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UNDERSTANDING REFLEX EPILEPSY: TRIGGERS, MECHANISMS, AND MANAGEMENT

FAIYYAZ UR REHMAN[1], JAMIR PITTON RISSARDO[2], NIGAR HASHMI[3], HAN GUL[4] ANA LETÍCIA FORNARI CAPRARA[5]

[1]Department of Internal Medicine, Pak-Oman Hospital, Pasni 91300, Pakistan
[2]Neurology Department, Cooper University Hospital, NJ 08103, USA
[3]Department of Anesthesiology, Jinnah Hospital, Lahore 54550, Pakistan
[4]Department of Medicine, Teaching Hospital Turbat, Kech Pakistan
[5]Neurology Department, Cooper University Hospital, NJ 08103, USA

ABSTRACT

Reflex epilepsy, a distinctive subset of epileptic disorders, is characterized by seizures reliably triggered by specific stimuli or activities. This comprehensive literature review synthesizes current knowledge and recent advancements in understanding reflex epilepsy, shedding light on its diverse triggers, clinical manifestations, and underlying neurophysiological mechanisms. The review explores the vast array of triggers, encompassing visual stimuli, specific movements, cognitive processes, and sensory inputs, revealing the intricate interplay between external stimuli and epileptic events. Significant clinical heterogeneity is observed in reflex epilepsy, with manifestations ranging from focal motor seizures to alterations in consciousness. Advances in neuroimaging and electroencephalography have contributed valuable insights into the neurophysiological basis of reflex epilepsy, emphasizing cortical hyperexcitability and abnormalities in sensory and cognitive processing. Genetic and molecular studies have uncovered potential markers associated with reflex epilepsy, offering a glimpse into its hereditary aspects and paving the way for future diagnostic and therapeutic strategies. The literature underscores the psychosocial impact of reflex epilepsy on the quality of life of affected individuals, prompting the need for holistic, patient-centered care approaches.

KEYWORDS: reflex epilepsy, triggers, seizure, musicogenic epilepsy, reflex, photosensitivity.

INTRODUCTION

Definition of Reflex Epilepsy

Reflex epilepsy is a subtype of epilepsy characterized by seizures that are consistently triggered by specific external stimuli or activities. In contrast to spontaneous seizures, which can occur without any identifiable cause, reflex epilepsy seizures are provoked by specific triggers [1]. These triggers can vary among individuals, including visual and auditory stimuli, particular movements, or activities such as

reading. The defining feature of reflex epilepsy is the reproducibility of seizures in response to particular stimuli [2]. Common triggers for reflex epilepsy include flickering lights, specific patterns, certain types of music, reading, and various movements. Not everyone with epilepsy experiences reflex seizures, and the particular triggers can differ from person to person [3]. The mechanisms underlying reflex epilepsy involve abnormal patterns of neuronal activity in response to the triggering stimuli. Genetic factors and environmental influences may contribute to an individual's susceptibility to reflex seizures [4]. The diagnosis of reflex epilepsy often involves a thorough clinical evaluation, including a detailed medical history, neurological examination, and diagnostic tests such as electroencephalography (EEG) or video EEG. Identifying specific triggers is crucial for understanding and managing reflex epilepsy effectively.

Treatment approaches for reflex epilepsy typically involve avoiding known triggers whenever possible. Additionally, antiseizure medications may be prescribed to help control seizures. In some cases, behavioral therapies and lifestyle modifications may play a role in managing the condition. Reflex epilepsy is a diverse and complex phenomenon, and understanding its triggers and underlying mechanisms is essential for providing appropriate care and support to patients affected by this condition.

Introduction to the Concept of Seizures Triggered by Specific Stimuli

The concept of seizures triggered by specific stimuli is a distinctive aspect of a subset of epilepsy known as reflex epilepsy. Unlike spontaneous seizures that occur without an apparent cause, reflex epilepsy is characterized by the predictable and reproducible nature of seizures in response to particular external stimuli or activities [5]. This phenomenon highlights the intricate relationship between sensory input and the electrical excitability of the brain. In reflex epilepsy, seizures are induced by specific triggers that vary among individuals. Common triggers include visual stimuli like flickering lights or particular patterns, auditory stimuli such as certain types of music, and activities like reading or specific movements. The key feature is the consistent and reliable association between exposure to these triggers and the onset of seizures [6].

This concept underscores the dynamic interplay between external stimuli and the delicate balance of neuronal activity. The triggers provoke abnormal electrical discharges, leading to a cascade of events that culminate in a seizure. Understanding these specific triggers is crucial for both diagnosis and management, as it provides valuable insights into the underlying mechanisms of epilepsy in affected individuals.

The study of reflex epilepsy not only enhances our comprehension of the intricate workings of the brain but also has practical implications for individuals living with this condition. Recognizing and avoiding known triggers become integral components of managing reflex epilepsy, allowing for the development of personalized strategies to minimize the risk of seizures. In this way, the present study aims to review the literature about reflex epilepsy narratively.

General Classification of Epilepsy

Epilepsy is a neurological disorder characterized by recurrent seizures. The classification of epilepsy is based on the type of seizures and their origin in the brain. Three main types of epilepsy are focal epilepsy, generalized epilepsy, and unknown [7].

Focal epilepsy, also known as partial epilepsy, originates in a specific area or focus within one hemisphere of the brain. Seizures in focal epilepsy start in a localized region, and their manifestations depend on the location of the brain affected [8]. Focal seizures can be simple, involving isolated sensations or movements, or complex, affecting consciousness and leading to more complex behaviors. Focal epilepsy can result from various factors, including brain injuries, infections, tumors, or genetic predisposition. Electroencephalography (EEG) and imaging studies help identify the specific brain region involved.

Generalized epilepsy involves seizures that originate in and affect both hemispheres of the brain simultaneously [9]. Seizures in generalized epilepsy typically involve widespread neuronal activity from the onset. Generalized seizures can manifest as tonic-clonic (grand mal) seizures, absence (petit mal) seizures, myoclonic seizures, or atonic seizures. Genetic factors, brain abnormalities, or unknown causes may contribute to generalized epilepsy. EEG patterns characteristic of generalized seizures help differentiate them from focal seizures.

Reflex epilepsy is characterized by seizures that are consistently triggered by specific external stimuli or activities. External stimuli such as flashing lights, certain patterns, specific music, reading, or particular movements can trigger seizures in individuals with reflex epilepsy. Seizures in reflex epilepsy are similar to those in other forms of epilepsy but are reliably induced by specific triggers [10]. Genetic predisposition and abnormal neuronal responses to particular stimuli contribute to reflex epilepsy. Avoiding known triggers is a crucial aspect of managing reflex epilepsy, along with the use of antiseizure medications when necessary.

Reflex Epilepsy: An In-Depth Look

Historical Perspective on Reflex Epilepsy

Recognition of seizures triggered by specific stimuli dates back centuries. However, the understanding of these phenomena has evolved significantly over time. In ancient civilizations, seizures were often attributed to supernatural causes or seen as divine interventions. Specific triggers may not have been identified, and mystical explanations prevailed [11].

