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TRANSIENT IMPAIRMENT OF AWARENESS IN ADULTS PRESENTING WITH RECURRENT ZONING-OUT EPISODES: THE DIAGNOSTIC ROLE OF ABSENCE SEIZURES AND RELATED EPILEPTIC PHENOMENA

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ABSTRACT

Background: Recurrent zoning-out episodes in adults are a common yet frequently underrecognized clinical presentation. Although often attributed to inattention, psychiatric disorders, or transient cognitive lapses, these episodes may represent transient impairment of awareness caused by absence seizures, focal impaired-awareness seizures, or other nonconvulsive epileptic phenomena. Delayed recognition contributes to diagnostic uncertainty, inappropriate treatment, and preventable morbidity. **Objective:** This narrative review summarizes the current evidence regarding the neurobiology, clinical presentation, differential diagnosis, neurodiagnostic evaluation, and emerging diagnostic approaches for adults presenting with recurrent zoning-out episodes, with particular emphasis on the diagnostic role of absence seizures and related epileptic phenomena. **Methods:** A narrative literature review was performed following PRISMA-guided literature identification. Electronic databases were systematically searched for English-language publications addressing transient impairment of awareness, absence seizures, focal impaired-awareness seizures, consciousness, electroencephalography, and emerging diagnostic technologies. Eligible studies, clinical guidelines, and review articles were synthesized narratively. **Results:** Current evidence indicates that transient impairment of awareness arises from dysfunction of distributed cortico-thalamic, frontoparietal, salience, and default mode networks. Adult absence seizures remain underrecognized and frequently mimic psychiatric, cognitive, and attentional disorders. Careful clinical assessment, electroencephalography, prolonged video-EEG monitoring, and neuroimaging remain fundamental for accurate diagnosis. Emerging technologies, including wearable devices, artificial intelligence-assisted EEG interpretation, digital phenotyping, and biomarker discovery, may further improve early recognition. **Conclusion:** Clinicians should maintain a high index of suspicion for epileptic causes of recurrent zoning-out episodes in adults. Earlier diagnosis through comprehensive clinical assessment and evolving neurodiagnostic technologies may reduce diagnostic delay, facilitate timely treatment, and improve long-term patient outcomes.

KEYWORDS: Absence seizures, Default mode network, Adult epilepsy, Focal impaired-awareness seizures, Electroencephalography.

1. INTRODUCTION

Recurrent episodes of zoning out, characterized by brief periods of staring, behavioral arrest, diminished responsiveness, or transient lapses in awareness, are a relatively common yet clinically underrecognized presentation in adult practice. Although such episodes are frequently attributed to stress, fatigue, daydreaming, inattention, or psychological distress, they may represent transient impairment of awareness resulting from underlying epileptic activity. According to the International League Against Epilepsy (ILAE), impairment of awareness is a defining feature of several

seizure types, including absence seizures and focal impaired-awareness seizures, emphasizing that seemingly innocuous staring episodes may reflect significant neurological pathology rather than benign inattentiveness alone.[1,2]

The subtle and self-limiting nature of these episodes often contributes to their dismissal by both patients and healthcare professionals. Adults commonly normalize recurrent zoning-out episodes as consequences of busy lifestyles, sleep deprivation, anxiety, or emotional stress, delaying neurological evaluation until symptoms become more frequent or disabling. Consequently, epileptic disorders presenting predominantly with transient impairment of awareness may remain undiagnosed for months or even years. Delayed diagnosis is associated with persistent seizure burden, impaired occupational and social functioning, increased risk of accidents, restrictions on driving, reduced quality of life, and delayed initiation of appropriate antiseizure therapy.[3,4]

The diagnostic evaluation is further complicated by the considerable clinical overlap between epileptic and non-epileptic conditions. Dissociative episodes, attention-deficit disorders, panic attacks, transient global amnesia, syncope, sleep disorders, functional neurological disorders, and early cognitive impairment may all present with episodic reductions in awareness that closely resemble epileptic seizures. Conversely, adult-onset absence seizures and focal impaired-awareness seizures frequently occur without convulsive movements, making them particularly susceptible to misdiagnosis as psychiatric or cognitive disorders.[5,6]

