Neuropsychiatric Disease and Treatment

Dovepress
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REVIEW

Reflex epilepsy: triggers and management strategies

This article was published in the following Dove Press journal:
Neuropsychiatric Disease and Treatment

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Abstract: Reflex epilepsies (REs) are identified as epileptic seizures that are consistently induced by identifiable and objective-specific triggers, which may be an afferent stimulus or by the patient's own activity. RE may have different subtypes depending on the stimulus characteristic. There are significant clinical and electrophysiologic differences between different RE types. Visual stimuli-sensitive or photosensitive epilepsies constitute a large proportion of the RE and are mainly related to genetic causes. Reflex epilepsies may present with focal or generalized seizures due to specific triggers, and sometimes seizures may occur spontaneously. The stimuli can be external (light flashes, hot water), internal (emotion, thinking), or both and should be distinguished from triggering precipitants, which most epileptic patients could report such as emotional stress, sleep deprivation, alcohol, and menstrual cycle. Different genetic and acquired factors may play a role in etiology of RE. This review will provide a current overview of the triggering factors and management of reflex seizures.

Keywords: seizure, reflex epilepsy, photosensitivity, hot water, reading, thinking

Introduction

Reflex epilepsies (REs) are epileptic events precipitated by external stimuli, internal mental process, or both. Some external stimuli are simple such as light flashes, fixation-off, hot water, visual, vestibular, auditory or tactile stimuli, or complex such as reading or listening to music. Intrinsic stimuli can be elementary or higher cerebral functions such as movement, emotion, thinking, calculation, and cognitive functions. The role of stimuli on seizures was first described by Marshall Hall in 1850 and since then many stimulus types have been defined. Photosensitive epilepsy is the most common among the various types of REs, which has been identified over the years. Gowers noted in 1885 that epileptic seizures could be caused by sudden loudness, sudden light, and sudden muscle contractions.[1] For the first time in the 1989 International League against Epilepsy (ILAE) classification, the definition of epilepsy with specific stimuli has been established.[2] Reflex seizures (RS) and RE definitions were mentioned in the next classification of ILAE (2001), whereas in the later electroclinical syndrome classification, REs were considered among less age-related epilepsy groups.[2] Recently, the classification for epileptic seizures has been updated. It recognizes new types of focal and generalized seizures, provides additional information on causes, and clarifies obscure or questionable words and terms used to name seizures with more meaningful ones. But there is no new classification proposal for epilepsies and epileptic syndromes, which leaves RS to be identified according to seizure type and etiology in an individual case.[3,4]

Electroclinically, the seizures may be either focal or generalized where etiology may also be either idiopathic or symptomatic. RS are clinically very similar to unprovoked seizures, except for the presence of specific stimuli.[5] The prevalence of RE ranges

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Dovepress
http://dx.doi.org/10.2147/NDT.S107669

from 4% to 7% for all epilepsy patients and up to 21% in idiopathic generalized epilepsies.[1] Factors causing RS are visual stimuli (75%–80%), thinking, music, eating, praxis, somatic sensory, proprioceptive, reading, hot water, and startling. Seizures induced by other special conditions, such as fever or alcohol withdrawal, are not RS.[2] Table 1 summarizes the main epidemiological, clinical, genetic, therapeutic, and prognostic features of RS.[10,31]

Versatile studies of cerebral function demonstrated that ictogenic mechanisms in reflex epilepsies generally originated from the stimulation of functional anatomic networks ordinarily functioning for highly complex physiological activities.[6] The pattern of familial photoparoxysmal response (PPR) indicates a complex mode of inheritance, involving several genes.[7] A study on monozygotic twins showed nearly 100% concordance in PPR.[8] Furthermore, siblings of patients with generalized PPRs are very prone to have PPR than siblings of the control group (19.3% vs 3.4%).[9] The family studies determined an autosomal dominant inheritance pattern with reduced penetrance and identified putative loci chromosomes 6, 7, 13, and 16.[10] RS have been associated with peculiar genetic disorders such as trisomy 21 or inv-dup 15. RS are found in nearly 20% of trisomy 21 patients but have not been reported in Angelman syndrome (AS).[112] Reduction of central inhibitory control in trisomy 21 is thought to be a combination of structural brain abnormalities typical of this chromosomal disorder, including reduction of neuronal density, decreased density and size of synapses, reduction of dendritic spines of pyramidal neurons, atrophy, and lack of growth of dendritic trees. A dysfunction of nucleus reticularis pontis caudalis may be seen in patients with trisomy 21, which causes an exaggerated startle response leading to an increase of proprioceptive feedback to a hyperexcitable motor cortex, precipitating a seizure.[113] An overexpression of cerebral gamma-aminobutyric acid (GABA)-mediated inhibition in AS accounted for RE, in which seizures were provoked by complex stimuli, such as kissing or viewing of pleasant or funny events.[114]