Some historical texts describe observations of seizures induced by specific activities or stimuli. For example, writings from ancient Greece and Rome refer to cases where individuals experienced seizures while reading or being exposed to particular visual patterns [12]. Hippocrates, often regarded as the father of medicine, made significant contributions to understanding epilepsy. While he did not specifically address reflex epilepsy, his writings acknowledged the variability of seizures and their potential association with external factors [13].

As medical knowledge advanced, there was a growing recognition of the diversity of epileptic seizures. Some physicians began documenting cases where seizures were consistently triggered by external stimuli such as light, sound, or specific activities. The early 20th century saw the advent of EEG, allowing for a more detailed examination of brain activity during seizures. This technological advancement played a pivotal role in identifying and classifying different types of epilepsy, including those triggered by specific stimuli. With the development of EEG and more sophisticated diagnostic tools in the mid-20th century, clinicians gained a deeper understanding of epilepsy subtypes. Reflex epilepsy was formally recognized as a distinct category, encompassing seizures consistently triggered by specific stimuli [14]. Further research and technological advancements in neuroimaging and genetic studies

enhanced the understanding of reflex epilepsy. Increased efforts to identify specific triggers, explore underlying mechanisms, and develop targeted therapeutic interventions.

Prevalence and Incidence Rates

Reflex epilepsy is considered a relatively rare subtype of epilepsy, and epidemiological data on its occurrence is limited compared to more common forms of epilepsy, such as focal or generalized epilepsy. The prevalence and incidence of epilepsy, in general, can vary widely based on factors such as geographical location, age group, and underlying causes. Reflex epilepsy, being a subset, is likely to have a lower prevalence compared to more common forms of epilepsy [15]

It's important to note that obtaining accurate prevalence and incidence rates for reflex epilepsy can be challenging due to several factors [16]. First, many patients with reflex epilepsy may not seek medical attention, especially if their seizures are infrequent or if they have identified and successfully avoided triggers. Second, reflex epilepsy can be challenging to diagnose accurately, and misclassification or underdiagnosis may occur. Third, reflex epilepsy encompasses various triggers, making it a heterogeneous category. Different triggers may have different prevalence rates. Fourth, given that reflex epilepsy is less common, there may be fewer dedicated studies on its prevalence compared to more prevalent forms of epilepsy.

Identification of Triggers

Identifying triggers in reflex epilepsy is a crucial aspect of managing the condition effectively. Triggers are specific stimuli or activities that consistently lead to the onset of seizures in individuals with reflex epilepsy. A detailed patient history is fundamental to understanding the particular triggers. Clinicians inquire about the circumstances surrounding each seizure, focusing on activities, environmental factors, or stimuli preceding the events.

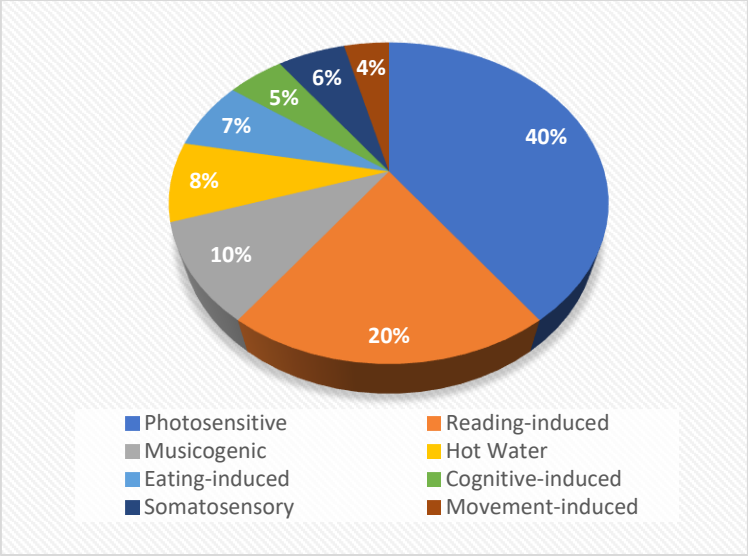

Figure 1 *(Prevalence of Different Types of Reflex Epilepsy.)*

The pie chart illustrates the percentage distribution of various reflex epilepsy types. Photosensitive epilepsy accounts for the largest proportion, followed by reading-induced epilepsy and other less common types. This highlights the heterogeneity in reflex epilepsy presentations.

Encouraging individuals with reflex epilepsy to maintain a seizure diary can be helpful. Recording details such as the time, location, activities, and any noticeable stimuli before a seizure can provide valuable insights. Direct interviews with patients, family members, or caregivers can reveal patterns and associations between certain activities or stimuli and the occurrence of seizures. Open- ended questions about daily routines and experiences are essential.

Video electroencephalography (EEG) monitoring allows for simultaneously recording the patient's behavior and brain activity during seizures. This method helps correlate specific behaviors or activities with corresponding changes in EEG patterns. In a controlled medical setting, clinicians may use activation procedures to provoke seizures intentionally. This can involve exposing the individual to known triggers under close monitoring to observe the resulting seizure activity.

Structural and functional neuroimaging studies, such as magnetic resonance imaging (MRI) and functional MRI (fMRI), can provide additional information about the brain's response to specific stimuli and potential areas of hyperexcitability. Performing EEG studies while exposing individuals to potential triggers can help capture real-time changes in brain activity associated with particular stimuli. In some cases, genetic testing may reveal underlying genetic factors contributing to the susceptibility to reflex seizures. Genetic information can inform the understanding of the disorder and potential triggers.

Photosensitive Epilepsy

Definition and Epidemiology

Photosensitive epilepsy, also known as visually induced epilepsy, is a specific form of reflex epilepsy where visual stimuli trigger seizures [17]. These stimuli typically involve light patterns, such as flickering lights, repetitive geometric patterns, or specific color contrasts. Photosensitive epilepsy is characterized by a consistent and reproducible response to these visual triggers, leading to seizures in susceptible individuals. Photosensitive epilepsy is relatively rare, accounting for a small percentage of overall epilepsy cases. It often manifests in adolescence or early adulthood, and there may be a slightly higher prevalence among females.

Types of Visual Stimuli That Trigger Seizures

Rapid changes in light intensity, especially with a high contrast, can trigger seizures. This includes flashing lights, strobe lights, or rapidly changing visual patterns. Some individuals with photosensitive epilepsy may be sensitive to specific geometric patterns, such as stripes or checkerboard designs [18]. Specific color combinations or contrasts may provoke seizures in susceptible individuals.

Pathophysiology

The pathophysiology of photosensitive epilepsy involves complex interactions between the visual stimuli, the visual processing pathways in the brain, and the underlying neuronal network susceptibility. While the precise mechanisms are not fully understood, research suggests that photosensitive epilepsy is associated with abnormal responses in the visual cortex and abnormal synchronization of neuronal activity [19].