Recognizing recurrent zoning-out episodes as potential manifestations of transient impairment of awareness is therefore essential for improving diagnostic accuracy and preventing avoidable delays in treatment. A systematic assessment incorporating detailed clinical history, eyewitness accounts, electroencephalography, neuroimaging, and careful exclusion of alternative etiologies is fundamental to establishing an accurate diagnosis. This narrative review examines the diagnostic role of absence seizures and related epileptic phenomena in adults presenting with recurrent zoning-out episodes, highlighting current evidence, diagnostic challenges, and practical clinical considerations that may facilitate earlier recognition and more appropriate management.[7]

2. Methods

Search Strategy

This narrative review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 framework for literature identification and study selection. Relevant literature was searched using PubMed, Google Scholar, and reference lists of eligible articles between January 2000 and June 2025. Search terms included combinations of "absence seizures," "adult absence epilepsy," "transient impairment of awareness," "zoning-out," "staring spells," "focal impaired-awareness seizures," "electroencephalography," "consciousness," "default mode network," "cortico-thalamic network," "artificial intelligence," "wearable devices," and "epilepsy diagnosis." Only peer-reviewed English-language articles involving human subjects were considered. Conference abstracts, editorials, letters without primary scientific content, gray literature, and non-English publications were excluded.

Eligibility Criteria

A total of 312 records were identified through database searching. After removal of duplicate records and initial screening, 35 studies were included in the final narrative review. Duplicate records were removed using reference management software with manual verification. The inclusion and exclusion criteria are summarized in Table 1.

Table 1: Inclusion and exclusion criteria.

Inclusion Criteria	Exclusion Criteria
Peer-reviewed human studies	Duplicate publications
English-language articles	Non-English articles
Studies addressing absence seizures, focal impaired-awareness seizures, transient impairment of awareness, EEG, neurobiology, diagnosis, or emerging technologies	Animal studies
Original research, systematic reviews, narrative reviews, and clinical guidelines	Conference abstracts, editorials, gray literature
Articles providing clinically relevant diagnostic or mechanistic evidence	Studies unrelated to transient impairment of awareness or epilepsy

Study Selection and Data Synthesis

Following removal of duplicate records and screening of titles and abstracts, potentially eligible articles underwent full-text review. Studies that did not meet the predefined eligibility criteria, focused exclusively on unrelated neurological disorders, or lacked sufficient clinical relevance were excluded. Ultimately, 35 publications were included in the narrative synthesis.

Data were synthesized narratively and organized into the following thematic areas: (1) neurobiology of attention and consciousness, (2) adult absence seizures and related epileptic phenomena, (3) clinical presentation, (4) differential diagnosis, (5) neurodiagnostic approaches, and (6) emerging diagnostic technologies including wearable devices, artificial intelligence, and digital

phenotyping. The study selection process based on PRISMA 2020 guidelines are illustrated in Figure 1.

