) absences in one patient in group I (patient 3); (4) simple partial seizures (ie, focal seizures with preserved consciousness) in three patients in group I. These consisted of clonic jerking of the right hand (patient 1); adversion to the left with subsequent tonic-clonic generalization (patient 6); and transient visual loss (patient 9); and (5) confusional episodes of uncertain classification (patient 9).

Risk factors for epilepsy included febrile convulsions in one individual in group I (patient 8) and in three in group II (cases 11, 25, and 35); a history of possible viral encephalitis in a person in group I (patient 9); and a family history of epilepsy in one subject in each group (patients 8 and 30).

The occurrence of one or more seizures other than febrile convulsions before the development of VGRS was significantly greater among persons in group I than in group II (Fisher's exact test $P = .02$). Pooling the data from both groups, 8 (23%) of 35 individuals had infrequent afebrile and 4 (11%) had experienced febrile seizures before the first occurrence of their VGRS.

Clinical Pattern of VGRS

The clinical features of the VGRS suffered by 35 patients in groups I (Table 1) and II (Table 2) were: (1) primary generalized tonic-clonic seizures in six individuals in group I (patients 2 and 5 through 9); and 16 in group II (cases 12 through 17, 19, 21, 24, 26 through 28, and 32 through 35); (2) absences in two subjects in group I (patients 3 and 10); (3) simple partial seizures in two persons in group I and four in group II. These consisted of blurring of vision followed by dizziness, headache, and jerking of the right hand (patient 1); multiple scotomata (patient 4); central visual loss (case 18); scintillating scotomata (case 20); "binasal" visual field defects (case 29); and clonic jerking and abduction of the right arm (case 30). Secondary tonic-clonic generalization occurred in five of these individuals (patients 1 and 4 and cases 18, 20, and 30); (4) complex partial seizures (ie, focal seizures with loss of consciousness) in four group II patients. These consisted of déjà vu and intense memory recall (case 11), scotomata followed by visual loss and impairment of consciousness (case 22), and vertiginous sensation (case 31). Secondary tonic-clonic generalization occurred in two of these individuals (cases 11 and 31); (5) confusional episodes of uncertain classification in one subject in group I (patient 9) and two in group II (cases 13 and 15); (6) attacks dominated by intense pounding headaches in one individual in group II (case 23).

When the data from the two groups were merged, VG-related primary generalized tonic-clonic seizures occurred in 22 (63%) of 35 individuals, absences in two (6%), simple partial seizures in six (17%), complex partial seizures in four (11%), and pounding headache in one (3%).

Remarkably, three individuals (patients 7, 8, and 23) suffered VGRS while not personally engaged in playing a VG but watching a game played by another person.

Neurologic Examination

The results of the neurologic examination were normal in all patients in groups I and II.

EEG Findings at Rest and During Hyperventilation

The EEGs recorded at rest and during hyperventilation were essentially normal for age in 4 of 10 patients in group I (patients 4, 5, 8, and 10). Of the remaining six individuals, five demonstrated at rest,