Photosensitive epilepsy is believed to involve abnormal neuronal excitability in response to specific visual stimuli. Certain individuals may be more susceptible to rapid changes in light intensity or particular patterns. The thalamus and the cortex play crucial roles in processing visual information [20].

Research suggests that abnormal interactions within thalamocortical circuits may contribute to the development of photosensitivity.

Abnormalities in the balance of neurotransmitters, such as gamma-aminobutyric acid (GABA) and glutamate, may contribute to cortical hyperexcitability [21]. This hyperexcitability can increase the likelihood of synchronous and abnormal firing of neurons in response to visual stimuli. Photosensitive epilepsy is associated with abnormal synchronization of neuronal activity, particularly in the visual cortex. The synchronized firing of neurons can lead to the generation and propagation of epileptic discharges [22].

Genetic factors play a role in photosensitivity [23]. Some individuals may have a genetic predisposition to abnormal responses to visual stimuli. Specific genetic mutations or variations may contribute to an individual's susceptibility to photosensitive seizures. Dysfunction of ion channels, particularly those involved in regulating neuronal excitability, may contribute to photosensitivity. Changes in the balance of sodium, potassium, and calcium ions can influence the threshold for neuronal firing [24].

The visual processing pathways from the retina to the visual cortex are critical in normal visual perception. In photosensitive epilepsy, there may be alterations in the way visual information is processed, leading to an increased risk of seizures in response to certain stimuli. Specific visual stimuli can activate seizure networks in susceptible individuals. The activation of these networks leads to the initiation and propagation of epileptic discharges, resulting in seizures. Cortical spreading depression, a phenomenon involving a wave of depolarization followed by a period of decreased neuronal activity, has been implicated in photosensitive epilepsy [25]. It may contribute to the generation of seizures in response to visual stimuli.

Clinical Manifestations and Seizure Characteristics

Photosensitive seizures can manifest as generalized tonic-clonic seizures, absence seizures, myoclonic seizures, or focal seizures with or without impairment of awareness [26]. Seizures may involve motor movements, altered consciousness, or other sensory experiences.

Management

The primary strategy is to avoid known triggers. This may involve lifestyle modifications to minimize exposure to flickering lights or specific visual patterns. Some individuals may benefit from using screen filters or wearing tinted glasses to reduce the intensity of visual stimuli [27]. In some cases, antiseizure medications may be prescribed to help control seizures. Individuals with photosensitive epilepsy may face restrictions on activities such as driving, mainly if their seizures are triggered by visual stimuli encountered on the road [25]. Public awareness campaigns often promote warnings about potential seizure-inducing visual stimuli, such as those in video games, movies, or public events.

Other Triggers in Reflex Epilepsy

Musicogenic Epilepsy

Musicogenic epilepsy is a rare form of reflex epilepsy in which specific musical stimuli trigger seizures. Individuals with musicogenic epilepsy experience seizures consistently in response to certain musical patterns, rhythms, or specific musical pieces [28]. The triggering musical elements can vary widely among affected individuals. Seizures in musicogenic epilepsy can be triggered by specific musical patterns, melodies, or rhythms. The triggers may involve specific frequencies, tones, or harmonies [29].

Specific musical instruments or a combination of instruments may sometimes act as triggers. The specific musical elements that trigger seizures can vary from person to person. Seizures in musicogenic epilepsy can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during seizures may include altered consciousness, motor movements, sensory experiences, or autonomic changes [30]. One key characteristic is the consistent association between exposure to specific musical stimuli and the occurrence of seizures.

The pathophysiology of musicogenic epilepsy is not fully understood, given the rarity of the condition and the complexity of the brain's response to auditory stimuli. However, researchers have explored various factors that may contribute to the development of musicogenic seizures. Musicogenic epilepsy involves abnormal processing of auditory stimuli in the brain. Specific neural networks responsible for auditory processing may demonstrate hyperexcitability or abnormal synchronization in response to certain musical elements. The temporal lobes, especially the superior temporal gyrus, are crucial for auditory processing [31]. Abnormalities in these regions may contribute to the susceptibility of individuals with musicogenic epilepsy to seizures triggered by music. Music involves auditory processing and sensory-motor integration, as individuals may perceive and respond to rhythmic or melodic patterns [32]. Dysfunction in the integration of sensory and motor information may play a role in the generation of seizures.

The limbic system, involved in emotion and memory, may be implicated in musicogenic epilepsy [33]. Emotional responses to music, combined with abnormal limbic system activation, could contribute to the initiation of seizures. Like other forms of epilepsy, musicogenic epilepsy may involve abnormal neuronal hyperexcitability and synchronization. Specific patterns or frequencies in music may act as triggers, leading to the synchronous firing of neurons and the generation of epileptic discharges. Genetic predisposition may contribute to an individual's susceptibility to musicogenic epilepsy. Genetic variations that influence neuronal excitability, auditory processing, or the function of ion channels may play a role. The specific elements in music that trigger seizures can vary widely among individuals with musicogenic epilepsy. This variability suggests that individualized factors may influence the susceptibility to certain musical stimuli. Exposure to specific musical stimuli may activate seizure networks in susceptible individuals. The initiation and propagation of epileptic discharges following exposure to triggering music could involve aberrant activation of neuronal networks. Cortical spreading depression, characterized by a wave of neuronal depolarization followed by decreased activity, has been implicated in some forms of epilepsy. It may also play a role in musicogenic epilepsy, influencing the generation of seizures. Emotional responses to music, coupled with abnormal brain responses, may create a unique interplay that contributes to the generation of seizures. The emotional content of music may modulate the excitability of brain regions involved in epilepsy [34].

The primary strategy for managing musicogenic epilepsy is to avoid exposure to specific musical triggers. Cognitive-behavioral therapy may help individuals develop coping strategies and modify responses to triggering stimuli [35]. In some cases, antiseizure medications may be prescribed to help control seizures. Musicogenic epilepsy can have implications for an individual's daily life, influencing choices related to leisure activities, social interactions, and employment.

Reading-Induced Epilepsy

Reading-induced epilepsy, also known as reading epilepsy or reflex epilepsy triggered by reading, is a rare form of epilepsy characterized by seizures specifically provoked by reading or exposure to written

text [36]. The seizures are often consistently triggered by visual stimuli associated with reading. Seizures are reliably triggered by reading or exposure to written text. The specific visual patterns associated with reading, such as eye movements across lines of text, can provoke seizures [37].

Reading-induced seizures can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes. Onset can occur at any age but is often reported in adolescence or early adulthood [38].

The pathophysiology of reading-induced epilepsy is not fully understood, and the mechanisms that lead to seizures specifically triggered by reading activities remain complex and multifaceted. Reading-induced epilepsy involves abnormalities in visual information processing, particularly related to reading. Reading involves complex visual patterns, including eye movements, text perception, and comprehension, which may trigger abnormal responses in susceptible individuals [39]. Similar to other forms of reflex epilepsy, reading-induced epilepsy may be associated with abnormal neuronal hyperexcitability. Specific neural networks responsible for visual processing may become excessively excitable in response to the visual stimuli related to reading.