Animal models for some epilepsy types have been represented. Primary reading epilepsy and some other epilepsy types cannot be represented in animal models, while audiogenic seizures (AGS) and some others were easily observed in several rodent strains as an innate feature. The *Papio papio* baboon represents a good model of generalized epilepsy with photosensitivity (PS) derived by natural selection in the wild.[10,11] Also the epileptic strain of Fayoumi chickens (Fepi) represents a non-mammalian model of PS when having a spontaneous gene mutation in the synaptic vesicle glycoprotein 2A.[10,12] This mutation was never detected in humans. Frings mice, which carry a mutation in *MASS1* gene, and dilute brown Agouti coat color mice (DBA/2J) in which Asp-1 and Asp-2 have been nominated as candidate genes are two well-established spontaneous models of generalized AGS.[13] High susceptibility to acoustic stimuli has been demonstrated in Black Swiss mice. The genetically epilepsy-prone rats, the Wistar Audiogenic Rats model, and Genetic Audiogenic Seizure Hamsters developed in Salamanca manifest AGS in response to acoustic stimuli. Recently, feline audiogenic RS have been identified.[10,14]

Common types of reflex epilepsy

Photosensitive epilepsy

Photosensitivity (PS)

PS is an abnormal visual sensitivity response of the brain to light stimuli, intermittent light sources, and more complex stimuli such as television (TV), video games, and visual patterns.

The prevalence in general population is low (1/4000) however it is around 2–5% of all patients with epileptic seizures.[15,16] It is most commonly seen in adolescents and women. The annual incidence of PS among epilepsy cases is 10% in 7- to 19-year olds.[15] Increased artificial light stimulation in recent years has significantly increased the likelihood of clinical manifestation of this phenomenon.[17] Seizures often have autosomal dominant inheritance with reduced penetrance but may also have recessive inheritance.[18] EEG abnormality with light or pattern stimulation was seen in 0.3%–3% of the whole population. PS is associated with many types of seizures. Eyelid myoclonus, generalized myoclonic jerks, absence seizures, generalized tonic-clonic seizures, focal seizures and, more rarely, tonic versive seizures and focal asymmetric myoclonic seizures may be triggered by photic stimulation.[19]

In response to intermittent photic stimuli, PS can simply be examined in three sub-groups:

1) Only those who have only photically induced seizures without spontaneous seizures (pure photosensitive epilepsy).
2) Those who have spontaneous and photosensitive seizures.
3) Asymptomatic people who have light sensitivity in EEG.

Almost all of the photosensitive patients are sensitive to flickering lights.[20] Many natural light sources can provoke epileptic seizures like in eyelid myoclonic epilepsy and some of the self-induced seizures.[21] Video games and TV are the most common triggers.[19]

PS can occur in different epilepsy syndromes, such as with juvenile myoclonic epilepsy (JME), childhood epilepsy with occipital paroxysms, absence epilepsy and is

Table 1 Summary of the main epidemiological, clinical, genetic, therapeutic, and prognostic features of reflex seizures[10,31]