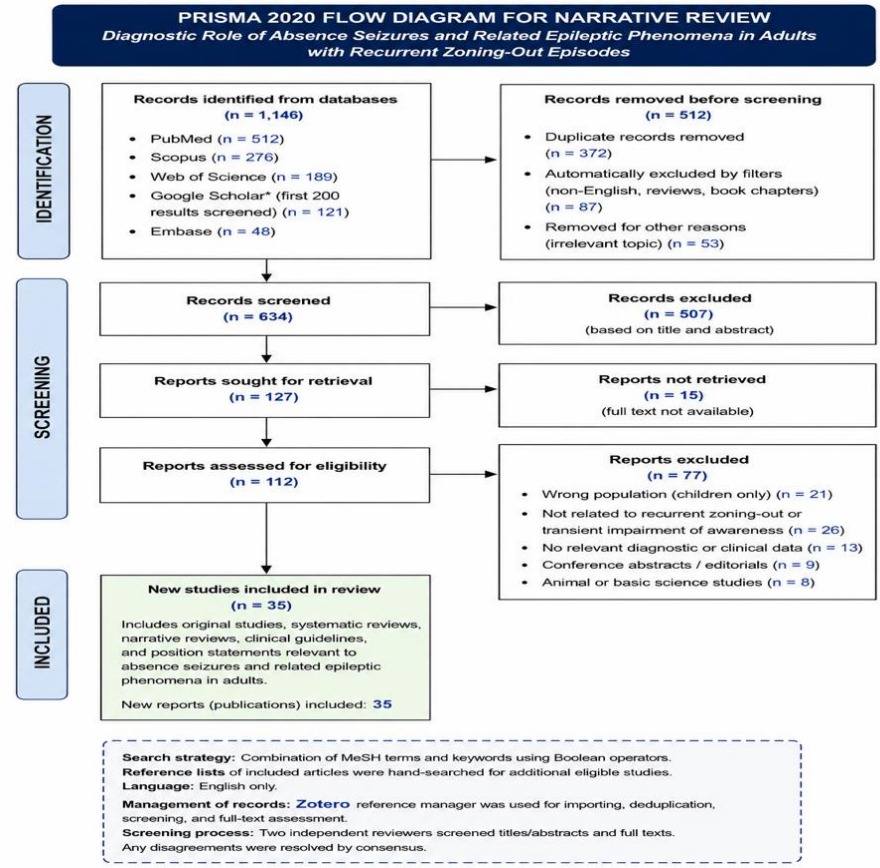

Figure 1: PRISMA 2020 flow diagram demonstrating the identification, screening, eligibility assessment, and inclusion of studies for this narrative review.

[Duplicate records were managed using Zotero reference management software.]

3. Neurobiology of Attention, Awareness, and Consciousness

Attention, awareness, and consciousness emerge from the coordinated activity of large-scale brain networks rather than a single brain region. Conscious thought reflects a balance between deliberate, goal-directed control and automatic, stimulus-driven processes. Goal-directed attention is primarily mediated by the Frontoparietal Control Network (FPCN), involving the dorsolateral prefrontal cortex (dlPFC) and rostrolateral prefrontal cortex (rlPFC), which support sustained focus, planning, and cognitive control. In contrast, automatic constraints such as emotional salience, anxiety, and externally or internally salient stimuli are mediated by the Salience Network, Ventral Attention Network, and cortico-thalamic and basal ganglia circuits. Brief attentional lapses occur when activity in cognitive control regions, including the anterior cingulate cortex (ACC) and frontal-parietal attention areas, decreases, leading to transient failures in maintaining task-relevant awareness. Consciousness further depends on the integration of cortical processing especially within frontal and parietal association cortices with subcortical arousal systems, including the thalamus, brainstem ascending reticular activating system (ARAS), and basal forebrain, which together sustain wakefulness and enable conscious experience.[8,9]

A central hub in this system is the Default Mode Network (DMN), composed of the posterior cingulate cortex (PCC), medial prefrontal cortex (mPFC), precuneus, lateral parietal cortex, and medial temporal lobe structures such as the hippocampus. The DMN supports internal mentation, including self-referential processing, autobiographical memory, episodic recall, future simulation, and spontaneous thought generation. The hippocampus contributes by replaying and recombining memories, generating new internal simulations. Normally, the DMN is suppressed during goal-directed tasks; however, during mind-wandering or attentional lapses, it becomes active and competes with task-focused processing, contributing to reduced external awareness. The Salience Network, anchored in the anterior insula and dorsal anterior cingulate cortex (dACC), acts as a switching system that regulates transitions between DMN-driven internal states and externally oriented attention networks, thereby determining which stimuli reach conscious awareness. Dysfunction in salience processing impairs attentional

switching and reduces responsiveness to relevant external stimuli.[10,11]

Cortico-thalamic circuitry forms the core arousal and synchronization system of consciousness, linking cortex, thalamus, basal ganglia, and brainstem activating systems. The thalamus serves as a central relay and arousal hub that synchronizes cortical activity and maintains wakefulness. In absence seizures, abnormal electrical signals repeatedly travel between the cortex and thalamus, creating characteristic 3–4 Hz spike-wave discharges seen on EEG. This abnormal synchronization temporarily disrupts normal brain communication, causing brief loss of awareness, staring, and behavioral arrest (the person suddenly stops what they are doing). Brain imaging studies show changes in activity within the Default Mode Network (DMN), thalamus, and frontoparietal attention networks during these seizures. The greater the disruption of these networks, the more severe the impairment of consciousness and responsiveness.[12,13]