Abnormalities in the occipital and temporal lobes of the brain, regions crucial for visual processing and language comprehension, may be implicated in reading-induced epilepsy [40]. Disturbances in these areas could contribute to generating seizures triggered by reading. Synchronizing neuronal activity in the cortex is essential for normal brain function. In reading- induced epilepsy, there may be abnormalities in the synchronization of neural circuits, leading to the initiation and propagation of epileptic discharges. Genetic predisposition may contribute to an individual's susceptibility to reading-induced epilepsy. Specific genetic variations affecting visual processing, neuronal excitability, or ion channel function may play a role.

The specific patterns or elements within reading that trigger seizures can vary among individuals. This variability suggests that individualized factors may influence susceptibility to reading-induced seizures. Exposure to specific visual stimuli associated with reading may activate seizure networks in susceptible individuals. The initiation and propagation of epileptic discharges following exposure to these stimuli could involve aberrant activation of neuronal networks. Cognitive and emotional factors related to reading, such as attention, comprehension, and emotional responses to the content, may modulate the excitability of brain regions involved in epilepsy. Abnormal interactions with these factors could contribute to the triggering of seizures. The coordination of eye movements during reading involves complex neural processes [41]. Abnormalities in the interaction between eye movements and visual processing may be relevant to the pathophysiology of reading-induced epilepsy.

The primary strategy for managing reading-induced epilepsy is to avoid exposure to reading stimuli. Individuals may need to modify their reading habits or find alternative ways to access information. Cognitive-behavioral therapy may help individuals develop coping strategies and change responses to reading triggers. In some cases, antiseizure medications may be prescribed to help control seizures.

Eating-Induced Epilepsy

Eating-induced epilepsy, also known as gustatory epilepsy or reflex epilepsy triggered by eating, is a rare form of epilepsy characterized by seizures specifically provoked by eating or tasting certain foods [42]. Gustatory stimuli, such as chewing, swallowing, or exposure to specific tastes, consistently trigger

seizures. Seizures are reliably triggered by eating or exposure to particular tastes. The specific gustatory stimuli associated with eating, such as the taste or texture of food, can provoke seizures [43]. Eating-induced seizures can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes. Onset can occur at any age but is often reported in adolescence or early adulthood.

Abnormalities in the processing of gustatory (taste-related) information involving regions such as the insula and frontal operculum may be implicated in eating-induced epilepsy [44]. Similar to other forms of reflex epilepsy, eating-induced epilepsy may involve abnormal neuronal hyperexcitability in response to specific gustatory stimuli.

The primary strategy for managing eating-induced epilepsy is to avoid exposure to specific gustatory triggers. Individuals may need to modify their diet or eating habits to minimize the risk of seizures. Cognitive-behavioral therapy may help individuals develop coping strategies and change responses to gustatory triggers. In some cases, antiseizure medications may be prescribed to help control seizures.

Hot Water Epilepsy

Hot water epilepsy is a rare form of reflex epilepsy triggered by exposure to hot water [45]. Seizures in this condition are consistently provoked by contact with hot water, typically during bathing or showering. The exact mechanisms underlying hot water epilepsy are not fully understood, but it is believed to involve specific temperature-related stimuli acting as triggers [46]. Seizures are reliably triggered by exposure to hot water. This can occur during activities like bathing, showering, or any other situation where the individual comes into contact with hot water. Hot water-induced seizures can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes. Onset can occur at any age, and there may be individual variability in the age of onset.

The pathophysiology of hot water epilepsy, also known as water-induced reflex epilepsy, involves the specific triggering of seizures by exposure to hot water. While the exact mechanisms are not fully understood, it is believed to be related to temperature-related stimuli acting as triggers for abnormal neuronal activity [47]. The core feature of hot water epilepsy is seizures consistently triggered by exposure to hot water. Temperature receptors in the skin and peripheral nerves that convey temperature information play a role in sensing and transmitting signals related to hot water exposure. Abnormal responses in thermoregulatory centers in the hypothalamus may contribute to generating seizures in response to hot water exposure [48]. Like other forms of reflex epilepsy, hot water epilepsy may involve abnormal neuronal hyperexcitability. While specific genetic factors associated with hot water epilepsy are not well-defined, genetic predisposition may play a role in an individual's susceptibility to reflex seizures triggered by temperature-related stimuli.The primary strategy for managing hot water epilepsy is to avoid exposure to hot water. Individuals may need to modify their bathing habits or use alternative methods for personal hygiene to minimize the risk of seizures.

Somatosensory-Evoked Reflex Seizures

Somatosensory-evoked reflex seizures refer to seizures that are triggered by specific somatosensory stimuli [49]. The somatosensory system involves the perception of sensations related to touch, pressure, vibration, and proprioception. In reflex epilepsy, seizures are induced by specific sensory inputs, and

when it comes to somatosensory-evoked seizures, triggers are related to tactile or proprioceptive stimuli [50].

Specific somatosensory stimuli consistently trigger seizures. These stimuli may include tactile sensations, pressure, vibration, or movements of particular body parts. Somatosensory-evoked reflex seizures can manifest as focal, generalized, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes, all triggered by somatosensory stimuli. Seizures may be triggered by touch, pressure, or other tactile inputs [51]. For example, brushing the skin, certain textures, or specific types of clothing may act as triggers. Movements or positions of particular body parts, such as limb movements or joint positions, may also induce seizures in susceptible individuals. Onset can occur at any age, and the age of onset may vary among individuals with somatosensory-evoked reflex seizures.

The pathophysiology involves abnormal processing of somatosensory information in the central nervous system [52]. Specific neural circuits involved in sensory processing may become hyperexcitable, generating seizures in response to somatosensory stimuli. As with other forms of reflex epilepsy, somatosensory-evoked reflex seizures may be associated with abnormal neuronal hyperexcitability in response to specific sensory inputs.

The primary strategy for managing somatosensory-evoked reflex seizures is to avoid exposure to specific triggers. Individuals may need to modify their environment or behavior to minimize the risk of seizures. Cognitive-behavioral therapy may help individuals develop coping strategies and change responses to somatosensory triggers. In some cases, antiseizure medications may be prescribed to help control seizures.

Proprioceptive-Induced Reflex Seizures

Proprioceptive-induced reflex seizures refer to seizures that are triggered by specific proprioceptive stimuli [53]. Proprioception involves the sense of the body's position, movement, and spatial orientation, and in reflex epilepsy, seizures are induced by particular stimuli related to proprioceptive input [54]. These stimuli may include movements or positions of specific body parts, joint movements, or changes in spatial orientation. Proprioceptive-induced reflex seizures can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes, all triggered by proprioceptive stimuli. Seizures may be triggered by specific joint movements or changes in joint positions [55]. For example, particular movements of the limbs or neck movements may act as triggers. Alterations in the body's spatial orientation, such as standing up or lying down, may also induce seizures in susceptible individuals. Onset can occur at any age, and the age of onset may vary among individuals with proprioceptive-induced reflex seizures.