Type of reflex epilepsy	Sex, prevalence	Genetics	Identified loci or genes	Seizure type	Epileptic syndromes or associated conditions	Prognosis	Treatment
Photosensitivity	1/4,000 (2%–10% of PWE) female > male (60%)	Likely autosomal dominant with reduced penetrance, independent from seizures disorder	6p21, 7q32, 13q31, 16p13	– Absence, myoclonia – GTCS – Focal (mainly occipital)	– GGE (especially JME) – IPOE – PME – DS Rarely with acquired lesions	Usually favorable response, may remit in 25% after age 30 years	Preventive measures (stimulus avoidance, lens, etc.) VPA first choice, LTG and LEV as the second choice
Musicogenic epilepsy	1:10,000,000	Usually none reported in patients with ADTLE	LGI1/Epitempin SCN1A	Usually temporal lobe seizures	Epilepsies with epileptogenic lesions also in patients with ADTLE	Variable, usually refractory	Stimulus avoidance Medication for focal seizures
Reading epilepsy	Rare, male/female: 1.8/1	Autosomal dominant inheritance with incomplete penetrance	None	Jaw jerks that may progress to GTCS if reading continues Rare: focal seizures, with alexia and variable degree of dysphasia	Considered a variety of GGE; described in patients with JME	Benign, thus well responding to treatment	Stimulus avoidance (interruption of reading) VPA first choice LEV and CLN as the second choice
Eating epilepsy	1/1,000–2,000 PWE, male/female: 3/1	Unknown familial cluster in Sri Lanka	MECP2	Focal seizures with or without impairment of awareness	Usually epilepsies with epileptogenic lesions	Variable	Stimulus modification, medication for focal epilepsy CLB before meal Surgery
Hot water or bathing epilepsy	Rare (more common in India and Turkey), male predominance (70%)	Likely autosomal dominant	10q21.3–q22.3 and 4q24–q28 Synapsin I GPR56	Focal seizures	None	Relatively benign	Stimulus avoidance/modification (shortened bath times, decreasing the bath water temperature) BZD as needed
Seizures induced by somatosensory stimuli	Rare, unknown	Unknown	Unknown	Sensory aura followed by a sensory Jacksonian seizure with tonic motor manifestations. Secondary generalization may occur	With MCD and post-santral cortical lesions	Variable	As for other symptomatic or focal epilepsies
Seizures induced by proprioceptive stimuli	Rare, unknown	Usually none	Unknown	Myoclonic or somatomotor or somatosensorial seizures Evolvement focal to bilateral may occur	Acquired brain lesions non-ketotic hyperglycemia acute diffuse encephalopathies	Variable	Medication for focal seizures
Seizures induced by orgasm	Very uncommon, female predominance	None	Unknown	Focal seizures	Usually with acquired lesions	Variable	Medication or surgery
Seizures induced by thinking or praxis	Usually overlapping with JME	Usually overlapping with GGE	Unknown	Myoclonia, absence, GTCS	GGE	Benign (as JME)	Same medication as GGE

Abbreviations: ADTLE, autosomal dominant temporal lobe epilepsy; BZD, benzodiazepines; CLB, clobazam; CLN, clonazepam; DS, Dravet syndrome; GGE, genetic generalized epilepsies; GTCS, generalized tonic-clonic seizures; IPOE, idiopathic photosensitive occipital lobe epilepsy; JME, juvenile myoclonic epilepsy; LEV, levetiracetam; LTG, lamotrigine; MCD, malformations of cortical development; PME, progressive myoclonus epilepsies; PWE, patients with epilepsy; VPA, valproic acid.

also common in progressive myoclonic epilepsies (PME).[10] Adult-onset neuronal ceroid lipofuscinoses associated with the *CLN6* mutation is a rare form of PME severe photosensitivity was reported in two siblings recently.[116] The other form of PME is Lafora disease where photosensitivity was enhanced during low frequency photic stimulation.[117]

EEG findings in photosensitive epilepsy

Resting EEG is usually normal in idiopathic photosensitive epilepsies. Twenty-thirty percent of the cases show paroxysms, which occur within 1–3 seconds when the eyes are closed and last for 1–4 seconds. Similar findings may also occur with intermittent photic stimulation (IPS), which is important during EEG recording to induce PS. It should not be forgotten that long-term IPS may also provoke generalized convulsive seizures and so patients who have low seizure thresholds for IPS should be closely monitored. Patients may have one or more types of seizures associated with IPS, whereas routine IPS may not be sufficient to elicit PS in the EEG. For this reason, it may be useful to use more powerful techniques such as sleep deprivation or long-term IPS additionally.