TABLE 2. VG-Related Seizures Reported Between 1981 and 1993 (Group II)*

Case No.	Author	Age, y/ Gender	Previous Seizures	VG Seizure Type	VG Played (VG Hardware System)	EEG PhP Responses (Flash Rate-Hz)	Treatment	Clinical Outcome
11	Rushton[5]	17/M	No	CP→GTC	"Astro Fighter" (ARC)	No	Abstinence	Seizure-free
12	Jeavons et al[6]	14/M	No	GTC	"Space Invaders" (HH)	Yes (15 and 18)	VPA	NI
13	Daneshmend and Campbell[7]	17/F	No	GTC and CE	"Dark Warrior" (ARC)	Yes (15–21)	Abstinence	Seizure-free
14	Ferry et al[8]	12/M	NI	GTC	"Chopper"	NI	NI	NI
15	Dahlquist et al[9]	15/M	No	GTC and CE	"Combat" and "Pac Man" (NI)	Yes (15 and 20)	1. PHT	1. Unprovoked and visual elicited szs
							2. VPA and Abstinence	2. Seizure-free
16	Helfgott and Meister[10]	8/M	No	GTC	NI (ARC)	NI	PB	NI
17	Glista et al[11]	14/M	Yes	GTC	Intellivision system (NI)	No	1. PB alone	1. VG seizure
							2. PB and Abstinence	2. Seizure-free
18	Gligta et al[11]	15/M	No	SP→GTC	"Turbo" (ARC)	No	Abstinence	Seizure-free
19	DeMarco and Ghersini[12]	8/M	No	GTC	"Invader" (NI)	Yes (20 and 22)	Abstinence	Seizure-free
20	DeMarco and Ghersini[12]	13/M	No	SP→GTC	NI (NI)	No	CBZ	VG seizure
21	Hart[13]	13/F	No	GTC	"Super Mario Brothers" (HVC)	Yes (8–16)	NI	NI
22	Maeda et al[14]	7/M	No	CP	"Super Mario Brothers" (HVC)	Yes (3–30)	VPA	Seizure-free
23	Maeda et al[14]	10/F	No	PH	"Link's Adventure Quest" (NI)	Yes (12–30)	VPA	Seizure-free
24	Maeda et al[14]	12/F	No	GTC	"Dream Factory" (NI)	No	Abstinence	Seizure-free
25	Maeda et al[14]	4/M	No	CP	"Wrecking Crew" (NI)	No	Abstinence	Seizure-free
26	Maeda et al[14]	12/M	No	GTC	"Link's Adventure Quest" (NI)	Yes (12–30)	Abstinence	Seizure-free
27	Maeda et al[14]	13/M	No	GTC	"Hydlide" (NI)	Yes (3–30)	1. Abstinence	1. Unprovoked seizures
							2. VPA	2. Seizure-free
28	Maeda et al[14]	9/M	No	GTC	"Dragon Quest 2" (NI)	Yes (3–30)	1. Abstinence	1. Unprovoked seizures
							2. VPA	2. Seizure-free
29	Fukusako et al[15]	12/M	No	SP	NI	NI	NI	NI
30	Vespignani et al[16]	19/M	No	SP→GTC	NI (NI)	No	Abstinence	Seizure-free
31	Vespignani et al[16]	36/F	No	CP→GTC	NI (NI)	No	Reduced VG	Seizure-free
32	Vespignani et al[16]	19/M	No	GTC	NI (NI)	Yes (13–15)	Abstinence	Seizure-free
33	Giroud et al[17]	11/M	No	GTC	NI (NI)	Yes (15)	NI	NI
34	Giroud et al[17]	14/F	Yes	GTC	NI (NI)	Yes (12)	NI	NI
35	Thompson[18]	15/M	No	GTC	NI (ARC)	No	Abstinence	Seizure-free

* Abbreviations: VG, video game; PhP, photoparoxysmal; Hz, Hertz; NI, not indicated; CP, complex partial; GTC, generalized tonic-clonic; ARC, arcade; HH, hand-held; VPA, valproic acid; CE, confusional episodes, unclassified; PHT, phenytoin; PB, phenobarbital; CBZ, carbamazepine; HVC, home video console; PH, "pounding headache"; szs, seizures.

during hyperventilation, or both, 3- to 4-Hz spike-and-slow-waves which occurred bilaterally synchronously over all head regions. These paroxysms, which lasted 1 to 12 seconds, showed no noticeable asymmetry in three subjects (patients 2, 7, and 9). By contrast, they displayed varying degrees of right-sided preponderance in one person (patient 1), and were sometimes preceded and followed by prolonged 1-Hz spike-and-slow-waves over the left frontocentral regions in another (patient 2). In this last individual, spike-and-slow-waves were detected in EEGs recorded both before and after the occurrence of VGRS. Clinical manifestations of absence were apparent during the most prolonged paroxysms only in one person (patient 2). The last individual in this group (patient 6) displayed at rest brief bursts of interictal multiple spikes as well as a clinically inapparent electrical seizure confined to the right occipital and posterior temporal areas.

The features of the EEGs recorded at rest and during hyperventilation were described in 11 patients in group II. Findings were said to include: normal patterns in six individuals (cases 15, 18, 30, 31, 33, and 35); occipital slow waves in two (cases 22 and 23); bursts of generalized spike-and-slow-waves in two (cases 15 and 17); left frontal as well as generalized spike-and-slow-waves in one (case 12); left focal multiple spike-and-slow-waves in unspecified location in one (case 16); and unilateral occipital spikes in two (cases 20 and 29).