The pathophysiology involves abnormal processing of proprioceptive information in the central nervous system. Specific neural circuits engaged in proprioception may become hyperexcitable, generating seizures in response to proprioceptive stimuli [56]. As with other forms of reflex epilepsy, proprioceptive-induced reflex seizures may be associated with abnormal neuronal hyperexcitability in response to specific sensory inputs.

The primary strategy for managing proprioceptive-induced reflex seizures is to avoid exposure to specific triggers. Individuals may need to modify their activities or environment to minimize the risk of seizures.

Orgasm-Induced Seizures

Orgasm-induced seizures, also known as orgasmic epilepsy, is a rare form of reflex epilepsy where seizures are triggered by sexual activity, specifically during orgasm [57]. This unique phenomenon involves the convergence of the physiological processes associated with sexual arousal and the abnormal neuronal activity characteristic of epilepsy. Seizures are consistently triggered by the physiological processes related to sexual climax or orgasm [58]. Orgasm-induced seizures can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes, all occurring in conjunction with orgasm. Onset can occur at any age, and the age of onset may vary among individuals with orgasm-induced seizures.

The pathophysiology of orgasm-induced seizures, also known as orgasmic epilepsy, is not fully understood, and the mechanisms that lead to seizures triggered explicitly by sexual activity remain complex. However, it is believed to involve the convergence of the neurophysiological response to sexual arousal and the abnormal neuronal activity characteristic of epilepsy. Sexual arousal involves complex hormonal changes, including releasing neurotransmitters such as dopamine, serotonin, and oxytocin [59]. These changes contribute to the pleasurable sensations and the physiological response leading to orgasm.

Similar to other forms of reflex epilepsy, orgasm-induced seizures may be associated with abnormal cortical excitability. The limbic system, which plays a crucial role in emotional and sexual responses, may be involved. Abnormal activity in limbic structures, such as the hippocampus and amygdala, could contribute to the manifestation of seizures during sexual activity [60]. Neurotransmitters like dopamine and serotonin, which are involved in the reward and pleasure pathways, may have a role in the pathophysiology. Dysregulation in these neurotransmitter systems could contribute to abnormal neuronal excitability.

In some cases, antiseizure medications may be prescribed to help control seizures. The choice of drugs is typically based on the individual's seizure type and medical history. Cognitive-behavioral therapy may be employed to help individuals and their partners manage the psychological aspects of living with orgasm-induced seizures.

Acute-provoked reflex seizures induced by thinking

Reflex seizures triggered by thinking, also known as cognitive-induced reflex seizures, are a rare form of epilepsy where specific cognitive activities or mental processes provoke seizures [61]. Certain cognitive tasks, thoughts, or mental activities can trigger seizures in these cases. Specific cognitive activities, thoughts, or mental processes consistently trigger seizures. These triggers can vary widely among individuals. Cognitive-induced reflex seizures can manifest as focal seizures, generalized seizures, or a combination of both. Various mental processes, such as specific thoughts, mental imagery, or cognitive tasks, may trigger seizures. The triggers can be specific to the individual's experiences. Onset can occur at any age, and the age of onset may vary among individuals with cognitive-induced reflex seizures [62].

The pathophysiology involves abnormal processing of cognitive information in the central nervous system. Specific neural circuits involved in cognitive tasks may become hyperexcitable, generating seizures in response to cognitive triggers. Similar to other forms of reflex epilepsy, cognitive-induced reflex seizures may be associated with abnormal neuronal hyperexcitability in response to specific cognitive inputs.

Movement-Induced Reflex Seizures

Epilepsy with seizures induced by movement is a type of reflex epilepsy where specific voluntary or involuntary movements trigger seizures [63]. In this condition, the act of moving or particular types of movements can provoke epileptic seizures. Specific voluntary or involuntary movements consistently trigger seizures. These movements include various motor activities such as walking, running, or gestures [64]. Movement-induced reflex seizures can manifest as focal seizures, generalized seizures, or a combination of both. The symptoms during a seizure may include altered consciousness, motor movements, sensory experiences, or autonomic changes, all occurring in response to movement triggers [65]. Seizures may be triggered by a wide range of movements, from gross motor activities like walking or jumping to finer motor activities like writing or specific hand gestures. Onset can occur at any age, and the age of onset may vary among individuals with movement-induced reflex seizures.

The pathophysiology involves abnormal processing of motor-related information in the central nervous system [66]. Specific neural circuits involved in motor planning and execution may become hyperexcitable, generating seizures in response to movement triggers. Similar to other forms of reflex epilepsy, movement-induced reflex seizures may be associated with abnormal neuronal hyperexcitability in response to specific motor inputs.

Mechanisms of Reflex Epilepsy

The neurophysiological processes underlying reflex epilepsy involve abnormal neuronal activity and excitability in response to specific stimuli or triggers. Reflex epilepsy is characterized by seizures that are reliably triggered by specific sensory or cognitive stimuli. Understanding the neurophysiological mechanisms involved is crucial for diagnosis, treatment, and management.

In reflex epilepsy, specific sensory stimuli act as triggers for seizures. These stimuli include visual, auditory, tactile, gustatory, or proprioceptive inputs [67]. Abnormalities in the processing of sensory information within neural pathways contribute to the initiation of seizures. In certain types of reflex epilepsy, cognitive processes or mental activities can act as triggers. This may involve abnormal neuronal activity in brain areas responsible for cognitive functions. Reflex epilepsy is often associated with cortical hyperexcitability, where the neurons in the cerebral cortex become more prone to generating epileptic discharges. The threshold for initiating a seizure is reduced, making individuals more susceptible to seizures in response to specific triggers.

Different forms of reflex epilepsy may involve specific neural circuits or networks that respond aberrantly to particular stimuli. For example, photosensitive epilepsy involves abnormal responses in the visual cortex. The thalamus plays a crucial role in relaying sensory information to the cortex [68]. Dysregulation in thalamocortical circuits may contribute to generating seizures in response to specific sensory stimuli. Genetic factors may contribute to an individual's predisposition to reflex epilepsy. Specific genetic mutations or variations could influence the development of abnormal neuronal pathways. Dysregulation of neurotransmitters, such as gamma-aminobutyric acid (GABA) and

glutamate, may contribute to altered excitability in the brain, making individuals more susceptible to reflex seizures.

The limbic system, involved in emotional and memory processing, may play a role in certain types of reflex epilepsy [69]. Emotional states or memories associated with specific stimuli may trigger seizures. Maladaptive neuroplastic changes in response to particular stimuli may contribute to the development and maintenance of reflex epilepsy. This involves alterations in the strength and connectivity of neuronal circuits.

Management

The management and treatment of reflex epilepsy involve a combination of strategies to reduce the frequency and impact of seizures triggered by specific stimuli. A thorough evaluation with a neurologist is crucial to identify particular triggers that provoke seizures. This involves obtaining a detailed history of seizure episodes and potential triggering factors.

