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An FP's guide to caring for patients with seizure and epilepsy

Optimizing your care requires that you distinguish between provoked and unprovoked seizures and focus on key elements of the patient's history.

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PRACTICE RECOMMENDATIONS

› *Consider treating a first-time seizure if electroencephalography shows particular epileptiform activity, if the neurologic exam or computerized tomography or magnetic resonance imaging results are abnormal, if the seizure is focal or nocturnal, or if there is a family history of seizures.* (A)

› *Consider valproate (except for women of childbearing age) and levetiracetam as first-line agents for generalized or unclassified epilepsy, and lamotrigine for focal epilepsies.* (A)

Strength of recommendation (SOR)

(A) Good-quality patient-oriented evidence

(B) Inconsistent or limited-quality patient-oriented evidence

(C) Consensus, usual practice, opinion, disease-oriented evidence, case series

Managing first-time seizures and epilepsy often requires consultation with a neurologist or epileptologist for diagnosis and subsequent management, including when medical treatment fails or in determining whether patients may benefit from surgery. However, given the high prevalence of epilepsy and even higher incidence of a single seizure, family physicians contribute significantly to the management of these patients. The main issues are managing a first-time seizure, making the diagnosis, establishing a treatment plan, and exploring triggers and mitigating factors.

Seizure vs epilepsy

All patients with epilepsy experience seizures, but not every person who experiences a seizure has (or will develop) epilepsy. Nearly 10% of the population has one seizure during their lifetime, whereas the risk for epilepsy is just 3%.[1] Therefore, a first-time seizure may not herald epilepsy, defined as repetitive (≥ 2) unprovoked seizures more than 24 hours apart.[2] Seizures can be provoked (acute symptomatic) or unprovoked; a clear distinction between these 2 occurrences—as well as between single and recurrent seizures—is critical for proper management. A close look at the circumstances of a first-time seizure is imperative to define the nature of the event and the possibility of further seizures before devising a treatment plan.

Provoked seizures are due to an acute brain insult such as toxic-metabolic disorders, concussion, alcohol withdrawal, an adverse effect of a medication or its withdrawal, or photic stimulation presumably by disrupting the brain's metabolic homeostasis or integrity. The key factor is that provoked seizures always happen in close temporal association with an acute insult. A single provoked seizure happens each year in 29 to 39 individuals per 100,000.[3] While these seizures typically occur singly, there is a small risk they may recur if the triggering insult persists or re-

peats.[1] Therefore, more than 1 seizure per se may not indicate epilepsy.[3]

Unprovoked seizures reflect an underlying brain dysfunction. A single unprovoked seizure happens in 23 to 61 individuals per 100,000 per year, often in men in either younger or older age groups.[3] Unprovoked seizures may occur only once or may recur (ie, evolve into epilepsy). The latter scenario happens in only about half of cases; the overall risk for a recurrent seizure within 2 years of a first seizure is estimated at 42% (24% to 65%, depending on the etiology and electroencephalogram [EEG] findings).[4] More specifically, without treatment the relapse rate will be 36% at 1 year and 47% at 2 years.[4] Further, a second unprovoked seizure, if untreated, would increase the risk for third and fourth seizures to 73% and 76%, respectively, within 4 years.[3]

Evaluating the first-time seizure

Ask the patient or observers about the circumstances of the event to differentiate provoked from unprovoked onset. For one thing, not all "spells" are seizures. The differential diagnoses may include syncope, psychogenic nonepileptic events, drug intoxication or withdrawal, migraine, panic attacks, sleep disorders (parasomnia), transient global amnesia, concussion, and transient ischemic attack. EEG, neuroimaging, and other relevant diagnostic tests often are needed (eg, electrocardiogram/echocardiogram/Holter monitoring to evaluate for syncope/cardiac arrhythmia). Clinically, syncopal episodes tend to be brief with rapid recovery and no confusion, speech problems, aura, or lateralizing signs such as hand posturing or lip smacking that are typical with focal seizures. However, cases of convulsive syncope can be challenging to assess without diagnostic tests.