Pooling data from both groups, interictal or ictal patterns were detected at rest or during overbreathing in the EEGs of 11 of 21 patients (52%) in whom adequate information was available.

EEG Findings During Stroboscopic, VG, and Other Stimulations

In 4 of 10 subjects in group I (patients 1 through 3 and 7), stroboscopic stimulation at 9 to 30 Hz elicited bilateral synchronous, generalized spike-and-slow-waves and multiple spike-and-slow-waves lasting from 1 to 10 seconds. Clinical changes associated with these evoked paroxysms in three of these individuals consisted of a myoclonic absence (patient 2), a sensation of dizziness (patient 1), and a feeling "like being hit by a bolt of lightening" (patient 7). Similar EEG photoparoxysmal responses were reported in 13 of 22 individuals in group II in whom the results of this test were described (cases 12, 13, 15, 19, 21 through 23, 26 through 28, and 32 through 34). However the clinical events associated with their aberrant responses to stroboscopic stimulation were not specified. It should be noted that EEG paroxysmal responses were not detected consistently in successive EEGs in patient 3 of group I and patients 15, 22, 23, 26, 27, and 32 through 34 of group II. The frequency of these responses was not significantly different in groups I and II (Fisher's exact test $P = 0.3$). When both groups were combined, stroboscopically elicited paroxysms occurred in at least one EEG of 17 of 32 patients with VGRS (53%).

In few patients in group II, the EEG effects of VG playing and stroboscopic stimulation were compared with those of eye opening,[12] presentation of geometric[9] or specifically checkerboard[6,14,15] patterns, and reading.[6] Paroxysmal responses were elicited by VG playing alone in two patients (cases 24 and 25) and by both VG playing and stroboscopic stimulation, but not by other visual stimuli in one (case 12). It was of further interest that interictal unilateral occipital spikes were activated by checkerboard stimulation in one patient (case 29) and blocked by eye opening in another (case 20).

Neuroradiologic Studies

Cranial computed tomographic or magnetic resonance imaging scans performed on seven patients in group I (patients 1, 4 through 7, 9, 10) and six in group II (cases 15, 17, 18, 22, 29, and 30) were normal in all individuals, except one (patient 7) who demonstrated a left temporal subarachnoid cyst.

Treatment and Clinical Outcome

Information was available on the treatment received in 27 of the 35 patients with VGRS. Two individuals in group I (Table 1) and 13 in group II (Table 2) were instructed to abstain from playing or watching others play VGs. This measure rendered 11 persons in group II (cases 11, 13, 18, 19, 24 through 26, 30 through 32, and 35) free of seizures. By contrast, two individuals in each group experienced recurrence of seizures. These consisted of an unprovoked generalized tonic-clonic attack (patients 1, 27, 28) and unprovoked or stroboscopically elicited absences (patient 2).

An anticonvulsant medication (valproic acid, carbamazepine, phenobarbital, or phenytoin) was given to five patients in group II only who apparently continued playing VGs. Two of these individuals (cases 22 and 23) had no further seizures, whereas the remaining three suffered one or more additional attacks, including generalized tonic-clonic seizures unprovoked or triggered by strong light stimuli (case 15) or elicited by VGs (case 17) and episodes characterized by scintillating scotomata followed by tonic-clonic generalization (patient 20).

In all eight patients in group I and four in group II who were given anticonvulsant medication, abstinence or avoidance of VGs was recommended. Combined anticonvulsant treatment and abstinence from VGs rendered free of seizures all patients in group II (cases 15, 17, 27, and 28) and three individuals in group I (patients 4, 5, and 10). The remaining five subjects in the latter group had recurrence of seizures. These consisted of: an unprovoked generalized tonic-clonic attack (patients 7 and 9); absences (patient 3); a television (TV)-elicited adversive seizure (patient 6); and a generalized tonic-clonic seizure triggered by light flashes during a performance staged in a family restaurant (patient 8). However, this last patient, an infant, experienced no further attacks after her parents took care to avoid exposing her to offending visual stimuli.

Statistically significant differences between groups I and II with regard to the effects of treatment could not be demonstrated because of the small number of cases available for analysis. By pooling data from both groups, it became apparent that freedom from seizures was achieved by: simple abstinence in 11 of 15 patients (73%); the administration of an anticonvulsant medication with continued VG playing in 3 of 6 individuals; and combined anticonvulsant and VG abstinence in 7 of 12 subjects (58%).