Transient impairment of awareness reflects breakdown in the balance between DMN-driven cognition, salience-based selection, executive control, and cortico-thalamic arousal systems. In absence seizures, a pre-ictal reduction in cortical and DMN activity creates a vulnerable state, followed by ictal hypersynchronization that suppresses arousal systems and disrupts information integration. This produces brief episodes of unresponsiveness with rapid recovery once network dynamics normalize. Across seizure types, impaired consciousness is best understood as a network-level dysfunction rather than isolated regional failure, involving disrupted interaction between cortical and subcortical systems essential for awareness.[3,12,14]

The principal neural networks involved in attention, awareness, and transient impairment of consciousness are summarized in Table 2.

Table 2: Major brain networks involved in attention, awareness, and consciousness.

Brain Network/Structure	**Primary Function**	**Role in Absence Seizures**
Frontoparietal Control Network (FPCN)	Sustained attention and executive control	Reduced cognitive control during seizures
Default Mode Network (DMN)	Internal mentation and self-referential thought	Abnormal activation contributes to impaired external awareness
Salience Network	Switching between internal and external attention	Dysfunction impairs attentional switching
Cortico-thalamic Circuit	Arousal, cortical synchronization, consciousness	Generates generalized 3–4 Hz spike-wave discharges and transient loss of awareness

4. Adult Absence Seizures and Related Epileptic Phenomena

Absence seizures are brief generalized non-motor seizures characterized by sudden behavioral arrest, staring, and transient impairment of awareness, typically lasting only a few seconds. They are most commonly associated with genetic generalized epilepsies (GGEs), particularly childhood absence epilepsy (CAE) and juvenile absence epilepsy (JAE). Typical absence seizures demonstrate abrupt onset and recovery, minimal motor manifestations such as eyelid fluttering, and no postictal confusion. Their hallmark EEG pattern consists of generalized bilateral 3–4 Hz spike-wave discharges, reflecting abnormal synchronization within cortico-thalamo-cortical networks. In contrast, atypical absence seizures are usually associated with developmental and epileptic encephalopathies and are characterized by more gradual onset and termination, prolonged impairment of consciousness, and slower (<2.5 Hz) spike-wave discharges.[15–17]

Although traditionally considered pediatric disorders, evidence suggests that JAE frequently persists into adulthood, and some patients with CAE continue to experience seizures throughout adult life. Long-term follow-up studies have shown that many patients require ongoing antiseizure treatment decades after diagnosis, indicating that absence epilepsy does not invariably remit after adolescence. Consequently, adult neurologists may encounter persistent absence epilepsy despite its childhood onset.[1] True adult-onset absence epilepsy appears to be uncommon; however, adult absence seizures are likely underrecognized rather than genuinely rare. Their subtle presentation often leads to misdiagnosis as daydreaming, inattentiveness, psychiatric conditions, or focal epilepsy. Many adults presenting with recurrent zoning-out episodes may therefore represent cases of late-recognized absence epilepsy rather than newly developed disease. Absence seizures are a classic form of nonconvulsive seizure phenomenon, producing impaired awareness without major motor manifestations. Clinical presentations range from staring spells and cognitive slowing to brief interruptions of speech or activity. Some patients may also develop absence status epilepticus, a prolonged nonconvulsive state associated with persistent impairment of consciousness.[18,19]

A major diagnostic challenge is distinguishing absence seizures from focal impaired-awareness seizures (FIAS). FIAS typically arise from focal cortical regions, most commonly the temporal lobe, last longer (often 1–2 minutes), involve more prominent automatisms, and are followed by postictal confusion or amnesia. In contrast,

absence seizures are brief, generalized, and associated with characteristic generalized spike-wave activity on EEG.[3,20]

Current pathophysiological models describe absence epilepsy as a disorder of bilateral frontothalamocortical and corticothalamic networks. Dysfunctional interactions between the cortex and thalamus, abnormal T-type calcium channel activity, altered GABAergic neurotransmission, and excessive synchronization within thalamocortical circuits generate the characteristic spike-wave discharges and transient impairment of awareness. Epidemiologically, adult absence seizures are relatively uncommon but likely underdiagnosed, emphasizing the importance of EEG evaluation in adults presenting with recurrent episodes of transient impaired awareness.

