First epileptic seizure in youth and adulthood

Habermehl, Lena; De Stefano, Pia; Gschwind, Markus

How to cite

HABERMEHL, Lena, DE STEFANO, Pia, GSCHWIND, Markus. First epileptic seizure in youth and adulthood. In: Clinical epileptology, 2026, vol. 39, n° 1, p. 28–33. doi: 10.1007/s10309-025-00806-9

This publication URL: https://archive-ouverte.unige.ch/unige:191244
Publication DOI: 10.1007/s10309-025-00806-9

Clin Epileptol 2026 · 39:28–33
https://doi.org/10.1007/s10309-025-00806-9
Received: 2 June 2025
Accepted: 17 November 2025
Published online: 9 December 2025

First epileptic seizure in youth and adulthood

Lena Habermehl[1,2] · Pia De Stefano[3] · Markus Gschwind[4,5]

[1] Epilepsy Center Hessen, Philipps-Universität Marburg, Marburg, Germany
[2] Department of Neurology, University Hospitals of Cleveland Medical Centre, Cleveland, USA
[3] Neuro-Intensive Care Unit, Department of Intensive Care, University Hospital of Geneva, Geneva, Switzerland
[4] Neurology Department, Cantonal Hospital Aarau, Aarau, Switzerland
[5] Department of Clinical Neuroscience, University of Geneva, Geneva, Switzerland

Abstract

Seizures are the third most common reason for presentation to the neurological emergency department. The diagnosis and management of (first) seizures are therefore of great importance in every neurology department. In this article, we provide an overview of the diagnostic tools needed to exclude causes requiring acute treatment, the assessment of recurrence risk to provide an accurate diagnosis, and the current recommendations for medications and fitness to drive.

Keywords
First seizure · Diagnosis · Risk of recurrence · Antiseizure medication · Driving regulations

Introduction

Seizures and seizure-related problems are the third most common reason for neurological emergency consultation after stroke and headaches, accounting for 10–15% of cases [15]. Epilepsy affects approximately 0.7% of the general population [11], while lifetime prevalence of an isolated unprovoked seizure reaches 8–10% [17]. The diagnosis and management of a first epileptic seizure are therefore an important task in acute neurology, both in hospital and private practice settings.

Clinical assessment of the patient after a supposed seizure must always address three key questions:

1. Was the transitory event truly epileptic or was it an epileptic mimic? Differential diagnoses include syncope, migraine, psychogenic non-epileptic seizures (PNES), sleep-related conditions such as parasomnia, movement disorders, transient global amnesia, etc., each requiring a distinct management approach. A comprehensive overview of epilepsy and epilepsy mimics is available online via the International League Against Epilepsy (ILAE) website (https://www.epilepsydiagnosis.org/epilepsy-imitators.html).
2. If the event is thought to be epileptic, was it an acute symptomatic seizure (ASS) or an unprovoked seizure? Acute symptomatic seizures are defined as seizures with a close temporal connection to an acute central nervous system (CNS) injury or a toxic or metabolic condition. The incidence of ASSs is 29 per 100,000 [3]. The incidence of unprovoked seizures (where no such cause can be found) ranges from 42 to 61 per 100,000 [17], depending on the study and methodology. Differentiating an ASS from an unprovoked seizure is critical, as it determines treatment choice and prognosis.
3. Finally, if the event is seen as an unprovoked seizure, are the diagnostic criteria for epilepsy met? According to the ILAE, epilepsy is diagnosed under one of the following conditions: The occurrence of at least two unprovoked