DISCUSSION

Our findings in 10 patients in group I were mostly consistent with those obtained by previous authors on 25 individuals in group II.[5–18] Thus, unless otherwise indicated, this discussion will refer mostly to the information provided jointly by all 35 patients.

Prevalence of VGRS

The prevalence of VG-related seizures is unknown. Playing VGs is very common, especially among children and adolescents in the United States where VG systems exist in most households (G. Gabelhouse, Fairfield Research, Inc, Lincoln, NE, personal communication, December 1, 1992).[21] Yet, the number of reported cases of seizures associated with this novel entertainment is very small so far.[5–18] These considerations would indicate that VGRS are infrequent. However, the finding of as many as 10 patients with this disorder in our pediatric population during a 3-year period suggests that VGRS may be more common than generally recognized. A large case-control design study would be required to determine the exact incidence of this disorder and to estimate chance association. However, that a relationship other than chance existed between VGs

and seizures in the 35 individuals under scrutiny was strongly suggested by several observations. These included: (1) the occurrence of one or more VGRS in previously seizure-free subjects; (2) the detection of EEG epileptiform discharges in some patients only when intent at playing a particular VG or even in coincidence with a given specific sequence within this game[14]; (3) the occasional demonstration of the exclusive epileptogenicity of VGs compared with other visual stimuli[14]; (4) the subsidence of seizures in a large proportion of subjects after VG abstinence alone.[5,7,11,12,14,16,18]

Relations to Age and Gender

At the onset of VGRS, the ages of the 35 patients in groups I and II ranged from 1 to 36 years with a mean of 13.2. Twenty-six of them (74%) were male. Both this age distribution and gender disproportion are likely related to VG player demographics because most players are males and between the ages of 5 and 24 years, with a mean age of 13.5 years (G. Gabelhouse, personal communication, December 1, 1992).[21] It should be noted that the male preponderance among individuals with VGRS contrasts with the female predominance among individuals with epileptic seizures triggered by stroboscopic stimuli.[4,22]

Other Seizures in Patients With VGRS

VG-unrelated epileptic manifestations, whether unprovoked or precipitated by instrumentally generated or naturally occurring repetitive light flashes, viewing normally or abnormally functioning TV screens, other visual stimuli such as those associated with playing board games, and hyperventilating, occurred in 9 of 35 patients (26%) before and in 12 individuals (34%) after the first occurrence of VGRS. In addition, photoparoxysmal responses with or without associated clinical changes were detected in the EEGs of 17 of 32 subjects (53%) in whom adequate information was available. These findings indicate that a substantial proportion of patients under consideration suffer from a seizure disorder antedating the manifestation of VGRS, an excessive sensitivity to certain particular visual stimuli, or both. Hence, it seems likely that playing or watching VGs does not cause normal persons to develop a seizure disorder, but only unmasks a peculiar proclivity of the brains of certain individuals to respond with an epileptic paroxysm to certain ordinarily inoffensive visual excitations.

Visual Mechanisms Underlying VGRS

The mechanisms underlying the precipitation of seizures during VGs are not clearly known. The study of our patients and a review of the literature suggest that in most cases attacks tend to occur primarily, if not exclusively, during certain particular VG sequences. These include one of the following: repetitive, high-intensity, multicolored or white flashes[5,7,8,11,14,16]; rapid changes of scene in the VG game[14]; swift displacements of images across the screen[16]; appearance of line patterns[4]; and rolling or flickering TV patterns.[10,13] The sensitivity of some individuals with VGRS to these particular visual excitations is so extreme that even a few light flashes or patterns displayed on a low-luminance, low contrast hand-held screen may have epileptogenic effects.[6] There is imposing experimental evidence, aptly summarized by Regan[23] that the features of visual stimuli most likely to trigger VGRS are processed in cortical areas that include occipital striate and peristriate as well as infratemporal and posterior parietal regions. Hence, it seems likely that a special convulsive susceptibility to certain particular visual stimuli of selected neuronal organizations within these cortices plays a major role in VGRS. A similar aberrant mechanism has been proposed for the seizures specifically elicited by the viewing of line patterns.[24] Observations adding substance to this conjecture include: (1) the finding that as many as 7 of 35 patients with VGRS (20%) suffered from simple partial visual seizures, (2) the detection in the EEGs of three additional individuals of interictal epileptiform discharges confined to the occipital and posterior temporal areas of one side, and (3) the occurrence in one of these last subjects of a similarly localized EEG seizure pattern. Ictal discharges arising from infratemporal or posterior parietal cortices and spreading to other cortical regions presumably account for the features of other partial, including complex partial seizures, demonstrated by four patients (11%). Under appropriate conditions of stimulation, the paroxysmal activity presumably arising in cortical areas processing visual information becomes generalized. The pathways of spread of the discharge in these circumstances are unknown but may be analogous to those described in the cat[25] for the irradiation of photic impulses under the effects of pentylenetetrazol. The marked sensitivity of a substantial proportion of our patients to stroboscopic stimulation contributes to the credibility of this analogy.