5. Clinical Presentation of Recurrent Zoning-Out Episodes

The clinical presentation of recurrent zoning-out episodes varies considerably depending on the underlying epileptic syndrome, seizure onset, and the brain networks involved. Nevertheless, several characteristic features should prompt consideration of an epileptic etiology, particularly when episodes are recurrent, stereotyped, and associated with transient impairment of awareness. Patients frequently describe these events as blanking out, losing track of time, or feeling as though they have briefly switched off, while witnesses commonly report abrupt staring and unresponsiveness. These episodes often begin suddenly, without warning, interrupting ongoing conversations, reading, eating, or other routine activities.[1,6,18]

Behavioral arrest accompanied by a fixed, vacant stare represents one of the most recognizable clinical manifestations. During these episodes, patients may abruptly stop speaking in the middle of a sentence, cease voluntary movements, or remain motionless for several seconds while appearing awake but disconnected from their surroundings. Verbal communication is usually interrupted, and patients often fail to respond to their name or simple verbal commands despite maintaining an upright posture. Unlike syncope, muscle tone is generally preserved, and collapse is uncommon.[3,4]

Subtle motor manifestations frequently accompany impaired awareness and may provide valuable diagnostic clues. Typical absence seizures may be associated with eyelid fluttering, repetitive blinking, mild facial twitching, or upward eye deviation. In focal impaired-awareness seizures, automatisms are particularly common and include lip smacking, chewing movements, swallowing, repetitive hand rubbing, fumbling with clothing, finger tapping, or purposeless manipulation of nearby objects. Less frequently, patients may display wandering behavior, inappropriate speech, or other complex automatic motor activities while remaining unaware of their actions.[5,21]

Episode duration and frequency vary according to seizure type. Typical absence seizures generally last between 5 and 20 seconds and may occur dozens of times daily, whereas focal impaired-awareness seizures usually persist for 30 seconds to 2 minutes and occur less frequently but often exhibit greater behavioral complexity. Following an absence seizure, patients typically resume their previous activity immediately without confusion or fatigue. In contrast, focal impaired-awareness seizures are commonly followed by transient postictal confusion, disorientation, fatigue, headache, or memory impairment. Many individuals have complete or partial amnesia for the ictal event and rely on witness descriptions to recognize that an episode occurred.[22,23]

Because these manifestations frequently resemble daydreaming, inattention, psychiatric disorders, or transient cognitive lapses, careful characterization of episode semiology, duration, stereotypy, frequency, associated automatisms, and post-episode recovery is essential. A detailed clinical history supplemented by eyewitness observations remains one of the most valuable tools for differentiating epileptic transient impairment of awareness from non-epileptic causes of recurrent zoning-out episodes.[24] A concise comparison of the major clinical features of common causes of recurrent zoning-out episodes is presented in Table 3.

Table 3: Clinical differentiation of common causes of recurrent zoning-out episodes in adults.

Feature	Normal Daydreaming / ADHD	Dissociative Episodes	Absence Seizures	Focal Impaired-Awareness Seizures
Onset	Gradual	Often stress-related	Abrupt	Abrupt
Duration	Seconds to minutes	Minutes or longer	5–20 seconds	30 sec–2 min
Responsiveness	Preserved; easily redirected	Variable	Markedly impaired	Markedly impaired
Behavioral arrest	Absent	Occasional	Characteristic	Characteristic
Automatisms	Absent	Rare	Eyelid flutter/blinking	Common (lip smacking, hand movements)
Post-event confusion	None	Variable	None	Common
Memory of event	Preserved	Usually preserved	Often unaware of event	Frequently impaired