Scan QR code & read article online

Table 1 Possible causes of acute symptomatic seizures	
Cause	**Comments**
Traumatic	Within < 7 days, associated with traumatic hemorrhage
Vascular	Ischemic stroke (< 7 days) Intracerebral hemorrhage Subarachnoid bleeding Cerebral vein thrombosis
Metabolic	Acute changes of renal or hepatic dysfunction Hyperammonemia (35 mM) Na < 115 mg/dL (< 5 mM) Mg < 0.8 mg/dL (< 0.3 mM) Ca < 5 mg/dL (< 1.2 mM) Glucose < 36 mg/dL (2.0 mM) or 450 mg/dL (25 mM) associated with ketoacidosis
Medication	Chlorpromazine, clozapine, maprotiline, clomipramine, bupropion, meperidine, flumazenil, cyclic antidepressants, theophylline, isoniazid, alkylating antineoplastic agents, cyclosporine; overdose of medication
Infections	Within < 7–14 days viral encephalitis, bacterial meningitis, degenerative phase of neurocysticercosis
Illicit drugs	Amphetamine-like drugs, cocaine, crack, angel dust (phencyclidine), heroin or THC-rich cannabis
Withdrawal, deficiency	Alcohol Benzodiazepine Barbiturate
Other	Posterior reversible encephalopathy syndrome; cerebral anoxia; eclampsia; multiple sclerosis within 7 days of relapse
Adapted from [3] and [8]	

(or reflex) seizures more than 24 h apart, or one unprovoked seizure in the presence of a condition associated with a more than 60% risk of recurrence over the next 10 years, or if an epilepsy syndrome can be recognized [13]. It is thus essential to clarify whether the initial event was in fact a first seizure, as up to 50% of patients who initially present with a first seizure later report prior unrecognized events when a detailed clinical history is taken [32]. Given that pathological findings on electroencephalography (EEG) or magnetic resonance imaging (MRI) are associated with a long-term recurrence risk exceeding 60%, these examinations are indispensable for risk stratification and diagnostic clarification [13, 22].

Work-up

To differentiate between an acute symptomatic seizure and an unprovoked seizure, laboratory tests are necessary (electrolytes, blood sugar, inflammation markers, but also markers of kidney and liver function). Toxicological screening may be useful in cases of prolonged postictal phase or anamnestic indications of intoxication (◘ **Table 1**; [4, 10]).

There is benefit in measuring creatine kinase (CK), lactate, phosphate, and prolactin after a suspected seizure. The serum CK rises in 45% of cases about 2–3 h after a bilateral or generalized tonic–clonic seizure (GTCS) and reaches a peak after 24–48 h. Both in psychogenic non-epileptic seizure (PNES) and syncope, there is no significant rise in CK [8]. Serum lactate levels rise within 15 min of a GTCS and are the best marker at this time point to distinguish between GTCS and any other unclear loss of consciousness (e.g., non-motor epilepsy, syncope or PNES; [37, 37]). However, 2–3 h later, lactate values may return to normal and indicate false-negative values. Also, if the tourniquet is applied for too long before venipuncture, this could lead to falsely high lactate values in the blood sample taken [30]. It is furthermore possible that GTCS of very short seizure duration may not trigger enough muscular contraction to cause elevated lactate levels. However, lactate is very easily obtainable through venous blood gas analysis and can therefore be performed very quickly also in emergency conditions [37]. Furthermore, data show that hypophosphatemia is associated with GTCS compared to non-epileptic loss of consciousness [2, 7].

Serum prolactin rises in about 60% of GTCSs and in 46% of focal impaired awareness seizures and reaches a maximum within 10–20 min and returns to baseline within 2–6 h. The specificity of a postictal prolactin increase is about 96% for GTCS in differentiation from PNES. Fluctuating prolactin levels must be considered, for example, with dopaminergic medication or depending on the time of day. In addition, a prolactin increase occurs after 60–80% of syncope cases [34]. Knowing these limitations, no single value can replace the diagnostic work-up, but their combination is meaningful and should be taken into account.

If the patient is neurologically normal and fully oriented on presentation, cranial computer tomography (cCT) does not need to be performed in the acute situation, as cranial MRI (cMRI) in the short term is superior to cCT in the search for epileptogenic lesions [20]. However, if there is still a neurological deficit, at least native CT and possibly also CT angiography should be performed to avoid overlooking an ASS in the case of ischemic stroke, intracranial hemorrhage, or brain tumor. Whether the cMRI performed includes only a routine protocol (to detect tumors, strokes, or hemorrhages) or incorporates dedicated epilepsy sequences according to the Harmonized Neuroimaging of Epilepsy Structural Sequences (HARNESS MRI protocol) for more sensitive detection of subtle potentially epileptogenic lesions (e.g., cortical malformations, focal cortical dysplasia; [6]) must be decided based on availability. It should be noted that potentially epileptogenic lesions will not automatically lead to a diagnosis of epilepsy. We propose critically scrutinizing whether the semiology of the seizure matches the location of the detected lesion to avoid overdiagnosis. On the other hand, the importance of not only a 3-T MRI and a dedicated MRI protocol, but also of a specialized trained reading of the MRI images in medically refractory epilepsies is ideal, as epileptogenic focal lesions were correctly reported by "non-expert" radiologists on standard MRI images in only 39% of cases, compared to expert neuroradiologists in 50%

of cases on standard MRI vs. 91% of cases on dedicated MRI images [36].

