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Photosensitivity in Idiopathic Generalized Epilepsies

Athanasios Covanis

Neurology Department, The Children Hospital "Agia Sophia," Athens, Greece

Summary: Photosensitivity is an abnormal visual sensitivity of the brain in reaction to flickering light sources or patterns and is expressed in the electroencephalogram as generalized spike-and-wave discharge and in more susceptible individuals as clinical seizures. The most common types of seizures are generalized tonic-clonic, followed by myoclonic and absence. The photosensitive epilepsies are classified as *pure photosensitive*, where seizures occur only with the flickering light source/pattern or during intermittent photic stimulation (IPS) in the laboratory, and *epilepsy with photosensitivity*, where spontaneous seizures also occur. Positive response to IPS in idiopathic epilepsy syndromes, which are included in the International Classification or are in development, is reported to range from 7.5% in juvenile absence epilepsy to 100% in pure photosensitive epilepsy.
The treatment of photosensitivity and pure photosensitive epilepsy with rare seizures includes general and specific protective measures. For most patients, however, combination treatment with antiepileptic drugs is necessary. Valproic acid monotherapy has a success rate of 73–86%. Levetiracetam appears to be a new alternative therapeutic option. Clobazam, lamotrigine, ethosuximide, and topiramate also have been recommended as second-choice therapies. **Key Words:** Photosensitivity—Intermittent photic stimulation—Antiepileptic drugs.

Photosensitivity is an abnormal visual sensitivity of the brain in reaction to flickering or intermittent light sources or patterns; it is expressed in the electroencephalogram (EEG) as a generalized spike-and-wave discharge (photoparoxysmal response, PPR). Typical PPRs are associated with epilepsy in approximately 95% of the cases and occur in 5% of patients with epilepsy.

Pathophysiology

The genesis of electroclinical manifestations in human and animals (*Papio papio* baboons) seems to be cortical (1), involving in particular the fronto-rolandic and occipital areas (2). The PPR is mediated by the parvocellular visual pathway (3), predicts clinical photosensitivity (4), and may be either wavelength or quantity-of-light dependent (5). The alternative hypothesis (6) supports the idea that in cortical hyperexcitability, the discharge may start in the magnocellular cells, where the magnocellular projections offer the greatest opportunity for synchronizing cortical activity; this activity in visually sensitive patients is uncovered, possibly because of an impairment of contrast gain-control mechanism (7). The spread of the discharges to the primary motor cortex or suprasylvian or infrasylvian cortical areas produces myoclonus or generalized tonic–clonic seizures, or complex partial seizures, respectively (8,9). The synchronization and generalization of the EEG paroxysmal discharges in both hemispheres is mediated by the corpus callosum. In pure photosensitive epilepsy and idiopathic generalized epilepsies (IGEs), it seems that there is a sole defect and a modifying defect, respectively, of the photosensitive pathway, while in the neurodegenerative disorders, the defect is induced by collateral abnormality.

Photosensitivity and trigger factors

The provocative trigger factors that produce seizures in photosensitive patients are television (TV), video and computer games, and natural (e.g., sunlight reflections) and environmental (e.g., fluorescent, stroboscope/disco) lighting (10). Any changes in luminance/contrast, pattern, and color can provoke seizures in photosensitive individuals. In TV epilepsy, the flickering light becomes risky above five flashes per second. For videogame players, in addition to the TV characteristics, the visual content of the game is also important (11). There is little doubt that variable patterns within the broadcast or videogame material with a high rate of image change of high-contrast stimuli can induce seizures in certain individuals. These broadcast-material pattern alterations are at risk for producing seizures if there are more than three repetitions per second. There are several other factors, such as binocular versus monocular stimulation (10), oscillating versus static and drifting patterns, alterations in lightness

Address correspondence and reprint requests to Dr. Ahanasios Covanis, Neurology Department The Children Hospital "Agia Sophia," 11527, Goudi, Athens, Greece. E-mail: graaepil@otenet.gr

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and color (12,13), and long-wavelength red color (14,15), that enhance the predisposition to seizures. Furthermore, the red-blue color is more provocative than is luminous red-green (16). Additional nonphotic factors such as cognitive abilities, anxiety and excitement, and fatigue and sleep deprivation may facilitate the occurrence of PPRs and seizures. Spontaneous seizures by chance do occur while viewing TV or playing a video game.