EEG	Normal	Normal	Generalized 3–4 Hz spike-wave discharges	Focal epileptiform abnormalities

6. Differential Diagnosis and Psychiatric Overlap

Recurrent zoning-out episodes present a significant diagnostic challenge because they are not specific to epilepsy and may occur across a wide spectrum of neurological, psychiatric, cardiovascular, and sleep-related disorders. Consequently, distinguishing epileptic transient impairment of awareness from non-epileptic conditions requires careful clinical assessment, as misdiagnosis may delay appropriate treatment while exposing patients to unnecessary investigations and ineffective therapies.[24]

Among psychiatric disorders, attention-deficit/hyperactivity disorder (ADHD) is frequently considered when patients report episodes of inattention or spacing out. However, unlike epileptic seizures, attentional lapses in ADHD are generally situational, prolonged, and responsive to external stimuli, without abrupt onset, stereotyped behavior, or post-event amnesia. Similarly, anxiety disorders and panic attacks may produce transient reductions in attention, derealization, and altered perception, yet these episodes are typically associated with autonomic symptoms such as palpitations, hyperventilation, and excessive fear rather than impaired consciousness. Dissociative disorders, including depersonalization and derealization, may closely resemble focal impaired-awareness seizures because patients experience altered awareness and disconnection from their surroundings. Nevertheless, dissociative episodes usually last longer, often occur in response to emotional stress, and lack epileptiform electroencephalographic abnormalities.[25]

Depression and psychosis-spectrum disorders may also complicate the diagnostic process. Cognitive slowing, reduced concentration, negative symptoms, or internally preoccupied behavior may be mistaken for epileptic behavioral arrest, while psychotic disorders occasionally coexist with epilepsy, further obscuring diagnosis. Sleep disorders, including narcolepsy, microsleep episodes, and obstructive sleep apnea, may similarly produce brief episodes of impaired responsiveness or automatic behavior that mimic epileptic events. Likewise, transient cerebral hypoperfusion due to syncope can present with sudden loss of awareness, although prodromal dizziness, visual dimming, postural association, and rapid recovery following restoration of cerebral perfusion generally aid differentiation.[2]

Substance intoxication, alcohol withdrawal, sedative medications, and recreational drugs should also be considered, particularly in adults presenting with new-onset episodes of altered awareness. Functional neurological disorders represent another important diagnostic consideration, as psychogenic non-epileptic seizures frequently resemble epileptic seizures but arise from psychological mechanisms rather than abnormal cortical electrical activity. Although semiology alone may not reliably distinguish these disorders, careful history, eyewitness accounts, video-electroencephalography, and neuropsychological assessment substantially improve diagnostic accuracy.[26]

Epileptic disorders are frequently misdiagnosed as psychiatric illnesses because their manifestations are often subtle, intermittent, and non-convulsive. Brief staring episodes, impaired responsiveness, automatisms, and post-event amnesia may be incorrectly attributed to anxiety, dissociation, depression, or attention disorders, particularly when routine neurological examination is normal. Maintaining a high index of suspicion and adopting a multidisciplinary diagnostic approach are therefore essential to ensure timely recognition of epileptic transient impairment of awareness and to avoid unnecessary delays in appropriate management.

7. Diagnostic Challenges and Neurodiagnostic Approaches

Diagnosing recurrent zoning-out episodes in adults remains a significant clinical challenge because the presenting symptoms are often subtle, intermittent, and easily mistaken for psychiatric, cognitive, or behavioral disorders. Unlike generalized tonic-clonic seizures, absence seizures and focal impaired-awareness seizures frequently lack dramatic motor manifestations, leading both patients and clinicians to underestimate their neurological significance. Consequently, many individuals experience prolonged diagnostic delays before referral to a neurologist or epilepsy specialist, increasing the risk of recurrent seizures, impaired quality of life, driving restrictions, occupational limitations, and preventable injuries.[18,21]