The presence of interictal epileptiform discharges (IEDs) in the EEG leads to a higher risk of seizure recurrence [13]. Between 22% and 53% of first-seizure patients show IEDs in EEG after a first epileptic seizure [1, 22]. An EEG performed within the first 24 h after the seizure increased the detection rate of IEDs in a study performed with adults and children [23]. In patients with suspected focal epilepsy, a prolonged EEG in sleep (focal epilepsy) or after awakening (juvenile myoclonic epilepsy) might show IEDs after normal routine EEG [12, 14]. In some centers, long-term EEG is already available in routine diagnostics, where it can provide considerable added value [41]. Also, the recording of a video in parallel with the EEG and the use of additional inferior temporal electrodes according to the IFCN 2017 recommendations are now standard and indispensable [40].

Routine cerebrospinal fluid diagnostics do not need to be performed after the first seizure in every case, as up to 10% of patients with an otherwise unremarkable clinical picture show post-ictal pleocytosis of up to 50/µL [42]. In the case of a suitable clinical syndrome with cognitive and psychiatric disorders and/or typical MRI changes in mesiotemporal structures that are suggestive of viral or autoimmune encephalitis, however, it must not be delayed due to the important therapeutic consequences [31].

Risk of recurrence

The risk of recurrence is directly related to the classification as unprovoked seizure, ASS, or definite epilepsy. The risk of seizure recurrence after an ASS depends on the cause. Cerebral pathologies have higher risks than metabolic causes due to the subsequent brain lesions. After an ASS, 30–40% of patients with ASS associated with viral encephalitis and 10–18% of patients with ASS after hemorrhagic stroke are diagnosed with epilepsy in the long term [19].

Most population-based studies have shown a recurrence risk after a first unprovoked seizure of approximately 36% after 1 year and 40–50% after 2 years [5]. This did not change significantly after the recently introduced change in diagnostic criteria of epilepsy [26]. The risk of recurrence increases after prolonged seizures, status epilepticus, a previous ASS, and also the presence of visible IEDs in the EEG recording [16]. Although first-ever unprovoked seizures from sleep occur in only 23% of cases, they are more likely to recur, independent of other risk factors (54% vs. 44%), albeit with a lower risk of seizure-related injury [25]. The risk of recurrence in patients with unprovoked seizures and without pathological findings on EEG or MRI is 10–26% within the first year and 30–35% within 5 years [22]. But this rate increases to > 60% in the presence of potentially epileptogenic lesions on MRI or IEDs on EEG or after a second seizure [5, 23].

Therapy

Acute symptomatic seizures encompass variable causes with varying risks of recurrence. Therefore, there is ongoing debate on how to best give clear recommendations regarding the introduction of antiseizure medication (ASM) and its duration, which should rarely exceed 3–6 months [18, 45].

Once the diagnosis of epilepsy is established, treatment with ASM is definitely indicated. Although there are around 30 different ASMs approved on the market, only two prospective, case-controlled studies—SANAD 1 and SANAD 2—have compared one drug with another [27, 29]. Balancing anticonvulsive effectiveness and side effects, current guidelines recommend lamotrigine (LTG) as first-line therapy for focal epilepsy and valproic acid (VPA) for primary generalized epilepsy. As an alternative in focal epilepsy, lacosamide (LCM) and levetiracetam (LEV) are recommended (although LCM is not reimbursed as monotherapy in Switzerland). In patients with primary generalized epilepsy (especially in women without birth control), LTG and LEV should be considered first [21]. Across all types of epilepsy, with good medication compliance, around 50% of patients become seizure-free with the first ASM and a further 10–15% with the second substance [9]. The specific drug selection should be based on comorbidities and co-medication to minimize drug interactions (e.g., with oncological treatment or concomitant anticoagulation). A non-response to the first drug reduces the chance of seizure control in subsequent years. However, true pharmacoresistance, i.e., non-response to two medications, is rare in the first few years, and the treating physicians or neurologists should therefore be encouraged to monitor blood levels. If seizures persist despite all efforts, further comprehensive inpatient assessment is indicated, as the diagnosis should be re-evaluated.