True convulsive seizures do not have the variability in clinical signs seen with psychogenic nonepileptic events (eg, alternating body parts involved or direction of movements). Transient global amnesia is a rare condition with no established diagnostic test and is considered a diagnosis of exclusion, although bitemporal hyperintensities on magnetic resonance imaging (MRI) may appear 12 to 48 hours after the clinical episode.[5] Blood work is needed in patients with medical issues treated with multiple medications to evaluate for metabolic derangements; otherwise, routine blood work provides minimal information in stable patients.

Region-specific causes. Neurocysticercosis is common in some regions, such as Latin America; therefore, attention should be paid to this aspect of patient history.

Is it really a first-time seizure? A "first," usually dramatic, generalized tonic-clonic seizure that triggers the diagnostic work-up may not be the very first seizure. Evidence suggests that many patients have experienced prior undiagnosed seizures. Subtle prior events often missed include episodes of *deja vu*, transient feelings of fear or unusual smells, speech difficulties, staring spells, or myoclonic jerks.[1] A routine EEG to record epileptiform discharges and a high-resolution brain MRI to rule out any intracranial pathology are indicated. However, if the EEG indicates a primary generalized (as opposed to focal-onset) epilepsy, a brain MRI may not be needed. If a routine EEG is unrevealing, long-term video-EEG monitoring may be needed to detect an abnormality.

Accuracy of EEG and MRI. Following a first unprovoked seizure, routine EEG to detect epileptiform discharges in adults has yielded a sensitivity of 17.3% and specificity of 94.7%. In evaluating children, these values are 57.8% and 69.6%, respectively.[6] If results are equivocal, a 24-hour EEG can increase the likelihood of detecting epileptiform discharges to 89% of patients.[7] Brain MRI may detect an abnormality in 12% to 14% of patients with newly diagnosed epilepsy, and in up to 80% of those with recurrent seizures.[8] In confirming hippocampus sclerosis, MRI has demonstrated a sensitivity of 93% and specificity of 86%.[9]

When to treat a first-time seizure. Available data and prediction models identify risk factors that would help determine whether to start an antiseizure medication after a first unprovoked seizure: abnormal EEG with particular epileptiform activity, abnormal neurologic exam, abnormal computerized tomography or MRI results, nocturnal seizure, focal seizure, or family history of sei-

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zures. In the absence of such risk factors, chances of further unprovoked seizures are not high enough to justify treatment with antiseizure medications. However, if a second unprovoked seizure were to occur, that would meet the definition of epilepsy, and treatment is indicated due to the high risk for further seizures.[10,11]

Epilepsy diagnosis

The International League Against Epilepsy (ILAE) previously defined epilepsy as 2 unprovoked seizures more than 24 hours apart. However, a more recent ILAE task force modified this definition: even a single unprovoked seizure would be enough to diagnose epilepsy if there is high probability of further seizures—eg, in the presence of definitive epileptiform discharges on EEG or presence of a brain tumor or a remote brain insult on imaging, since such conditions induce an enduring predisposition to generate epileptic seizures.[2] Also, a single unprovoked seizure is enough to diagnose epilepsy if it is part of an epileptic syndrome such as juvenile myoclonic epilepsy. Further, a time limit was added to the definition—ie, epilepsy is considered resolved if a patient remains seizure free for 10 years without use of antiseizure medications during the past 5 years. However, given the multitude of variables and evidence, the task force acknowledged the need for individualized considerations.[2]

Seizure classification

Classification of seizure type is based on the site of seizure onset and its spread pattern—ie, focal, generalized, or unknown onset.