Our observations and a review of the literature suggest that the VGRS displayed by individual subjects are epileptic events preferentially triggered by one of many types of visual stimuli generated during VGs. Most of these excitations, including repetitive light flashes, line patterns, and the visual effects of well functioning or malfunctioning TV screens, are already well known as triggers of epileptic seizures in photosensitive subjects. Only few stimuli, such as nongeometric images moving rapidly on a contrasting background, may be encountered most commonly during and delivered most effectively by VGs. There seems to be little ground for regarding VGRS either as an entirely novel stimulus-sensitive epileptic disorder or as a subtype of any of the other varieties of photosensitive epilepsy.

Factors Possibly Contributing to VGRS

Based on their clinical observations, some authors[16,17] expressed the opinion that looking at a close distance at the TV screen displaying VG images and placing the TV monitor in a dark environment favored the occurrence of VGRS. They further conjectured that increasing the player's distance from the monitor, illuminating the surroundings, and wearing tinted glasses would have opposite effects. Apart from the questionable practicality of these measures,

it seems likely that their effects will prove more complex and difficult to predict than it has been anticipated. Experimental quantification of their consequences and interactions is desirable.

The influence on VGRS of proprioceptive stimuli from ocular muscles engaged in image-pursuit eye movements[17] as well as the role played by thought processing per se during game playing[26,27] deserve consideration. However, the former mechanism is hypothetical so far. Moreover, some doubt is cast on the relevance of the latter factor by the observation that one patient studied by Maeda et al[14] as well as two of our subjects experienced seizures while not actively playing but watching VGs played by another person. Fatigue, sleep deprivation, or both that were common among our patients, as well as those studied by others,[5,10,13,16] are more likely contributory factors, although their operating mechanisms are unclear.

Diagnosis of VGRS

The findings discussed so far indicate that the diagnosis of VGRS should be considered whenever a generalized or partial seizure occurs in a person playing or watching a VG. This suspicion is strengthened by: (1) the repeated occurrence of seizures during VGs, (2) a history of previous epileptic manifestations triggered by other visual stimuli, and (3) the laboratory demonstration of EEG paroxysmal responses to a variety of visual excitations, including actual VG playing, stroboscopic stimulation, and presentation of line patterns. However, considerable temporal variability has been demonstrated in the epileptogenic effects of these excitations in the same individuals.[9,14,28] Thus, failure of the EEG to respond paroxysmally to any or all of these stimuli should by not regarded as invalidating the clinical diagnosis of VGRS.

Treatment of VGRS

Our experience and the experience of previous investigators strongly suggest that abstinence from playing or watching VGs is the treatment of choice for patients whose seizures occur exclusively in response to visual excitations. This abstinence should be complemented by avoidance of other offending visual stimuli, especially intermittent light flashes, to the extent that this is possible. Other measures, such as increasing the distance at which the VG screen is viewed and wearing tinted glasses are of questionable practicality and unproved effectiveness. The protracted administration of anticonvulsant medications is warranted only for those individuals who cannot refrain from playing VGs, suffer from seizures triggered by other unavoidable visual stimuli, or also have unprovoked epileptic attacks. The demonstrated effectiveness of valproic acid in photosensitive epilepsy,[29] provides a rationale for the use of this drug in the treatment of VGRS. Because of the rarity of the seizures suffered by most patients with VGRS, timely discontinuation of anticonvulsant medication after the subsidence of seizures should be considered.

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