Delay in diagnosis and treatment

An increasing number of studies have shown that timely evaluation after a first unprovoked seizure is critical. Several studies found that initiating ASM after a single seizure may reduce recurrence risk in the first 2 years, but it may not improve long-term remission [28]. Nonetheless, multiple prior unrecognized seizures are a strong predictor of future relapses despite therapy [24]. Therefore, proper work-up and treatment should not be delayed, particularly after a second seizure.

Delayed diagnosis is even more common, especially in focal epilepsies with nonmotor presentations. These subtle symptoms are often overlooked, leading to misdiagnosis and under-recognition, which can result in diagnostic delays exceeding a decade in some cases [38]. Such delays are associated with increased seizure frequency, a higher risk of injury, drug resistance, and impaired psychosocial functioning. Longer delays are also linked to structural brain pathology and worse seizure outcomes [44].

In this context, dedicated clinical pathways for first seizures ("first seizure clinics")—such as those implemented in most tertiary centers—improve diagnostic precision, reduce inappropriate testing, and enable early therapeutic decisions, finally improving long-term outcome [35].

Driving

In Austria, Germany, and Switzerland driving regulations for individuals having experienced a seizure follow the European guidelines, requiring a minimum seizure-

Table 2 Driving ban for private drivers (Group 1)	
Acute symptomatic seizure	
A	6 months (or shorter if the provoking factor can instantly be removed)
D	3 months
CH	3 months
Unprovoked seizure	
A	6 months (or shorter if the provoking factor can instantly be removed)
D	6 months
CH	6 months
Epilepsy	
A	12 months (ASM allowed)
D	12 months (ASM allowed) Only sleep-related seizures under observation of > 3 years: 0 months Only non-disabling seizures w/o LOC under observation of > 1 year: 0 months
CH	12 months (ASM allowed) Only sleep-related seizures under observation of > 2 years: 0 months Only non-disabling seizures w/o LOC at discretion of treating neurologist
A Austria, *ASM* antiseizure medication, *CH* Switzerland, *D* Germany, *LOC* loss of consciousness, *w/o* without	

Table 3 Driving ban for professional drivers (Group 2)	
Acute symptomatic seizure	
A	5 years (no ASM)
D	12 months (no ASM)
CH	6 months (no ASM)
Unprovoked seizure	
A	5 years
D	2 years (ASM allowed)
CH	Trucks: 2 years (no ASM), Bus: 5 years (no ASM)
Epilepsy	
A	10 years (no ASM) Only sleep-related seizures observed during > 3 years: reduction to 5 years by expert possible
D	5 years (no ASM)
CH	Trucks: 5 years (no ASM); buses: not allowed Only non-disabling seizures w/o LOC at discretion of treating neurologist
A Austria, *ASM* antiseizure medication, *CH* Switzerland, *D* Germany, *LOC* loss of consciousness, *w/o* without	

Table 4 Driving ban for psychogenic non-epileptic seizures		
Country	**Group 1**	**Group 2**
A	–	–
D	1 year	10 years
CH	At the discretion of treating neurologist	At the discretion of treating neurologist
A Austria, *CH* Switzerland, *D* Germany		

free period before being considered fit to drive. On one hand, the distinction between seizure type, such as ASS, first unprovoked seizure, or confirmed epilepsy, is essential for classifying the required duration of driving ban; on the other hand, it is important to differentiate between driving license types, such as private driving (motor bike or car) or professional driving (taxis, ambulances, trucks, and buses). Due to the much longer daily driving times of professional drivers, often operating heavy vehicles and transporting passengers, all countries are imposing much stricter rules for this group (Group 2; [33]). An overview is provided in ◘ **Tables 2 and 3**.