Focal-onset seizures originate "within networks limited to one hemisphere," although possibly in more than 1 region (ie, multifocal, and presence or absence of loss of awareness).[12] Focal seizures may then be further classified into "motor onset" or "nonmotor onset" (eg, autonomic, emotional, sensory).[2]

Generalized seizures are those "originating at some point within, and rapidly engaging, bilaterally distributed networks."[13] Unlike focal-onset seizures, generalized seizures are not classified based on awareness, as most generalized seizures involve loss of awareness (absence) or total loss of consciousness (generalized tonic-clonic). They are instead categorized based on the presence of motor vs nonmotor features (eg, tonic-clonic, myoclonic, atonic). Epilepsy classification is quite dynamic and constantly updated based on new genetic, electroencephalographic, and neuroimaging discoveries.

Treatment of epilepsy

Antiseizure medications

Treatment with antiseizure medications (ASMs; formerly known as *antiepileptic drugs*) is the mainstay of epilepsy management. Achieving efficacy (seizure freedom) and tolerability (minimal adverse effects) are the primary goals of treatment. Factors that should govern the selection of an ASM include the seizure type/epilepsy syndrome, adverse effect profile of the ASM, pharmacodynamic/pharmacokinetic considerations, and patient comorbidities.

The Standard and New Antiepileptic Drugs (SANAD I and II) trials provide data from direct, unblinded, and longitudinal comparisons of existing and new ASMs and their utility in different seizure types. In the SANAD I cohort of patients with generalized and unclassified epilepsies, valproate was superior to lamotrigine and topiramate for 12-month remission and treatment failure rates, respectively.[14] However, valproate generally is avoided in women of childbearing age due its potential adverse effects during pregnancy. In focal epilepsies, lamotrigine was superior to carbamazepine, gabapentin, and topiramate with respect to treatment failure, and noninferior to carbamazepine for 12-month remission.[15] In the SANAD II trial, levetiracetam was noninferior to valproate for incidence of adverse events in patients with generalized and unclassified epilepsies although was found to be neither more clinically effective nor more cost effective.[16] For patients of childbearing potential with generalized and unclassified epilepsies, there is evidence to support the safe and effective use of levetiracetam.[17] In focal epilepsies, lamotrigine was superior to levetiracetam and zonisamide with respect to treatment failures and adverse events and was noninferior to zonisamide for 12-month remission.[18] In

Levetiracetam and valproate (not to be used in women of childbearing potential) are considered appropriate first-line agents for generalized and unclassified epilepsies.

summary, levetiracetam and valproate (not to be used in women of childbearing potential) are considered appropriate first-line agents for generalized and unclassified epilepsies while lamotrigine is deemed an appropriate first-line agent for focal epilepsies (**TABLE 1**[19-28]).

Drug level monitoring. It is standard practice to periodically monitor serum levels in patients taking first-generation ASMs such as phenytoin, carbamazepine, phenobarbital, and valproic acid because of their narrow therapeutic range and the potential for overdose or interaction with other medications or foods (eg, grapefruit juice may increase carbamazepine serum level by inhibiting CYP3A4, the enzyme that metabolizes the drug). Patients taking newer ASMs may not require regular serum level monitoring except during titration, with hepatic or renal dosing, when concomitantly used with estrogen-based oral contraceptives (eg, lamotrigine), before or during pregnancy, or when nonadherence is suspected.

Can antiseizure treatment be stopped?

Current evidence favors continuing ASM therapy in patients whose seizures are under control, although the decision should be tailored to an individual's circumstances. According to the 2021 American Academy of Neurology (AAN) guidelines, adults who have been seizure free for at least 2 years and discontinue ASMs are possibly still at higher risk for seizure recurrence in the long term (24-60 months), compared with those who continue treatment.[29] On the other hand, for adults who have been seizure free for at least 12 months, ASM withdrawal may not increase their risk for status epilepticus, and there are insufficient data to support or refute an effect on mortality or quality of life with ASM withdrawal in this population. The decision to taper or maintain ASM therapy in seizure-free patients also should take into consideration other clinically relevant outcome measures such as the patient's lifestyle and medication adverse effects. Therefore, this decision should be made after sufficient discussion with patients and their caregivers. (Information for patients can be found at: www.epilepsy.com/treatment/medicines/stopping-medication.)