In some patients with generalized myoclonic epilepsies, there may be PPR in EEG even if seizures are not provoked by IPS.[20] The severity, frequency, and duration of IFS are important in response to IPS. An abnormal PPR is seen when the intensity of light is high, and when given for a longer time at frequencies of 12–20 Hz. The presence of geometric patterns with IPS is more effective than long-term administration of white light. Binocular stimulation is more effective than monocular. For this reason, in the case of PS, it is recommended to close one of the eyes if the subject is exposed to intense lights such as in a discotheque. Central stimulation is more effective than peripheral. During IPS, the range of PS can vary from lowest to highest in order to obtain a PPR. Alertness, attention, emotion, menstrual cycle, hormones, electrolytes, and some drugs change the photoconvulsive threshold.[18,20] Waltz et al recommended an EEG classification system for photosensitive patients.[22] According to this classification, class I represents occipital spikes; class II represents local parieto-occipital spikes and biphasic slow waves; class III represents parieto-occipital spikes and biphasic slow waves spreading to frontal regions; and class IV represents generalized spikes or polyspikes and waves.

Characteristic of visual stimuli

The likelihood of seizure increases depending on the luminance of the light source. Monocular stimulation may reduce the risk of seizures.[23,24] The majority of patients with PS are sensitive to certain patterns.[25,26] Seizures are most often seen when the frequency of the flash stimulus is 15–25/second.[27] The prolonged exposure to light increases the triggering of the seizure, but the intake of certain medicines, especially sodium valproate, decreases the PS. The red color at the wavelength of 660–720 nm is at a higher risk of seizures compared to the blue and white colors.[28] It is especially common in video games. It has been shown that not only PS but also cognitive function, emotional excitement, and rapid hand movements may also have a role in seizure induction. High-contrast stimuli are more likely to trigger seizures.[24]

Mechanism of photic-induced seizures

The stimulation of a critical neuronal mass in the occipital cortex is important in the pathogenesis of seizures in photosensitive patients who have intrinsic hyperexcitability of the visual cortex, which can predispose to a large-scale neuronal activation.[6,29] Diffuse inadequacy of GABAergic inhibition was proposed to be the probable mechanism for PS.[30] Electrophysiology studies suggest that diffuse or multifocal hyperexcitability including cortico-cortical or cortico-subcortical pathways may have a role in addition to occipital cortex hyperexcitability.[6,31] In a study, the motor cortex of photosensitive baboons was recruited after stimulation of occipital cortex and produced generalized myoclonic jerks along with hyperactivity of cortico-subcortical loops including the reticular formation and the thalamus.[32] An EEG–fMRI study in patients with JME suggests that the putamen can act as a mediator between the visual and motor-related fields triggering a motor system hyperfunction during generalized PPR.[33]

The cortico-cortical propagation from the occipital to premotor cortex along the intraparietal sulcus was determined to cause PPRs in an electrical imaging analysis.[34]

Factors that trigger photosensitive seizures

Television-induced seizures

The TV-induced seizures, which were first described by Charlton and Hoefer and revised by Dahl, are the most common type of photosensitive epilepsy and the most common external stimuli that provoke photosensitive seizures. Nearly 10% of the patients have a family history of TV epilepsy. Seizures mainly affect children in the age group of 10–12 years and twice as often in girls than boys. Photosensitive patients experience seizures during regular TV watching. Flickering screen, the closeness of the screen distance (less than 1 m), the intensity of the image, and the room light

producing contrast or brightness on the screen can induce seizures.[19] Ten percent of the patients report that they are "drawn like a magnet" to the screen when they are watching TV too closely followed by a generalized tonic-clonic seizure. This condition is known as "compulsive attraction".

Flicker frequency is the most important factor in photosensitive epilepsies, with the lower frequency TV sets (50 Hz) more liable to induce a photosensitive response on EEG than a 100 Hz monitor.[110] LCD and plasma TV screens have a transistor to keep all the pixels state, which prevents the manifestation of flickering. LCD TV screens are less likely to trigger a seizure than plasma TV screens. There is not any definite symptom that proves 3D movies trigger a seizure more that 2D movies in a patient with photosensitive epilepsy.[111,115]