Photosensitivity is essentially a laboratory finding and can be established only by using an adequate technique. Important factors include the photostimulator, the methodology, and the interpretation (10,17).

Incidence

The incidence of photosensitive epilepsy is 1.1 per 100,000 persons per year among the general population (18), representing about 2% of all new cases of epilepsy. The highest incidence rate of seven per 100,000 persons per year is found among those between the ages of 7 and 19 years (19), representing 10% of all new cases of epilepsy. The proportion of photosensitive subjects who do not suffer from epilepsy in the general population is not known; however, photosensitivity was found in one of 2,732 apparently healthy aircrew individuals (20). There are several inconsistent reports suggesting racial (21), environmental, and constitutional differences associated with photosensitivity (22) that were not confirmed by other studies (23).

Genetics

There is evidence for a genetic cause or component in photosensitivity. The evidence includes familial occurrence, association with IGE, association with monogenic disorders, genetic and experimental animal models, and the increased risk in siblings. Siblings of children with generalized PPR have a 19.3% risk of photosensitivity, versus a risk of 3.4% for control subjects. This risk is increased to 50% if one parent is also affected (24). It is, therefore, obvious that PPR is an epilepsy-related EEG trait with a high prevalence in idiopathic epilepsies, particularly in IGEs such as childhood absence epilepsy (CAE) and juvenile myoclonic epilepsy (JME). Recent evidence suggests a linkage for PPR on chromosomes 7q32 at D7S1804 and 16p13 PPR at D16S3395 (25).

TYPES OF SEIZURES CAUSED BY FLICKERING LIGHTS OR PATTERNS

Photosensitivity can manifest merely as a paroxysmal EEG response to intermittent photic stimulation (IPS) and in more susceptible individuals as clinical seizures. The effectiveness of IPS depends on its frequency, which suggests hypersynchronization. The sleep-wake video-EEG remains essential for the accurate classification of seizures, epilepsies, and syndromes; it guides the clinician toward therapy, prognosis, and selecting patients for genetic studies. On the basis of our personal experience, the following types of seizures are observed during IPS.

Generalized tonic–clonic seizures

Generalized tonic–clonic seizures (GTCS) are the most common type of seizure, occurring in about 80% of photosensitive individuals and in nearly 100% of pure photosensitive individuals. GTCS may follow repeated myoclonic jerks or may occur without any preceding clinical phenomena, usually after long exposure to visual stimulus. Secondary generalization from the occipital lobes can also occur even minutes after the exposure.

Myoclonic seizures

Myoclonic jerks are synchronous (massive), asynchronous (fleeting), symmetrical, asymmetrical, bilateral, unilateral, or focal. They occur at the beginning of, during, at the end of, or immediately after the generalized spike-and-wave discharge. The visual stimulus provokes jerks identical to those seen spontaneously in the same patient, usually involving the upper part of the body (head, shoulders, upper limbs). In infancy and childhood, they may have a scissoring pattern or present like a slight movement, flicker, or tremor. In self-induced epilepsy, jerks usually involve the waving hand. The intensity varies from a subjective feeling to a just-recorded, visible or moderate, and marked myoclonic jerk that causes the patient to have a minor accident in the form of dropping things, clumsiness, or even a fall. Massive jerks involving all four limbs and the body in flexion do occur, particularly in younger children. The visual stimulus induces jerks more easily during drowsiness, which, if sustained, can cause repeated jerks that may lead to GTCS.

Absence seizures

Absence seizures may be very brief and inconspicuous occurrences that include eye opening and closing, a stare, or a jerk and stare observed during video recordings. Complex absence seizures that are evoked by IPS and outlast the stimulus do occur and are easily observed. They may intermix with myoclonic jerks of the face (eyebrow, perioral) or follow a jerk.