For children, the AAN guideline panel recommends discussing with family the small risk (2%) for becoming medication resistant if seizures recur during or after ASM withdrawal.[29] For children who have been seizure free for 18 to 24 months, there is probably not a significant long-term (24-48 months) difference in seizure recurrence in those who taper ASMs vs those who do not. However, presence of epileptiform discharges on EEG before discontinuation of an ASM indicates increased risk for seizure recurrence.[29]

Intractable (refractory) epilepsy

While most patients with epilepsy attain complete seizure control with appropriate drug therapy, approximately 30% continue to experience seizures ("drug-resistant" epilepsy, also termed *intractable* or *refractory*).[30] In 2010, the ILAE defined drug-resistant epilepsy as "failure of adequate trials of two tolerated, appropriately chosen and used anti-epileptic drug schedules (whether as monotherapy or in combination) to achieve sustained seizure freedom" (defined as cessation of seizures for at least 3 times the longest pre-intervention inter-seizure interval or 12 months, whichever is longer).[21,31] It should be noted that drug withdrawal due to adverse effects is not counted as failure of that ASM. Recognition of drug-resistant epilepsy may prompt referral to an epileptologist who can consider rational combination drug therapy or surgical resection of the seizure focus, vagus nerve stimulation, electrical stimulation of the seizure focus, or deep brain (thalamic) stimulation.

Seizure triggers and mitigating factors

Epilepsy mostly affects patients during seizure episodes; however, the unpredictability of these events adds significantly to the burden of disease. There are no reliable methods for predicting seizure other than knowing of the several potential risks and recognizing and avoiding these triggers.

Noncompliance with antiseizure medications is a common seizure trigger affecting up to one-half of patients with epilepsy.[32]

Medications may provoke seizures in susceptible individuals (**TABLE 2**[33-35]).

Sleep deprivation is a potential sei-

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TABLE 1

Commonly prescribed antiseizure medications assessed in the SANAD trials[19-28]