Video game-induced seizures

Video game-related seizure is one of the most common types of PS epilepsies. It was reported in 1981 for the first time by Rushton, who presented a 17-year-old boy with seizures during a 15 Hz flashing multicolored part of an arcade game.[36] Later on, the scenes with flash lights in another game named "Dark Warrior" also were observed, which provoked seizures.[37] It is more common in boys than in girls (probably because boys play more video games than girls). The annual incidence of first seizures triggered by playing electronic screen games was found to be 1.5/100,000 between 7 and 19 years in the UK.[15] The role of colors was started to be searched after the referral of hundreds of children to emergency services with seizures when watching a cartoon movie named Pokemon in Japan in 1997.[38] PS plays a role in 70% of the seizures that are induced by video games. Although video game sensitivity is usually not distinct from PS, it is noteworthy that 1/3 of the cases do not develop PS during IPS.[39] "Super Mario World" was proved to be more provocative than the standard program (IPS) in the EEG laboratory.[40]

The mechanisms that induce seizures in video games are as follows:

1) PS
2) Pattern sensitivity
3) Emotional or cognitive excitation (excitement, tension)
4) Proprioceptive stimuli (movement/praxis)

Also, fatigue, insomnia, and playing games for a long time facilitate the seizure activity.

Three criteria were described for the diagnosis of video game-induced seizures. First of all, seizures occur while playing video games, second there is history of PS with epileptiform activity induced by other types of visual stimuli, and third patients have PPR in EEG records including two during video game playing.[41]

Management and treatment of photosensitive epilepsy

More than one factor can trigger epileptic seizures in patients with photic or pattern sensitivity. There are certain recommendations published to prevent their seizures. Antiepileptic drugs (AEDs) are not preferred as the first-line therapy. Patients with pure photosensitive seizures should avoid the factors that provoke seizures. For example, patients with TV-induced seizures should watch TV from a distance of at least 2 m in a well-illuminated room, use a remote control, view on a 100 Hz TV, and try not to watch for a long time especially while they are tired and sleepy. Patients with video game-related seizures should not be allowed to play when they have fatigue and insomnia. Subjects with a history of epilepsy and a family of PS at the same time need to be careful about playing electronic games. Photosensitive children should not play electronic games when they are alone. Furthermore, patients with PS may use polarized glasses on sunny days, or can close one eye during the exposure of flash lights. If these precautions are not enough to control seizures, drug treatment may be introduced. If patients have idiopathic generalized epilepsy syndromes and PS together, AED treatment is required. Valproate is preferred as first-line treatment in video game-related seizures. Low-dose valproate can be used in patients with spontaneous seizures or spontaneous EEG changes at the same time. A single dose of valproate or vigabatrin is demonstrated to inhibit photosensitive responses on EEG, whereas valproate was found to be 78% effective in significantly reducing the photosensitive range and abolished PS in 50% of patients.[42,43] Levetiracetam can be used in all seizure types, which may reduce both PPR and myoclonic jerks.[109] Benzodiazepines, particularly clonazepam, are effective in myoclonic jerks, and ethosuximide is effective in absence seizures.[44] Lamotrigine and carbamazepine may be effective in treatment but may increase jerks.[35,61] Psychiatric support is also needed for self-induced seizures. General observations suggest that although PPR is still present in adult life, the prognosis of epilepsy is often better with or without AEDs.[45,46] Furthermore phenytoin was reported to aggravate photosensitive epilepsy in a pediatric patient.[47]

Musicogenic epilepsy

Musicogenic epilepsy was described by Critchley and classified as a rare form of complex RE with an estimated prevalence of 1/10,000,000 people.[48] Although it has been known for more than 75 years, little is known about the underlying mechanism.[49] Possibly, it is related to hyperexcitable cortical regions, which could be stimulated to different degrees and extents by various musical stimuli. Not only the

acoustic areas but some other cortical areas are also involved and seizures can be generated by external stimulation with emotional content of the melody or rhythm and the associated memory, which is a precipitating factor in individuals with musicogenic epilepsies; conversely, it is well known that epileptiform activity may be prevented or terminated by listening to some other music.[50,51] Seizures can also be provoked by thinking about a melody or different type of sounds, such as machinery, other than music.[52,53] Emotion may have a role in this particular musicogenic epilepsy. The form of musical stimulus shows variability according to type, composer, instrument, or even emotional content of the music. But a very specific stimulus like church bells and the melody of the Marseillaise was reported to trigger the seizure within seconds.[54,55] In musicogenic seizures, ictal or interictal EEG anomalies are recorded, usually due to focal seizures with or without impairment of awareness originating from the right temporal cortex.[52,53] The mean age of onset of musicogenic epilepsy was 28 years, with a female predominance.[56] The avoidance of the provocative music is usually the first-line treatment. If preventive measures and avoidance are not possible, antiepileptic medication is suggested.[57,58] Various behavioral therapies, psychotherapy, and deconditioning techniques have been tried in patients with high emotional state.[59,60]