While in the United States all 50 states have highly heterogeneous rules, the American Academy of Neurology has very recently published a position statement about seizures and driver licensure [43]. There is consensus that epileptic seizures modestly increase the risk of motor vehicle accidents (MVAs), especially when seizure frequency is high. However, the risk of fatal MVAs among individuals with epilepsy is not elevated compared to the general population and is markedly lower than in those with alcohol use disorder or in young drivers [39]. Conversely, it must be taken into consideration that driving is an important factor for patients' quality of life and their social and occupational engagement, and many individuals with epilepsy drive against medical advice, suggesting that more personalized, less burdensome regulations might improve compliance.

The consensus paper lists several factors that could help the treating neurologist achieve a less strict ruling, such as focal aware seizures that do not interfere with motor control and established seizures occurring exclusively during sleep, both already implemented in the D-A-CH rulings. Other alleviating factors are knowledge of the unlikely recurrence risk of some ASSs and recognition that medication changes that were prescribed by the doctor may have provoked a seizure. The consensus also discerned unfavorable factors that argue for stricter decisions, such as compliance problems with medical visits and medications, substance use disorder, prior crashes due to seizures, and prior records of MVA and violations of driving regulations. Finally, situations with either a recently increased number of seizures, patients with recurrent seizures of which they are unaware, and the presence of structural brain lesions warrant special attention.

Evidence of MVA risk due to PNES is scarce. Available data indicate that individuals with functional seizures experience MVAs linked to their episodes and may have a higher overall accident rate compared to those with epilepsy, although the resulting injuries tend to be less severe. In patients with the potential for altered responsiveness or involuntary movements during PNES, driving restrictions similar to those for epilepsy are recommended. De-

cisions regarding seizure-free intervals, especially for those seeking professional driving licenses, should be based on individualized clinical assessments ([43]; ◘ **Table 4**).

Conclusion

Epileptic seizures are a frequent finding that requires an early and accurate diagnosis, rapid initiation of antiseizure medication (ASM) in appropriate cases, minimization of diagnostic delays, and adoption of structured clinical pathways. Evaluation aims to exclude epilepsy mimics and to achieve an adequate assessment of recurrence risk, beginning with a detailed medical history and clinical examination, followed by standardized electroencephalography (EEG) and imaging. At minimum, routine EEG and magnetic resonance imaging (MRI) following a predefined epilepsy protocol are recommended. Correlation of seizure semiology with EEG and MRI findings is essential. These interventions collectively reduce morbidity, improve long-term outcomes, and enhance the overall quality of care.

Corresponding address

Lena Habermehl, MD
Epilepsy Center Hessen, Philipps-Universität Marburg
Baldingerstraße, 35043 Marburg, Germany
lhaberme@med.uni-marburg.de

Funding. This article was not funded in any way.

Author Contribution. Drafting the work: LH, MG. Revising the work for important intellectual content: all authors. Final approval of the final version to be published: all authors.

Funding. Open Access funding enabled and organized by Projekt DEAL.

Declarations

Conflict of interest. L. Habermehl, P. De Stefano and M. Gschwind declare that they have no competing interests.

For this article no studies with human participants or animals were performed by any of the authors. All studies mentioned were in accordance with the ethical standards indicated in each case.