Video electroencephalography (EEG) monitoring is often employed to capture seizures in real time and correlate them with specific triggers. This helps confirm the reflex nature of seizures. The primary strategy is to minimize exposure to known triggers. Individuals with reflex epilepsy may need to make lifestyle adjustments to avoid specific stimuli that provoke seizures.

CBT may be beneficial for individuals to develop coping strategies and modify their responses to triggering stimuli. This therapeutic approach addresses the emotional and psychological aspects of living with epilepsy [70]. In some cases, antiseizure medications may be prescribed to help control seizures. The choice of medication depends on the type of reflex epilepsy and individual patient characteristics.

Treatment plans should be individualized based on the specific triggers, seizure types, and the patient's overall health. For some instances of refractory epilepsy, implantable devices like VNS or RNS may be considered [71]. In cases where seizures are not controlled with medications and lifestyle modifications, epilepsy surgery may be considered.

Future Directions

Future studies in reflex epilepsy should focus on advancing our understanding of the underlying mechanisms, improving diagnostic methods, and developing targeted therapeutic interventions. The development and application of advanced neuroimaging technologies, such as functional magnetic resonance imaging (fMRI) and magnetoencephalography (MEG), can provide more detailed insights into the neurophysiological processes underlying reflex epilepsy. Research may aim to identify specific genetic markers associated with reflex epilepsy. Understanding the genetic basis could improve risk prediction and personalized treatment approaches. The search for reliable biomarkers associated with reflex epilepsy could enhance diagnostic accuracy and provide objective measures for monitoring treatment response. Utilizing quantitative EEG measures and machine learning algorithms may contribute to a more precise characterization of seizure patterns, aiding in diagnosis and understanding individual variability.

Investigating the cognitive and behavioral aspects of reflex epilepsy could provide insights into the interplay between mental processes and seizure generation. Further subtyping of reflex epilepsies based on specific triggers and clinical characteristics may improve our ability to tailor treatment

strategies for different subgroups. Developing targeted therapies based on the particular triggers and underlying pathophysiology of reflex epilepsy may improve treatment outcomes while minimizing side effects. Future studies in reflex epilepsy should aim to bridge gaps in our understanding of the disorder, ultimately leading to improved diagnosis, treatment options, and quality of life for individuals living with reflex epilepsy.

CONCLUSIONS

In conclusion, reflex epilepsy is characterized by seizures that are reliably triggered by specific stimuli, encompassing a broad range of sensory and cognitive triggers. Reflex epilepsy exhibits a remarkable diversity of triggers, including visual stimuli, particular movements, mental processes, and sensory inputs. Understanding the array of triggers is essential for accurate diagnosis and personalized management. The clinical presentation of reflex epilepsy varies widely, encompassing focal and generalized seizures, with manifestations ranging from motor symptoms to alterations in consciousness. This clinical heterogeneity underscores the complexity of reflex epilepsies. Ongoing genetic and molecular studies are uncovering potential genetic markers associated with reflex epilepsy. These insights contribute to our understanding of the hereditary aspects of the disorder and may inform future diagnostic and therapeutic strategies.

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Author Contributions

Rehman FU: Literature Review, Data Synthesis & Interpretation, Writing – Review & Editing, Writing - Final Approval of the Manuscript, Critical Review
Rissardo JP: Conceptualization, Literature Review, Data Synthesis & Interpretation, Writing – Original Draft, Writing – Final Approval of the Manuscript
Hashmi N: Methodology, Writing – Review & Editing, Visualization.
Gul H: Literature Review, Writing – Review & Editing, Data Curation,
Caprara ALF: Conceptualization, Methodology, Validation, Visualization, Supervision, Project Administration

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REFERENCES

1. Irmen F, Wehner T, Lemieux L. Do Reflex Seizures and Spontaneous Seizures Form a Continuum? - Triggering Factors and Possible Common Mechanisms. Seizure. 2015; 25:72-79. doi: 10.1016/j.seizure.2014.12.006.
2. Illingworth JL, Ring H. Conceptual Distinctions between Reflex and Nonreflex Precipitated Seizures in the Epilepsies: A Systematic Review of Definitions Employed in the Research Literature. Epilepsia. 2013; 54:2036-2047. doi:10.1111/epi.12340.
3. Okudan ZV, Özkara Ç. Reflex Epilepsy: Triggers and Management Strategies. Neuropsychiatr Dis Treat. 2018; 14:327-337. doi:10.2147/NDT.S107669.
4. Aird RB. The Importance of Seizure-Inducing Factors in the Control of Refractory Forms of Epilepsy. Epilepsia. 1983; 24:567-583. doi:10.1111/j.1528-1157. 1983.tb03421. x.
5. Engel J. Seizures and Epilepsy. Oxford University Press; 2013.
6. Fisher RS, Harding G, Erba G, Barkley GL, Wilkins A. Photic- and Pattern-Induced Seizures: A Review for the Epilepsy Foundation of America Working Group. Epilepsia. 2005; 46:1426-1441. doi:10.1111/j.1528-1167.2005.31405. x.
7. Falco-Walter J. Epilepsy-Definition, Classification, Pathophysiology, and Epidemiology. Semin Neurol. 2020; 40:617-623. doi:10.1055/s-0040-1718719.
8. Anwar H, Khan QU, Nadeem N, Pervaiz I, Ali M, Cheema FF. Epileptic Seizures. Discoveries (Craiova). 2020;8: e110. doi:10.15190/d.2020.7.
9. Gloor P, Fariello RG. Generalized Epilepsy: Some of Its Cellular Mechanisms Differ from Those of Focal Epilepsy. Trends Neurosci. 1988; 11:63-68. doi:10.1016/0166-2236(88)90166-x.
10. Hanif S, Musick ST. Reflex Epilepsy. Aging Dis. 2021; 12:1010-1020. doi:10.14336/AD.2021.0216.
11. Bou Nasif M, Koubeissi M, Azar NJ. Epilepsy - from Mysticism to Science. Rev Neurol (Paris). 2021; 177:1047-1058. doi: 10.1016/j.neurol.2021.01.021.
12. Panteliadis CP, Vassilyadi P, Fehlert J, Hagel C. Historical Documents on Epilepsy: From Antiquity through the 20th Century. Brain Dev. 2017; 39:457-463. doi: 10.1016/j.braindev.2017.02.002.
13. Bobkov I. About Reflex Epilepsy. Kazan Med J. 1938; 34:309-314.
14. Duvivier EH, Pollack Jr CV. Seizures. In: Rosen's Emergency Medicine: Concepts and Clinical Practice. 7th ed. Philadelphia, Pa: Mosby Elsevier; 2009.
15. Moshé SL, Perucca E, Ryvlin P, Tomson T. Epilepsy: New Advances. Lancet. 2015; 385:884-898. doi:10.1016/S0140-6736(14)60456-6.
16. Abramovici S, Bagić A. Epidemiology of Epilepsy. Handb Clin Neurol. 2016; 138:159-171. doi:10.1016/B978-0-12-802973-2.00010-0.
17. Fisher RS, Acharya JN, Baumer FM, French JA, Parisi P, Solodar JH, et al. Visually Sensitive Seizures: An Updated Review by the Epilepsy Foundation. Epilepsia. 2022; 63:739-768. doi:10.1111/epi.17175.
18. Covanis A, Solodar J. Photosensitive and Pattern-Sensitive Epilepsy: A Guide for Patients and Caregivers. In: The Importance of Photosensitivity for Epilepsy. 2021;393-406.
19. Wilkins AJ, Bonanni P, Porciatti V, Guerrini R. Physiology of Human Photosensitivity. Epilepsia. 2004;45 Suppl 1:7-13. doi:10.1111/j.0013-9580.2004.451009. x.