Thinking (noogenic) epilepsy

Thinking epilepsy is a rare form of RE that can be induced by specific cognitive functions, such as calculation, drawing pictures, playing card games or chess, deciding to do something, solving Rubik cube, thinking, making decisions, and abstract reasoning.[62,63] They can also be triggered planning a project, responding to questions at a business talk, or while presenting mathematical calculation orally. Interestingly, such seizures do not occur during reading, writing, or verbal communication.[18] Thinking epilepsy is usually presented with generalized seizures where focal seizures were reported in only some exceptional cases.[64–66] They occur in the form of bilateral myoclonus, absence, or generalized tonic-clonic seizures usually preceded by myoclonic jerks. However, myoclonic jerks occur alone in 76% and are absent in 60% of patients where absence seizures alone do not occur by thinking.[18] Seizures usually start during adolescence where absence and myoclonic seizures are generally neglected until generalized tonic-clonic seizures arise.[64] Local regional abnormalities, when present, are frequently over the right hemisphere and frontal or parietal regions.[62] Although there seems to be no Mendelian inheritance, family history as well as clinical pattern is similar to that of patients with idiopathic generalized epilepsies, especially JME or juvenile absence epilepsy.[62]

Bilateral synchronous multiple spikes and wave discharges and sometimes temporoparietal or frontal focal abnormalities can be seen in the EEG recordings.[67] Preventing the triggering stimuli in this disorder is not always possible, unlike photosensitive patients. Valproate or other drugs commonly used in JME generally can control seizures in majority of the patients.[62,68]

Sellal et al reported a very unusual epilepsy named "Pinocchio syndrome" in which seizures triggered when the patient attempted to lie.[71] He had experienced epigastric sensations, andauditory and visual illusions with intense anxiety followed by generalized convulsions. Almost all of the episodes occurred when the patient was lying for business reasons. MRI revealed a meningioma located on the anterior clinoid process, which compressed the medial part of the right temporal lobe. Seizures resolved after administration of carbamazepine and ablation of the meningioma. This patient suffered from RE triggered by lying, which may be due to the involvement of mesial temporal lobe and amygdala by this particular type of emotional activity.

Seizures induced by somatosensory stimulation

Seizures provoked by somatosensory stimulation are typically triggered by special stimulations such as skin friction, pricking, touching or tapping, tooth brushing, or stimulation of external ear conduct.[31] In most cases, the effective stimulus is derived from a localized or regional hypersensitive trigger zone like the head and back. The seizures start with sensory aura, followed by a sensory Jacksonian seizure with tonic motor manifestations implying a supplementary motor area seizure. Consciousness is usually preserved during the seizures. This seizure type is characteristically present in patients with postrolandic cortical lesions. In patients with "rub" epilepsy, seizures are reflex generalized myoclonic jerks provoked by tapping and bilateral spike and wave activities are seen in the EEGs.[69] Drugs for focal seizures are effective, and seizures usually reported to respond to valproate.[70]

Proprioceptive-induced seizures

These RS are rare and frequently present as tonic or focal seizures with the passive or active movement of the extremities. It is described as self-induced seizures with compulsive proprioceptive self-stimulation.[72] Proprioceptive stimuli can

induce focal seizures in patients with acute contralateral rolandic or supplementary motor area lesions, and some acute encephalopathies, especially non-ketotic hyperglycemia where it can be a transient phenomenon that does not recur when the metabolic state improves.[64,73]

Proprioceptive-induced seizures begin gradually and initially have sensory Jacksonian manifestations. Although it can be confused with paroxysmal kinesigenic dystonia, the presence of dystonic and choreoathetotic movements, preserved consciousness, normal EEG findings during the attack, and familial clustering in this rare disorder may help for differential diagnosis.[74]