References

1. Baldin E, Hauser WA, Buchhalter JR, Hesdorffer DC, Ottman R (2014) Yield of epileptiform electroencephalogram abnormalities in incident unprovoked seizures: a population-based study. Epilepsia 55(9):1389–1398
2. Barras P, Siclari F, Hügli O, Rossetti AO, Lamy O, Novy J (2019) A potential role of hypophosphatemia for diagnosing convulsive seizures: A case-control study. Epilepsia 60(8):1580–1585
3. Beghi E, Carpio A, Forsgren L, Hesdorffer DC, Malmgren K, Sander JW, Tomson T, Hauser WA (2010) Recommendation for a definition of acute symptomatic seizure. Epilepsia 51(4):671–675
4. Beleza P (2012) Acute symptomatic seizures: a clinically oriented review. Neurologist 18(3):109–119
5. Berg AT, Shinnar S (1991) The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. Neurology 41(7):965–972
6. Bernasconi A, Cendes F, Theodore WH, Gill RS, Koepp MJ, Hogan RE, Jackson GD, Federico P, Labate A, Vaudano AE, Blümcke I, Ryvlin P, Bernasconi N (2019) Recommendations for the use of structural magnetic resonance imaging in the care of patients with epilepsy: A consensus report from the International League Against Epilepsy Neuroimaging Task Force. Epilepsia 60(6):1054–1068
7. Binks SN, Zorkin D, Liem B, Sen A (2025) Hypophosphataemia in suspected seizures evaluated in the first seizure clinic and neurology consults
8. Brigo F, Igwe SC, Erro R, Bongiovanni LG, Marangi A, Nardone R, Tinazzi M, Trinka E (2015) Postictal serum creatine kinase for the differential diagnosis of epileptic seizures and psychogenic non-epileptic seizures: a systematic review. J Neurol 262(2):251–257
9. Chen Z, Brodie MJ, Liew D, Kwan P (2017) Treatment Outcomes in Patients With Newly Diagnosed Epilepsy Treated With Established and New Antiepileptic Drugs: A 30-Year Longitudinal Cohort Study. JAMA Neurol 75(3):279–286
10. Delanty N, Vaughan CJ, French JA (1998) Medical causes of seizures. Lancet 352(9125):383–390
11. Fiest KM, Sauro KM, Wiebe S, Patten SB, Kwon C-S, Dykeman J, Pringsheim T, Lorenzetti DL, Jetté N (2017) Prevalence and incidence of epilepsy: A systematic review and meta-analysis of international studies. Neurology 88(3):296–303
12. Fisch L, Lascano AM, Vernaz Hegi N, Girardin F, Kapina V, Heydrich L, Rutschmann O, Sarasin F, Vargas MI, Picard F, Vulliémoz S, Héritier-Barras AC, Seeck M (2016) Early specialized care after a first unprovoked epileptic seizure. J Neurol 263(12):2386–2394
13. Fisher RS, Acevedo C, Arzimanoglou A, Bogacz A, Cross JH, Elger CE, Engel J, Forsgren L, French JA, Glynn M, Hesdorffer DC, Lee BI, Mathern GW, Moshé SL, Perucca E, Scheffer IE, Tomson T, Watanabe M, Wiebe S (2014) ILAE official report: a practical clinical definition of epilepsy. Epilepsia 55(4):475–482
14. Fittipaldi F, Currà A, Fusco L, Ruggieri S, Manfredi M (2001) EEG discharges on awakening: a marker of idiopathic generalized epilepsy. Neurology 56(1):123–126
15. Hansen CK, Fisher J, Joyce N, Edlow JA (2011) Emergency department consultations for patients with neurological emergencies. Eur J Neurol 18(11):1317–1322
16. Hauser WA, Anderson VE, Loewenson RB, McRoberts SM (1982) Seizure recurrence after a first unprovoked seizure. N Engl J Med 307(9):522–528
17. Hauser WA, Beghi E (2008) First seizure definitions and worldwide incidence and mortality. Epilepsia 49(1):8–12
18. Herzig-Nichtweiß J, Salih F, Berning S, Malter MP, Pelz JO, Lochner P, Wittstock M, Günther A, Alonso A, Fuhrer H, Schönenberger S, Petersen M, Kohle F, Müller A, Gawlitza A, Gubarev W, Holtkamp M, Vorderwülbecke BJ (2023) Prognosis and management of acute symptomatic seizures: a prospective, multicenter, observational study. Ann Intensive Care 13(1):85
19. Hesdorffer DC, Benn EKT, Cascino GD, Hauser WA (2009) Is a first acute symptomatic seizure epilepsy? Mortality and risk for recurrent seizure. Epilepsia 50(5):1102–1108
20. Ho K, Lawn N, Bynevelt M, Lee J, Dunne J (2013) Neuroimaging of first-ever seizure: Contribution of MRI if CT is normal. Neurol Clin Pract 3(5):398–403
21. Holtkamp M (2023) Erster epileptischer Anfall und Epilepsien im Erwachsenenalter, S2k-Leitlinie. In: Berkenfeld R (ed) May TW* (*geteilte Erstautorenschaft)
22. Kim LG, Johnson TL, Marson AG, Chadwick DW (2006) Prediction of risk of seizure recurrence after a single seizure and early epilepsy: further results from the MESS trial. Lancet Neurol 5(4):317–322
23. King MA, Newton MR, Jackson GD, Fitt GJ, Mitchell LA, Silvapulle MJ, Berkovic SF (1998) Epileptology of the first-seizure presentation: a clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. Lancet 352(9133):1007–1011
24. Krumholz A, Wiebe S, Gronseth GS, Gloss DS, Sanchez AM, Kabir AA, Liferidge AT, Martello JP, Kanner AM, Shinnar S, Hopp JL, French JA (2015) Evidence-based guideline: Management of an unprovoked first seizure in adults: Report of the Guideline Development Subcommittee of the American Academy of Neurology and the American Epilepsy Society. Neurology 84(16):1705–1713
25. Lawn ND, Pang EW, Lee J, Dunne JW (2023) First seizure from sleep: Clinical features and prognosis. Epilepsia 64(10):2714–2724
26. Linka L, Magnus B, Habermehl L, Tsalouchidou P-E, Zahnert F, Möeller L, Krause K, Knake S, Menzler K (2023) Effect of the revised definition of epilepsy on treatment decisions and seizure recurrence after a first epileptic seizure. Eur J Neurol 30(6):1557–1564