20. Vaudano AE, Ruggieri A, Avanzini P, Gessaroli G, Cantalupo G, Coppola A, et al. Photosensitive Epilepsy Is Associated with Reduced Inhibition of Alpha Rhythm Generating Networks. Brain. 2017; 140:981-997. doi:10.1093/brain/awx009.
21. Bahi-Buisson N, El Sabbagh S, Soufflet C, Escande F, Boddaert N, Valayannopoulos V, et al. Myoclonic Absence Epilepsy with Photosensitivity and a Gain of Function Mutation in Glutamate Dehydrogenase. Seizure. 2008; 17:658-664. doi: 10.1016/j.seizure.2008.01.005.
22. Martins da Silva A, Leal B. Photosensitivity and Epilepsy: Current Concepts and Perspectives-A Narrative Review. Seizure. 2017; 50:209-218. doi: 10.1016/j.seizure.2017.04.001.
23. Stephani U, Tauer U, Koeleman B, Pinto D, Neubauer BA, Lindhout D. Genetics of Photosensitivity (Photoparoxysmal Response): A Review. Epilepsia. 2004;45 Suppl 1:19-23. doi:10.1111/j.0013-9580.2004.451008. x.
24. Poleon S, Szaflarski JP. Photosensitivity in Generalized Epilepsies. Epilepsy Behav. 2017; 68:225-233. doi: 10.1016/j.yebeh.2016.10.040.
25. 25, Kasteleijn-Nolst Trenité DGA, Verrotti A, Di Fonzo A, Cantonetti L, Bruschi R, Chiarelli F, et al. Headache, Epilepsy and Photosensitivity: How Are They Connected? J Headache Pain. 2010; 11:469-476. doi:10.1007/s10194-010-0229-9.
26. Xue J, Gong P, Yang H, Liu X, Jiang Y, Zhang Y, et al. Genetic (Idiopathic) Epilepsy with Photosensitive Seizures Includes Features of Both Focal and Generalized Seizures. Sci Rep. 2018; 8:6254. doi:10.1038/s41598-018-24644-0.
27. Takahashi Y, Sato T, Goto K, Fujino M, Fujiwara T, Yamaga M, et al. Optical Filters Inhibiting Television-Induced Photosensitive Seizures. Neurology. 2001; 57:1767-1773. doi:10.1212/wnl.57.10.1767.
28. Maguire MJ. Music and Epilepsy: A Critical Review. Epilepsia. 2012; 53:947-961. doi:10.1111/j.1528-1167.2012.03523. x.
29. Maguire MJ. Wired for Sound: The Effect of Sound on the Epileptic Brain. Seizure. 2022; 102:22-31. doi: 10.1016/j.seizure.2022.09.016.
30. Critchley M. Musicogenic Epilepsy. In: Music and the Brain. Elsevier; 1977. p. 344-353.
31. Stern J. Musicogenic Epilepsy. In: Handbook of Clinical Neurology. Elsevier; 2015. p. 469-477.
32. Trimble MR, Hesdorffer D, Hećimović H, Osborne N. Personalised Music as a Treatment for Epilepsy. Epilepsy Behav. 2024; 156:109829. doi: 10.1016/j.yebeh.2024.109829.
33. Vizioli R. Musicogenic Epilepsy. Int J Neurosci. 1989; 47:159-164. doi:10.3109/00207458908987428.
34. Pelliccia V, Villani F, Gozzo F, Gnatkovsky V. Musicogenic Epilepsy: Systematic Review of the Literature. Epilepsy Behav. 2013; 28:313-317. doi: 10.1016/j.yebeh.2013.08.024.
35. Dawit, S., & Crepeau, A.Z. (2020). When drugs do not work: Alternatives to antiseizure medications. Current Neurology and Neuroscience Reports, 20, 37. https://doi.org/10.1007/s11910-020-01061-3
36. Koutroumanidis, M., Koepp, M.J., Richardson, M.P., Camfield, C., Agathonikou, A., Ried, S., Papadimitriou, A., Plant, G.T., Duncan, J.S., & Panayiotopoulos, C.P. (1998). The variants of reading epilepsy: A clinical and video-EEG study of 17 patients with reading-induced seizures. Brain, 121(Pt 8), 1409–1427. https://doi.org/10.1093/brain/121.8.1409

37. Safi, D., Lassonde, M., Nguyen, D.K., Denault, C., Macoir, J., Rouleau, I., Béland, R. (2011). Reflex reading epilepsy: Effect of linguistic characteristics on spike frequency. Epilepsy & Behavior, 20, 659–667. https://doi.org/10.1016/j.yebeh.2011.01.030
38. Koepp, M.J. (2011). Primary reading epilepsy. In The causes of epilepsy: Common and uncommon causes in adults and children (pp. 700–702). Cambridge University Press.
39. Gavaret, M., Guedj, E., Koessler, L., Trébuchon-Da Fonseca, A., Aubert, S., Mundler, O., Chauvel, P., & Bartolomei, F. (2010). Reading epilepsy from the dominant temporo-occipital region. Journal of Neurology, Neurosurgery & Psychiatry, 81, 710–715. https://doi.org/10.1136/jnnp.2009.175935
40. Puteikis, K., Mameniškienė, R., & Wolf, P. (2023). Reading epilepsy today: A scoping review and meta-analysis of reports of the last three decades. Epilepsy & Behavior, 145, 109346. https://doi.org/10.1016/j.yebeh.2023.109346
41. Wolf, P. (2017). Reflex epileptic mechanisms in humans: Lessons about natural ictogenesis. Epilepsy & Behavior, 71, 118–123. https://doi.org/10.1016/j.yebeh.2015.01.009
42. Girges, C., Vijiaratnam, N., Wirth, T., Tjoakarfa, C., Idaszak, J., & Seneviratne, U. (2020). Seizures triggered by eating: A rare form of reflex epilepsy: A systematic review. Seizure, 83, 21–31. https://doi.org/10.1016/j.seizure.2020.09.013
43. El Bouzidi, K., Duncan, S., Whittle, I.R., & Butler, C.R. (2010). Lesional reflex epilepsy associated with the thought of food. Neurology, 74, 610–612. https://doi.org/10.1212/WNL.0b013e3181cff6c3
44. Aupy, J., Noviawaty, I., Krishnan, B., Suwankpakdee, P., Bulacio, J., Gonzalez-Martinez, J., Najm, I., & Chauvel, P. (2018). Insulo-opercular cortex generates oroalimentary automatisms in temporal seizures. Epilepsia, 59, 583–594. https://doi.org/10.1111/epi.14011
45. Satishchandra, P. (2003). Hot-water epilepsy. Epilepsia, 44 (Suppl. 1), 29–32. https://doi.org/10.1046/j.1528-1157.44.s.1.14.x
46. Bebek, N., Gürses, C., Gokyigit, A., Baykan, B., Ozkara, C., & Dervent, A. (2001). Hot water epilepsy: Clinical and electrophysiologic findings based on 21 cases. Epilepsia, 42, 1180–1184. https://doi.org/10.1046/j.1528-1157.2001.31000.x
47. Meghana, A., Sinha, S., Sathyaprabha, T.N., Subbakrishna, D.K., & Satishchandra, P. (2012). Hot water epilepsy clinical profile and treatment—a prospective study. Epilepsy Research, 102, 160–166. https://doi.org/10.1016/j.eplepsyres.2012.05.011
48. Tezer, F.I., Ertas, N., Yalcin, D., & Saygi, S. (2006). Hot water epilepsy with cerebral lesion: A report of five cases with cranial MRI findings. Epilepsy & Behavior, 8, 672–676. https://doi.org/10.1016/j.yebeh.2006.01.009
49. Caprara, A.L.F., Tharwat Ali, H., Elrefaey, A., Elejla, S.A., & Rissardo, J.P. (2023). Somatosensory auras in epilepsy: A narrative review of the literature. Medicines (Basel), 10, 49. https://doi.org/10.3390/medicines10080049
50. Kanemoto, K., Watanabe, Y., Tsuji, T., Fukami, M., & Kawasaki, J. (2001). Rub epilepsy: A somatosensory evoked reflex epilepsy induced by prolonged cutaneous stimulation. Journal of Neurology, Neurosurgery & Psychiatry, 70, 541–543. https://doi.org/10.1136/jnnp.70.4.541