Tonic seizures

Tonic seizures are a rare, IPS-triggered phenomenon. Some jerking of the head may evolve into a sustained versive posture of head and eyes to one side, lasting as long as the stimulus (26) or outlasting the stimulus and leading in some cases to GTCS.

Partial seizures with simple (27–30) or complex visual symptoms (31) or early limbic symptoms (27,32) may occur. Other subjective phenomena in the form of dizziness, funny or pleasurable feelings, epigastric sensation, nausea, or simple visual hallucinations may or may not also present in association with mild generalized atypical discharges during IPS.

TABLE 1. *Photosensitivity in epilepsy*

Pure photosensitive epilepsy (40%)
Seizures only with flickering light source or visual pattern
Epilepsies with photosensitivity (60%)
Seizures with flickering light source or visual pattern and spontaneous, including
Early-onset absence epilepsy
Childhood absence epilepsy
Myoclonic epilepsy in infancy/childhood
Juvenile absence epilepsy
Juvenile myoclonic epilepsy
Eyelid myoclonia and absences (Jeavons syndrome)
Epilepsy with generalized tonic–clonic seizures on awakening
Other myoclonic epilepsies/syndromes[a]
Focal epilepsies
Progressive myoclonic epilepsies

[a]Epilepsy with myoclonic absences, severe myoclonic epilepsy in infancy, facial myoclonias with absences, myoclonic astatic epilepsy.

GENERALIZED EPILEPSIES AND SYNDROMES

The classification of epileptic seizures in 1981 (33) and the classification of epilepsies and epileptic syndromes in 1989 (34) were challenged in 2001 by a new, more flexible, and dynamic classification scheme (35).

Clinical photosensitivity is characterized by seizures occurring only with the flickering light source/pattern or during IPS in the laboratory (pure photosensitive epilepsy), or spontaneous seizures and seizures in association with visual stimulus (epilepsy with photosensitivity) (36–39). In the latter group, photosensitivity is associated with syndromes of idiopathic generalized/partial or cryptogenic epilepsy and some progressive myoclonic encephalopathies (Table 1).

Early-onset absence epilepsy, under 3 years of age

The mean age of onset is 2.3 ± 0.7 years. The classification into myoclonic (63%) and nonmyoclonic absence seizures (37%) and their subdivisions is reminiscent of well-defined syndromes in infancy and childhood, and has predictive and prognostic implications (40). Photosensitivity occurs in 60% of patients in the myoclonic group, while a few nonmyoclonic, particularly those evolving to JME, may show IPS-positive response later during drug withdrawal.

Myoclonic epilepsy in infancy/childhood

Myoclonic seizures in infancy (onset at 13 ± 4.8 months) or childhood (onset at 3.0 ± 0.9 years) may be the only type of seizures (41%), or they may be associated with GTCS (25%), absences (21%), or both (13%). In 56 cases, photosensitivity was found in 20% of patients.

Myoclonic astatic epilepsy of early childhood

The main seizure type in myoclonic astatic epilepsy (MAE) is astatic and myoclonic, but GTCS and absence and tonic seizures occur in at least two thirds of cases. PPRs in patients between the ages of 5 and 15 years have been observed in the majority of cases (41). In our series, photosensitivity was found in 11% of cases. An overlap exists between MAE and benign myoclonic epilepsy in infants and children.

Severe myoclonic epilepsy in infants

With severe myoclonic epilepsy in infants, seizures begin during the first year of life, usually as generalized or unilateral febrile seizures. Myoclonias and psychomotor retardation become apparent from the second year of life onward. The EEG shows generalized spike and polyspike waves and early photosensitivity in 30%–40% of cases (42).

Epilepsy with myoclonic absences

Absences start around 7 years of age and are associated with myoclonic jerks and body deviation or staggering (43). PPR was observed in 17% of our cases.