27. Marson A, Burnside G, Appleton R, Smith D, Leach JP, Sills G, Tudur-Smith C, Plumpton C, Hughes DA, Williamson P, Baker GA, Balabanova S, Taylor C, Brown R, Hindley D, Howell S, Maguire M, Mohanraj R, Smith PE (2021) The SANAD II study of the effectiveness and cost-effectiveness of levetiracetam, zonisamide, or lamotrigine for newly diagnosed focal epilepsy: an open-label, non-inferiority, multicentre, phase 4, randomised controlled trial. Lancet 397(10282):1363–1374
28. Marson A, Jacoby A, Johnson A, Kim L, Gamble C, Chadwick D (2005) Immediate versus deferred antiepileptic drug treatment for early epilepsy and single seizures: a randomised controlled trial. Lancet 365(9476):2007–2013
29. Marson AG, Al-Kharusi AM, Alwaidh M, Appleton R, Baker GA, Chadwick DW, Cramp C, Cockerell OC, Cooper PN, Doughty J, Eaton B, Gamble C, Goulding PJ, Howell SJL, Hughes A, Jackson M, Jacoby A, Kellett M, Lawson GR, Leach JP, Nicolaides P, Roberts R, Shackley P, Shen J, Smith DF, Smith PEM, Smith CT, Vanoli A, Williamson PR (2007) The SANAD study of effectiveness of valproate, lamotrigine, or topiramate for generalised and unclassifiable epilepsy: an unblinded randomised controlled trial. Lancet 369(9566):1016–1026
30. Matz O, Zdebik C, Zechbauer S, Bündgens L, Litmathe J, Willmes K, Schulz JB, Dafotakis M (2016) Lactate as a diagnostic marker in transient loss of consciousness. Seizure 40:71–75
31. McGinty RN, Handel A, Moloney T, Ramesh A, Fower A, Torzillo E, Kramer H, Howell S, Waters P, Adcock J, Sen A, Lang B, Irani SR (2021) Clinical features which predict neuronal surface autoantibodies in new-onset focal epilepsy: implications for immunotherapies. J Neurol Neurosurg Psychiatry 92(3):291–294
32. McIntosh AM, Tan KM, Hakami TM, Newton MR, Carney PW, Yang M, Saya S, Marco DJT, Perucca P, Kwan P, O'Brien TJ, Berkovic SF (2021) Newly diagnosed seizures assessed at two established first seizure clinics: Clinic characteristics, investigations, and findings over 11 years. Epilepsia Open 6(1):171–180
33. Möller L, Krämer G, Habermehl L, Menzler K, Knake S (2023) Driving regulations for epilepsy in Europe. Seizure 109:83–91
34. Nass RD, Sassen R, Elger CE, Surges R (2017) The role of postictal laboratory blood analyses in the diagnosis and prognosis of seizures. Seizure 47:51–65
35. Neligan A, Heaney D, Rajakulendran S (2021) Is a separate clinical pathway for first seizures justified? Appraisal of the first seizure pathway at a tertiary neuroscience centre. Seizure 84:108–111
36. von Oertzen J, Urbach H, Jungbluth S, Kurthen M, Reuber M, Fernández G, Elger CE (2002) Standard magnetic resonance imaging is inadequate for patients with refractory focal epilepsy. J Neurol Neurosurg Psychiatry 73(6):643–647
37. Patel J, Tran QK, Martinez S, Wright H, Pourmand A (2022) Utility of serum lactate on differential diagnosis of seizure-like activity: A systematic review and meta-analysis. Seizure 102:134–142