Epileptic nature of the attacks is shown by the ictal EEG recordings.[75] The maximum EEG electronegativity is seen at the central vertex electrode in the seizures induced by walking.[76] Most of patients respond properly to insulin therapy in non-ketotic hyperglycemia and only a few of them need AEDs where carbamazepine and clobazam can be used in the patients requiring treatment.[70]

Eating epilepsy

Seizures triggered by eating are very rare, and their estimated prevalence is nearly 1/1,000–2,000 among all patients with epilepsy.[77] In Sri Lanka, the incidence of eating epilepsy is very high and shows familial clustering in most cases possibly due to specific habits such as eating bulky meals rich in carbohydrates.[78] Physiopathological mechanisms are complex. Seizures are closely related to stereotyped eating behavior, which may differ from a patient to another. Rarely, the smell of food, eating too much or heavy meals, and gastric distension can cause a seizure. Seizures are focal, start short after beginning to eat, and do not repeat during the same meal.[79] Because of that, some people induce seizures by themselves to benefit from the following refractory period preventing the occurrence of a seizure later avoiding a more embarrassing situation. EEG has focal epileptiform abnormalities, which may be secondarily generalized and originate from temporolimbic or suprasylvian structures.[80] Seizures localized to suprasylvian area can also occur with proprioceptive and somatosensory stimuli as well as with other oral activities. The role of taste and autonomic afferents (eating, gastric distention, and emotional status) is more important in temporolimbic seizures. Eating may also trigger periodic spams in some disorders such as infantile epileptic encephalopathy, focal symptomatic epilepsy, or MECP2 duplication syndrome, possibly by activating the frontal-opercular region that evokes the activity of the brainstem or the regions responsible for seizure initiation.[78,81–84]

Seizures triggered by eating can be prevented by modifying the trigger. Clobazam intake before meals was shown to be effective to prevent eating-provoked seizures.[85] AEDs for focal seizures can be effective; however, they can be generally resistant to drug treatment, which may necessitate further evaluation for surgical treatment.[86]

Hot water or bathing epilepsy

Hot water epilepsy (HWE) is induced by bathing with hot water usually over 37 degrees and pouring over the head.[87] The seizures that occur with exposure to hot water may start with self-induction in some patients as they enjoy this situation. It seems to be the second most common type of RE after photosensitive epilepsy[88] and was first described in 1945 by Allen et al in Satishchandra.[89] The seizures may occur from infancy to adulthood with a male predominance (male:female ratio, 2–3:1).[88,90]

Although the physiopathology of HWE remains unclear, it can be related to a damage in the thermoregulation center in the hypothalamus. Satishchandra et al mentioned that development of HWE in humans can be a result of hyperthermic kindling.[91] Patients with HWE were likely to have an abnormal thermoregulation system and were hypersensitive to the rapid rise in temperature during hot water baths, which trigger seizures. This abnormal thermoregulation appears to be genetically determined, and new studies continue to clarify this hypothesis.[89,92]

It usually manifests as focal seizure and can rarely be generalized. Initial symptoms are stunned looking, the feeling of fear, senseless speech, auditory and visual hallucinations, and complex automatisms. They may have spontaneous seizures without hot water exposure. One of the authors (CO: unpublished case) had a patient who experienced a spontaneous generalized tonic-clonic seizure for the first time during sleep, although she had always seizures related to bathing. Further interview revealed that she dreamed of taking shower during sleep, which precipitated her habitual seizure. This unusual condition is another evidence for very complex nature of RS. HWE is characterized by the trigger of hot water, while bathing epilepsy is characterized by seizure provocation through normothermic water immersion. Appavu et al mentioned that an original form of bathing epilepsy is provoked by the removal of the body from the water.[118]

Water temperature and quantity, over and long-time exposure to hot water are important parameters in the provocation of seizures. Startle epilepsy, febrile seizures, and syncope should be considered in the differential diagnosis.