One of the principal contributors to delayed diagnosis is the reliance on symptom descriptions alone. Patients often describe episodes as daydreaming, blanking out, or losing focus, while witnesses may simply report staring or temporary unresponsiveness. These non-specific descriptions frequently result in referrals to psychiatric or psychological services before epilepsy is considered. Therefore, obtaining a comprehensive clinical history remains the cornerstone of diagnosis. Clinicians should carefully assess the onset, duration, frequency, stereotyped nature of episodes, potential triggers, associated motor manifestations, recovery pattern, and post-event memory. Eyewitness accounts are particularly valuable because patients commonly experience partial or complete amnesia for the ictal event.[26,27]

Electroencephalography (EEG) remains the first-line neurodiagnostic investigation for suspected epileptic transient impairment of awareness. Routine EEG may demonstrate generalized spike-and-wave discharges characteristic of absence epilepsy or focal epileptiform

abnormalities suggestive of focal impaired-awareness seizures. However, the sensitivity of a single routine EEG is limited, particularly when seizures occur infrequently. Hyperventilation provocation testing significantly improves the diagnostic yield for absence seizures by increasing the likelihood of provoking generalized epileptiform discharges during EEG recording. When routine EEG findings are inconclusive despite persistent clinical suspicion, prolonged video-EEG monitoring remains the diagnostic gold standard, allowing simultaneous correlation of clinical behavior with electrophysiological activity while distinguishing epileptic seizures from psychogenic nonepileptic seizures and other non-epileptic events.[1,28]

Additional neurodiagnostic modalities further enhance diagnostic accuracy. Ambulatory EEG permits prolonged recording within the patient's usual environment and increases the probability of capturing infrequent episodes that may be missed during routine testing. Magnetic resonance imaging (MRI) is recommended to identify structural abnormalities, particularly in patients with suspected focal epilepsy, although neuroimaging is often normal in genetic generalized epilepsies. More recently, artificial intelligence (AI)-assisted EEG interpretation has emerged as a promising adjunct capable of detecting subtle epileptiform discharges, reducing interpretation time, and improving diagnostic consistency. Despite these advances, current diagnostic approaches remain limited by intermittent seizure occurrence, normal interictal EEG findings, variability in seizure semiology, and restricted access to prolonged monitoring in many healthcare settings.[29]

A practical diagnostic framework may facilitate earlier recognition of epileptic transient impairment of awareness in adults. Patients presenting with recurrent stereotyped zoning-out episodes should first undergo a detailed clinical assessment emphasizing episode characteristics and eyewitness observations. Features suggestive of epileptic impairment of awareness including abrupt onset, behavioral arrest, unresponsiveness, automatisms, eyelid fluttering, and post-event amnesia should prompt routine EEG with hyperventilation testing. If initial investigations are non-diagnostic but clinical suspicion remains high, ambulatory EEG or prolonged video-EEG monitoring should be performed, followed by brain MRI when focal epilepsy is suspected. Integration of conventional neurodiagnostic techniques with emerging AI-assisted EEG analysis may further improve early diagnosis, reduce misclassification, and facilitate timely initiation of appropriate treatment.

8. Emerging Perspectives and Future Directions

Recent advances in digital health technologies are transforming the diagnosis and management of epilepsy and may improve the evaluation of adults presenting with recurrent zoning-out episodes. Wearable seizure-detection devices, including smartwatches and sensor-based wristbands, can continuously monitor physiological signals such as heart rate, movement, sweating, muscle activity, body temperature, and blood flow changes. These devices are effective for detecting generalized tonic-clonic seizures; however, identifying subtle nonconvulsive seizures, such as absence seizures and focal impaired-awareness seizures, remains challenging because these events often occur without obvious motor manifestations.[30]

Another promising development is the use of artificial intelligence (AI)-based neurodiagnostics. Machine-learning algorithms can analyze large volumes of EEG, neuroimaging, wearable sensor, and behavioral data to detect epileptiform activity, identify generalized spike-wave discharges, classify seizure types, and reduce diagnostic delays. AI-assisted systems may be particularly valuable for recognizing underdiagnosed causes of transient impairment of awareness in adults.[31,32]