38. Pellinen J, Tafuro E, Yang A, Price D, Friedman D, Holmes M, Barnard S, Detyniecki K, Hegde M, Hixson J, Haut S, Kälviäinen R, French J (2020) Focal nonmotor versus motor seizures: The impact on diagnostic delay in focal epilepsy. Epilepsia 61(12):2643–2652
39. Schubert, K. M., Zieglgänsberger, D., Bicciato, G., Abraira, L., Santamarina, E., Álvarez-Sabín, J., Ferreira-Atuesta, C., Katan, M., Sinka, L., Terziev, R., Deligas, N., Erdélyi-Canavese, B., Felbecker, A., Siebel, P., Winklehner, M., Oertzen, T. J. von, Wagner, J. N., Gigli, G. L., Nilo, A., Janes, F., Merlino, G., Valente, M., Zafra-Sierra, M. P., Mayor-Romero, L. C., Conrad, J., Evers, S., Alet, M., Fukuma, K., Ihara, M., Landau, B., Lochner, P., Roell, F., Brigo, F., Bentes, C., Peralta, A. R., Pinho E Melo, T., Keezer, M. R., Duncan, J. S., Sander, J. W., Tettenborn, B., Koepp, M. J., and Galovic, M. (2025) Association of the Timing and Type of Acute Symptomatic Seizures With Poststroke Epilepsy and Mortality. Stroke 56(7):1748–1757
40. Seeck M, Koessler L, Bast T, Leijten F, Michel C, Baumgartner C, He B, Beniczky S (2017) The standardized EEG electrode array of the IFCN. Clin Neurophysiol 128(10):2070–2077
41. de Stefano P, Ménétré E, Stancu P, Mégevand P, Vargas MI, Kleinschmidt A, Vulliémoz S, Wiest R, Beniczky S, Picard F, Seeck M (2023) Added value of advanced workup after the first seizure: A 7-year cohort study. Epilepsia 64(12):3246–3256
42. Süße M, Saathoff N, Hannich M, von Podewils F (2019) Cerebrospinal fluid changes following epileptic seizures unrelated to inflammation. Eur J Neurol 26(7):1006–1012
43. Tolchin B, Krauss GL, Spanaki MV, Joshi C, Pack AM, Krishnamurthy KB, Bonnie RJ (2025) Seizures, Driver Licensure, and Medical Reporting Update: An AAN Position Statement. Neurology 104(7):e213459
44. Yang M, Tan KM, Carney P, Kwan P, O'Brien TJ, Berkovic SF, Perucca P, McIntosh AM (2022) Diagnostic delay in focal epilepsy: Association with brain pathology and age. Seizure 96:121–127
45. Yardi R, Vasireddy RP, Galovic M, Punia V (2025) Antiseizure medication use in acute symptomatic seizures: A narrative review. Epilepsia 66(4):955–969

Zusammenfassung

Erster epileptischer Anfall im Jugendlichen- und Erwachsenenalter

Anfälle sind der dritthäufigste Vorstellungsgrund in der neurologischen Notaufnahme. Diagnose und Management von (ersten) Anfällen sind daher in jeder Neurologie von großer Bedeutung. Der vorliegende Artikel verschafft einen Überblick über die notwendige Diagnostik zum Ausschluss von akut behandlungsbedürftigen Ursachen, über die Abschätzung der Rezidivwahrscheinlichkeit zur Ermöglichung einer adäquaten Diagnosestellung und auch über die aktuellen Empfehlungen zur medikamentösen Therapie und Fahreignung.

Schlüsselwörter
Erster epileptischer Anfall · Diagnostik · Rezidivrisiko · Anfallssuppressive Medikation · Fahreignung