Medication (brand names) Indication(s)	Adverse effects	Mechanism and kinetics	Clinical considerations	Common dosing,[a] efficacy
Valproate[19,20] (Depakene, Depakote, Epilim, Stavzor) For generalized-onset seizures, also effective for focal-onset seizures	Dose related: gastrointestinal upset, tremor, confusion; idiosyncratic: hair loss, weight gain, polycystic ovaries, hyperammonemia, hepatotoxicity	Inhibits T-type calcium- and voltage-dependent sodium channels; increases GABA concentration; 90% protein-bound; metabolized in liver	Risk for teratogenicity (eg, spina bifida) and neurodevelopmental delay of offspring for WWE if used during pregnancy	Maintenance: 15-60 mg/kg/d SE dosing: 20-40 mg/kg IV over 10 mins 50% responder rate[b] at > 3 mo: 50-80%[21]
Lamotrigine[19,22] (Lamictal, Subvenite) For focal-onset seizures, also effective for generalized-onset seizures (may exacerbate myoclonus and absences)	Dose related: drowsiness, insomnia, headache; idiosyncratic: rash (~ 3.5% of patients) that may be severe (SJS)	Stabilizes voltage-dependent sodium channels; 50% protein-bound; metabolized in liver	Slow initial titration (4-6 wk) to avoid rash; initial concern for cleft defect in offspring of WWE; serum concentration decreases during pregnancy	Maintenance: 100-500 mg/d (dose up titration over 6-8 wk) Not used for SE 50% responder rate[b]: 20%-66.7%[23]
Topiramate[24] (Topamax, Trokendi, Qudexy) For focal-onset seizures, also effective for generalized-onset seizures	Dose related: drowsiness, slowing of cognition and articulation; idiosyncratic: weight loss, kidney stones	Blocks voltage-gated sodium channels, AMPA, and kainate receptors, carbonic anhydrase; enhances GABA	Used in migraine prevention and weight loss	Maintenance: 200-400 mg/d (dose up titration over 4-6 wk) Not used for SE 50% responder rate[b]: 35%-47.8%[23]
Levetiracetam[19,25] (Keppra, Roweepra, Spritam) For focal-onset seizures, also effective for generalized-onset seizures (preferred to valproate in WWE during pregnancy)	Dose related: fatigue; idiosyncratic: irritability, anxiety, and mood changes	Binds to synaptic vesicle glycoprotein 2A; not protein bound; excreted by kidneys largely unchanged	Widely considered to have no significant medication interactions; good safety profile in pregnancy	Maintenance: 1000-3000 mg/d SE dosing: 60 mg/kg IV over 15 mins 50% responder rate[b]: 31.6%-48.1%[23]
Carbamazepine[26] (Carbatrol, Epitol, Equetro, Tegretol) For focal seizures (may exacerbate myoclonus and absences)	Dose related: fatigue; idiosyncratic: irritability, anxiety, and mood changes	Stabilizes voltage-gated sodium channels; strongly protein-bound; metabolized in liver	Monitor for hyponatremia in elderly or patients taking diuretics; HLA genotyping needed for Asian patients (B*1502 mutation) to identify risk for SJS	Maintenance: 800-1200 mg/d Not used for SE Seizure free at 26 wk: 75%[21] Seizure free at 48 wk: 38%[21] Seizure free at 48-96 wk: 34%[21]
Gabapentin[27] (Neurontin, Gralise) For focal seizures	Dose related: sedation, ataxia, dizziness; idiosyncratic: peripheral edema, tremor	Binds presynaptic GABA subunit of calcium channel	Lower dose required in renal insufficiency; concentration may increase with morphine	Maintenance: 900-1800 mg/d Not used for SE 50% responder rate[b]: 25%-33%[23]
Zonisamide[28] (Zonegran) For focal-onset seizures; also effective for generalized-onset seizures	Dose related: sedation, ataxia, dizziness; idiosyncratic: rash, kidney stones, heatstroke	Blocks voltage-gated sodium channels, T-type calcium currents, glutamate transmission, carbonic anhydrase	May increase contraceptive clearance	Maintenance: 400-600 mg/d Not used for SE 50% responder rate[b]: 29.9%-43%[23]

AMPA, α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; GABA, gamma-aminobutyric acid; HLA, human leukocyte antigen; SANAD, Standard and New Antiepileptic Drugs; SE, status epilepticus; SJS, Stevens-Johnson syndrome; WWE, women with epilepsy.

[a] Clinical circumstances often require boosting the approved maximum dose.

[b] 50% responder rate indicates ≥ 50% reduction in seizure frequency.

zure trigger in people with epilepsy based on observational studies, case reports, patient surveys, and EEG-based studies, although data from randomized controlled studies are limited.[36] The standard best practice is to encourage appropriate sleep hygiene, which involves getting at least 7 hours of sleep per night.[37]

Alcohol is a GABAergic substance like benzodiazepines with antiseizure effects. However, it acts as a potential precipitant of seizures in cases of withdrawal or acute intoxication, or when it leads to sleep disruption or nonadherence to antiseizure medications. Therefore, advise patients with alcohol use disorder to slowly taper consumption (best done through a support program) and avoid sudden withdrawal. However, complete abstinence from alcohol use is not often recommended except in special circumstances (eg, a history of alcohol-related seizures). Several studies have demonstrated that modest alcohol use (1-2 drinks per occasion) does not increase seizure frequency or significantly alter serum concentrations of commonly used ASMs.[38]

Cannabis and other substances. The 2 main biologically active components of marijuana are delta-9-tetrahydrocannibinol (THC), the main psychoactive constituent, and cannabidiol (CBD). Animal and human studies have demonstrated anticonvulsant properties of THC and CBD. But THC, in high amounts, can result in adverse cognitive effects and worsening seizures.[39] A purified 98% oil-based CBD extract (Epidiolex) has been approved as an adjunctive treatment for certain medically refractory epilepsy syndromes in children and young adults—ie, Dravet syndrome, Lennox-Gastaut syndrome, and tuberous sclerosis complex syndrome.[40] There are no reliable data on the effect of recreational use of marijuana on seizure control. Other illicit substances such as cocaine may lower seizure threshold by their stimulatory and disruptive effects on sleep, diet, and healthy routines.