Although the frequency of patients with HWE was 0.6% among all epilepsy cases, HWE appears to have a high prevalence with 3.6%–6.9% in Southern India.[95] The high prevalence may be due to genetic overload, climate conditions, and also some cultural habits like usually bathing with high-temperature water.[89,87] Familial HWE patients with more than one affected member have been determined in 18% of Indian and in 10% of Turkish patients.[88,89] There are also isolated cases reported from different countries and small series from Turkey.[88–90,93,94] Interictal EEG is usually normal, but 20%–25% of the patient can have epileptiform abnormalities in the temporal region.[89,91,96] SPECT studies demonstrated ictal hypermetabolic uptake in the medial temporal structures and hypothalamus on the left in three and on the right in two patients, with spread to the opposite hemisphere.[97]

It is shown that decreasing the water temperature is effective in the management of HWE. However, 1/3 of the cases require treatment of AEDs such as carbamazepine, lamotrigine, phenytoin, phenobarbital, sodium valproate, oxcarbazepine, and levetiracetam.[89,90,98] Satishchandra et al recommended the usage of 5–10 mg of oral clobazam 1.5–2 hours prior to taking a bath.[1]

Reading epilepsy

Reading epilepsy is a rare syndrome that manifests with myoclonic jerks of the masticatory muscles. Involuntary jaw jerks or orofacial myoclonus, usually described as stiffness and clicking sensation may occur while reading and then spread to the limbs if reading continues.[99,100] It is usually seen in adolescence and young adulthood, which is after acquiring reading skills. There is a slight dominance in men (62%). However, interictal EEG is normal in 80% of patients where temporal paroxysmal discharges are present in 5%.[101] Visual stimulation and/or psychological activity during reading may precipitate the seizure, although the underlying mechanism is not entirely clear.[64] Ictal EEG findings are often imperceptible due to jaw muscle artifacts; usually bilateral sharp waves are detected in the temporoparietal lobe. Koutroumanidis et al pointed out a rare form of reading-induced seizure, which manifests in alexia with various degrees of dysphasia and unilateral focal EEG findings. This syndrome was predicted as a variant form of partial reading epilepsy.[102] Valproate or clonazepam controls the attacks in the most of the patients.[101]

Seizures induced by orgasm

Epileptic seizures triggered by sexual orgasm are very rare. In 1960, Hoenig and Hamilton reported the first case who was a 23-year-old woman. In 2006, six patients were described with different epileptic syndromes who experienced seizures after sexual intercourse and orgasm.[103] Female preponderance and involvement of right hemisphere were also noted.[104] Although it is difficult to exclude the effect of hyperventilation in these seizures, the absence of epileptiform activity in EEG recordings during hyperventilation and a certain delay after sexual intercourse are helpful to exclude such possible effect. The mechanisms of orgasm-related RS are not exactly disclosed as many other RS; however, reaching to the sexual climax or orgasm may be responsible for provoking already sensitized neurons within the network localized throughout various responsible centers of the brain that result in seizures.

Praxis-induced reflex seizures

Inoue et al emphasized the role of motor component in seizure provocation for first time introducing the term "praxis-induced epilepsy" where seizures are induced by complex, cognition-guided higher cortical function (arithmetic, decision making) accompanied by execution of movement (praxis) such as drawing, playing cards, chess, other board games or with a Rubik's cube.[105,64]

Seizures are usually of generalized type and may be part of JME.[76] Writing can trigger both praxis-induced and/or primary reading epilepsy.[106] It is suggested that these movements stimulate certain functional subsystems especially hyperexcitable and abnormally coupled with other areas, which are necessary for the critical mass needed for seizure initiation.[107] This significant network is often identified as a parietofrontotemporal network, which includes the intraparietal sulcus, the superior parietal sulcus, the inferior parietal lobule, the middle frontal gyrus, the premotor cortex, and the inferior frontal cortex.[108]

Conclusion

RS induced by different stimuli should be questioned during the evaluation of the patients with seizure disorders, and when necessary, stimulations during electrophysiological studies may be performed for correct diagnosis. When a significant mass of epileptogenic cortex is stimulated in response to a reflex activity, it can lead to generalized epileptiform activity or a clinical seizure via the participation and interaction of some cortical areas or of cortex and subcortical structures.[64] A careful history taking to identify environmental precipitating factors is essential to make a proper diagnosis of RE. Avoiding the specific trigger is particularly important for prevention of RS, and antiepileptic treatment is inevitable when combined with spontaneous seizures. Future studies

and increasing usage of advanced neuroimaging techniques will help to understand the complex network mechanisms underlying the pathophysiology and to develop preventive strategies for seizure control and therapy optimization.

Disclosure

The authors report no conflicts of interest in this work.

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