51. Bortel, A., Yao, Z.S., & Shmuel, A. (2019). A rat model of somatosensory-evoked reflex seizures induced by peripheral stimulation. Epilepsy Research, 157, 106209. https://doi.org/10.1016/j.eplepsyres.2019.106209
52. Shibasaki, H., Yamashita, Y., Neshige, R., Tobimatsu, S., & Fukui, R. (1985). Pathogenesis of giant somatosensory evoked potentials in progressive myoclonic epilepsy. Brain, 108(Pt 1), 225–240. https://doi.org/10.1093/brain/108.1.225
53. Italiano, D., Ferlazzo, E., Gasparini, S., Spina, E., Mondello, S., Labate, A., Gambardella, A., & Aguglia, U. (2014). Generalized versus partial reflex seizures: A review. Seizure, 23, 512–520. https://doi.org/10.1016/j.seizure.2014.03.014
54. Kornev, V., Belyakova-Bodina, A., & Broutian, A. (2020). Reflex seizures: The classification axis. Human Physiology, 46, 859–869.
55. Szűcs, A., Rásonyi, G., Orbay, P., Sólyom, A., Holló, A., Arányi, Z., Janszky, J., Erőss, L., & Kamondi, A. (2012). Are proprioceptive-induced reflex seizures epileptically-enhanced stretch reflex manifestations? Epileptic Disorders, 14, 149–154. https://doi.org/10.1684/epd.2012.0499
56. Proprioceptive-induced (stimulus-sensitive) reflex seizures. (2020). United States, 94.
57. Ozkara, C., Ozdemir, S., Yilmaz, A., Uzan, M., Yeni, N., Ozmen, M. (2006). Orgasm-induced seizures: A study of six patients. Epilepsia, 47, 2193–2197. https://doi.org/10.1111/j.1528-1167.2006.00648.x
58. Sengupta, A., Mahmoud, A., Tun, S.Z., & Goulding, P. (2010). Orgasm-induced seizures: Male studied with ictal electroencephalography. Seizure, 19, 306–309. https://doi.org/10.1016/j.seizure.2010.04.007
59. Sayin, H.Ü., & Schenck, C.H. (2019). Neuroanatomy and neurochemistry of sexual desire, pleasure, love, and orgasm. SexuS Journal, Winter, 4, 907–946.
60. 60 Velíšková J, Desantis KA. Sex and hormonal influences on seizures and epilepsy. Horm Behav. 2013;63(2):267–77. doi: 10.1016/j.yhbeh.2012.03.018.
61. Millichap JG. Cortical triggers in generalized reflex epilepsies. Pediatr Neurol Briefs. 2005; 31:31–2.
62. Nevler N, Gandelman-Marton R. Acute provoked reflex seizures induced by thinking. Neurologist. 2012;18(6):415–7. doi: 10.1097/NRL.0b013e31827450c9.
63. Lishman WA, Symonds CP, Whitty CW, Willison RG. Seizure induced by movement. Brain. 1962;85(1):93–108. doi: 10.1093/brain/85.1.93.
64. Yakovlev PI. Neurologic mechanism concerned in epileptic seizures. Arch Neurol Psychiatry. 1937;37(4):523–54.
65. Pierelli F, Di Gennaro G, Gherardi M, Spanedda F, Marciani MG. Movement-induced seizures: A case report. Epilepsia. 1997;38(9):941–4. doi: 10.1111/j.1528-1157. 1997.tb01261. x.
66. Leibetseder A, Eisermann M, LaFrance WC Jr, Nobili L, von Oertzen TJ. How to distinguish seizures from non-epileptic manifestations. Epileptic Disord. 2020;22(5):716–38. doi: 10.1684/epd.2020.1234.
67. Lin K, Guaranha M, Wolf P. Reflex epileptic mechanisms in ictogenesis and therapeutic consequences. Expert Rev Neurother. 2016;16(6):573–85. doi: 10.1586/14737175.2016.1169174.

68. Brigo F, Bongiovanni LG, Nardone R, Trinka E, Tezzon F, Fiaschi A, Manganotti P. Visual cortex hyperexcitability in idiopathic generalized epilepsies with photosensitivity: A TMS pilot study. Epilepsy Behav. 2013;27(1):301–6. doi: 10.1016/j.yebeh.2013.02.010.
69. Ferlazzo E, Zifkin BG, Andermann E, Andermann F. Cortical triggers in generalized reflex seizures and epilepsies. Brain. 2005;128(3):700–10. doi: 10.1093/brain/awh446.
70. Doţen N. Psychological interventions in the care of people diagnosed with epilepsy. Stud Univ Moldaviae (Ser Sci Educ). 2024; 175:212–8.
71. Gouveia FV, Warsi NM, Suresh H, Matin R, Ibrahim GM. Neurostimulation treatments for epilepsy: Deep brain stimulation, responsive neurostimulation and vagus nerve stimulation. Neurotherapeutics. 2024;21: e00308. doi: 10.1016/j.neurot. 2023.e00308.