Childhood absence epilepsy

CAE is characterized by frequent daily absences (pyknolepsy) in otherwise normal children, more frequently in girls. CAE is associated with photosensitivity in 18% (44), and in a few cases, absence seizures evoked by IPS may follow myoclonic jerks that are only observed during IPS. Photosensitivity combined with brief generalized spike-wave discharges is an unfavorable EEG feature not observed in pure childhood absence epilepsy (44).

Facial myoclonias with absences

Typical absence seizures in some children are associated with perioral or eyebrow myoclonias or both. The mean age of onset is 5 ± 3 years, with spanioleptic or pyknoleptic absences associated with spike or polyspike waves and wave activity of 1.5–4 Hz. In our 13 cases, PPR was evoked in one child (7%) by switching on the TV screen and consisted of generalized polyspike activity.

Eyelid myoclonia and absences

In eyelid myoclonia and absences (ELMA), the combination of clinical and EEG phenomena is unique and pathognomonic of this syndrome (Jeavons syndrome). There is marked jerking of the eyelids, often with upward deviation of the eyes, associated with spike-wave discharges that are often irregular, immediately after eye closure, and invariably evoked on IPS (45,46). ELMA represents 2.5% of all epilepsies and up to 13% of IGEs (46,47). In our population of 50 cases studied by sleep-wake video EEG after sleep deprivation, absences were seen in all. Absences in ELMA may be as brief as a transient glare or a hesitation or gap in counting; during hyperventilation and IPS, absences are also seen without eyelid myoclonias (46). Myoclonic jerks other than eyelid jerks occurred in 34% (46) and 54.5% (47) of children and adults, respectively. GTCS occurred in 50% of our population of children, and were the usual referral symptom.

Photosensitivity was seen in 92% and marked in 76%. In the remaining 8% (age range 0.5–3.5 years) (46), the negative response may be attributed to poor cooperation during testing.

Juvenile absence epilepsy

Juvenile absence epilepsy is characterized by fewer absences per day (spaniolepsy), less abrupt onset, and less profound loss of consciousness than is CAE. In this form of absence, GTCS occurs in 80% of cases, myoclonic jerks occur in 15%–20% of cases, and photosensitivity occurs in 7.5% of the cases (48).

Juvenile myoclonic epilepsy

The most characteristic clinical manifestation of juvenile myoclonic epilepsy (JME) is the occurrence of myoclonic jerks involving the upper part of the body, usually within 15–30 min after awakening. GTCS, absences, and PPRs occur in our experience in 60%, 48%, and 75% of cases, respectively. In almost 50% of cases, the response to IPS is marked. The positive response to IPS increases to 83% in patients with JME with onset under the age of 12 years. Seventy-six percent of patients with photosensitive JME experience mixed seizures (jerks and absences, 24%; jerks and GTCS, 23%; jerks, absences, and GTCS, 29%); 24% experience jerks only. Others have reported photosensitivity rates in JME ranging from 30% to 54% (36,48,49).

Epilepsy with generalized tonic-clonic seizures on awakening

GTCS characteristically occur during awakening, drowsiness, relaxation, and excessive alcohol intake. The age of onset extends from early childhood to adolescence, with a peak around puberty. The EEG shows generalized spike-wave discharges of 2.5–4.5 Hz. In our 72 cases, PPR was recorded in 45 patients (62.5%). Others have found PPR rates ranging from 13% to 33% (36,50).