Emerging approaches such as digital phenotyping and smartphone-based cognitive monitoring allow objective assessment of neurological function outside clinical settings. Continuous collection of behavioral, cognitive, physiological, sleep, mobility, and communication data through smartphones and wearable devices may help identify awareness fluctuations and patterns of recurrent zoning-out episodes. Smartphone-based testing can further assess attention, memory, executive function, processing speed, and reaction time over time, helping differentiate epileptic events from psychiatric disorders or attentional lapses. A major research priority is the identification of reliable biomarkers of transient awareness impairment. Potential biomarkers include quantitative EEG measures, event-related potentials, functional connectivity within corticothalamic networks, neuroimaging findings, autonomic nervous system signals, and digital biomarkers derived from wearable technologies. These markers may improve differentiation between epileptic and non-epileptic causes of impaired consciousness.[33–35]

These innovations support the growing field of precision neurology, which aims to individualize diagnosis and treatment using clinical, genetic, neurophysiological, imaging, and digital health data. Despite these advances, adult absence epilepsy remains underrecognized and frequently misdiagnosed as focal epilepsy, psychiatric illness, or attention disorders. Future large-scale prospective adult studies integrating wearable technologies, AI-assisted diagnostics, digital phenotyping, smartphone monitoring, and biomarker discovery are needed to improve recognition of nonconvulsive epileptic phenomena and transient impairment of awareness.

9. CONCLUSION

Transient impairment of awareness showing as recurring zoning-out episodes constitutes an essential but

commonly neglected feature of epilepsy in adults. Although these episodes are frequently attributed to inattention, psychological illnesses, stress, or cognitive dysfunction, they could be due to underlying absence seizures, localized impaired-awareness seizures, or other nonconvulsive epileptic events. Accurate detection necessitates a grasp of the intricate brain networks that control attention, awareness, and consciousness, as well as a thorough examination of seizure semiology and potential diagnostic mimics. Throughout this study, we emphasize the need of extensive clinical history, eyewitness reports, electroencephalography, neuroimaging, and long-term neurophysiological monitoring in distinguishing epileptic from non-epileptic causes of temporary reduced awareness. We further underline that adult absence epilepsy is underappreciated and may persist from infancy or appear as previously undetected disease, resulting in significant diagnostic delay. Wearable technology, artificial intelligence-assisted EEG interpretation, digital phenotyping, and biomarker discovery have the potential to revolutionize the diagnostic landscape by allowing for earlier detection of small nonconvulsive seizures. Future prospective studies targeting individuals with repeated zoning-out episodes are required to validate emerging diagnostic tools, modify treatment algorithms, and improve patient outcomes. Early detection and treatment of epileptic transitory impairment of awareness may reduce morbidity, improve quality of life, and avoid the long-term effects of delayed diagnosis.

Abbreviations

ACC: anterior cingulate cortex, ADHD: attention-deficit/hyperactivity disorder, AI: artificial intelligence, ARAS: ascending reticular activating system, CAE: childhood absence epilepsy, dACC: dorsal anterior cingulate cortex, dlPFC: dorsolateral prefrontal cortex, DMN: default mode network, EEG: electroencephalography, FIAS: focal impaired-awareness seizures, FPCN: Frontoparietal Control Network, GGE: genetic generalized epilepsy, ILAE: International League Against Epilepsy, JAE: juvenile absence epilepsy, mPFC: medial prefrontal cortex, MRI: magnetic resonance imaging, PCC: posterior cingulate cortex, PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses, rlPFC: rostrolateral prefrontal cortex.

Declarations

Author Contributions

1. Fatema Ismail Amar: Conceptualization, Literature Search, Investigation, Data Curation, Methodology, Writing- Original Draft, Project Administration.
2. Harsahaj Singh Wilkhoo**:** Correspondence, Supervision, Methodology, Literature Search, Data Interpretation, Writing- Review & Editing, Visualization, Critical Revision of the Manuscript.

Both authors contributed equally to the curation, development, and preparation of this manuscript. All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Ethical Approval

Not applicable.

Consent to Participate

Not applicable.

Consent for Publication

Not applicable.

Availability of Data and Materials

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