Pure photosensitive epilepsy

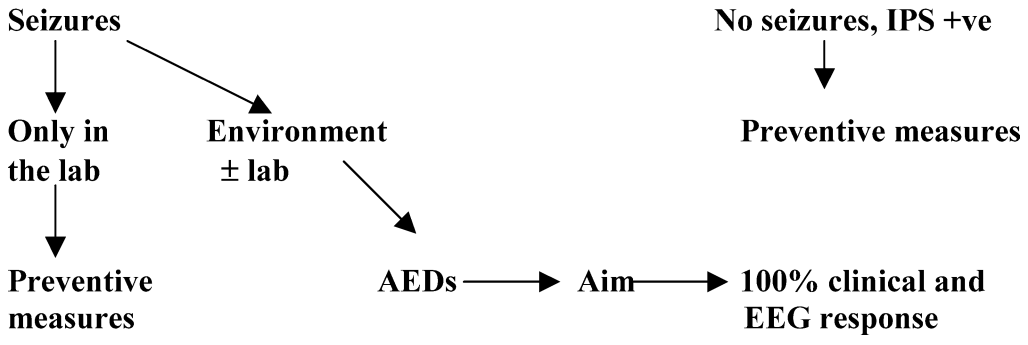
In pure photosensitive epilepsy, visually induced seizures have increased after the introduction of different visual display units and other provocative light sources. The seizures provoked by IPS, pattern, or both are GTCS alone, combined with myoclonic or absence seizures, or mixed seizures. Positive response to IPS/pattern is usually marked and has a wide range (under 5–60 flashes/sec).

TREATMENT OF PHOTOSENSITIVITY

The effectiveness of stimulus avoidance and modification (glasses, video hazard blockers, special TV screens) will depend on the individual's degree of photosensitivity, awareness of subtle signs and symptoms when exposed to potentially provocative stimuli, and coping strategies. Patients with seizures exclusively triggered by visual stimuli can be treated without drugs. For most patients, however, combination treatment with antiepileptic drugs is necessary, especially because avoidance of visual stimuli in modern daily life is becoming increasingly difficult (51). Treatment algorithms are shown in Tables 2 and 3. The first choice of drug is valproic acid monotherapy, which has success rates that range from 73% to 86% (51). Levetiracetam is an alternative choice for patients with IGE and photosensitivity (52). Levetiracetam and valproic acid are well tolerated when combined, adding to each other's effectiveness. Clobazam, lamotrigine, ethosuximide, and topiramate also have been recommended as second choices.

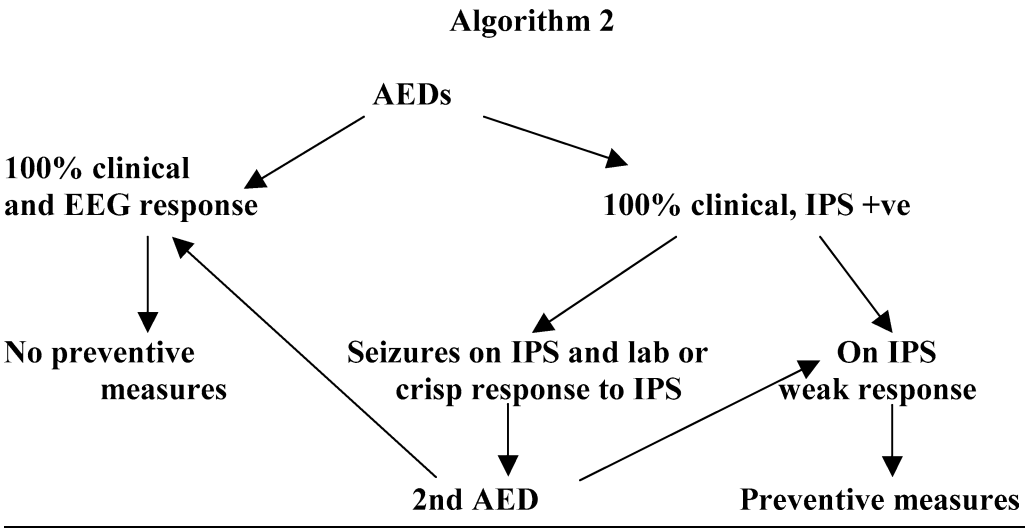
TABLE 2. *Treatment of photosensitivity and photosensitive epilepsies*

Algorithm 1



IPS, intermittent photic stimulation; AEDs, antiepileptic drugs; EEG, electroencephalogram.

TABLE 3. *Treatment of photosensitivity and photosensitive epilepsies*



IPS, intermittent photic stimulation; AEDs, antiepileptic drugs; EEG, electroencephalogram.

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