Special clinical cases

Pregnancy and epilepsy

Despite the potential adverse effects of ASMs on fetal health, the current global consensus is to continue treatment during pregnancy, given that the potential harm of convulsive seizures outweighs the potential risks associated with in-utero exposure to ASMs. There is not enough evidence to indicate significant harm to the fetus caused by focal, absence, or myoclonic seizures. Low-dose folic acid is used to minimize the risks of ASMs during pregnancy.

As the fetus develops, there are changes in volume of ASM distribution, renal clearance, protein binding, and hepatic metabolism, which require checking serum levels at regular intervals and making dosage adjustments.

The ongoing study evaluating Maternal Outcomes and Neurodevelopmental Effects of Antiepileptic Drugs (MONEAD)[41] has led to multiple landmark studies guiding the choice of preferred ASMs during pregnancy in patients with epilepsy.[42,43] This has culminated in today's use of lamotrigine and levetiracetam as the 2 preferred agents (while avoiding valproate) in pregnant patients with epilepsy.[44]

Psychogenic nonepileptic seizures

A form of conversion disorder, psychogenic nonepileptic seizures (PNES) manifests as abnormal motor or behavioral events mimicking seizures but without associated epileptiform discharges on EEG. This is observed in 10% of patients seen in epilepsy clinics and even more often in those admitted to epilepsy monitoring units (25%-40%).[45] Diagnosis of PNES requires EEG monitoring both for confirmation and for discernment from true epileptic seizures, in particular frontal lobe epilepsy that may clinically mimic PNES. PNES often is associated with underlying psychological tensions or comorbid conditions such as depression, anxiety, or traumatic life experiences. There is no treatment for PNES per se, and its management is focused on controlling any underlying psychological comorbidities that may not always be obvious. There is some evidence suggesting that these patients experience an innate inability to verbally express their emotions and instead subconsciously resort to psychosomatics to express them in a somatic dimension.[46,47]

CONTINUED

Illicit substances such as cocaine may lower seizure threshold by their stimulatory and disruptive effects on sleep, diet, and healthy routines.

TABLE 2

Medications with potential to trigger seizures[33-35]

Antimicrobial agents[33,a]	Chemotherapy agents[34]	Psychotropic medications[35]	Antiepileptics
Ciprofloxacin	Cisplatin	*At therapeutic doses*:	*Paradoxical increased risk for seizure in specific cases:*
Imipenem	Methotrexate	Bupropion	Carbamazepine used in absence or myoclonic epilepsy
Beta-lactam antibiotics (eg, penicillins, cephalosporins, fluorochinolons, carbapenems)	Interferon alpha	Clomipramine	
	Busulphan	Maprotiline	
	Chlorambucil	Chlorpromazine	
	5-florouracil	Clozapine	
	Cyclosporin A		

[a] Via interaction with inhibitory GABA (gamma-aminobutyric acid)-A receptor. Risk might be compounded by comorbidities such as renal dysfunction, brain lesions, epilepsy, and the underlying infection that can produce cytokines that lower seizure threshold.

Status epilepticus

Defined as prolonged seizures (> 5 min) or 2 consecutive seizures without regaining awareness in between, status epilepticus (SE) is a potentially fatal condition. Subclinical nonconvulsive SE, especially in comatose patients, can be diagnosed only via EEG monitoring. Untreated SE may manifest as a diagnostic dilemma in unresponsive or critically ill patients and can increase the risk for mortality.[48]

Febrile seizures

Febrile seizures affect 2% to 5% of children most often in the second year of life.[49] The use of preventive antiseizure medication is not recommended; instead, the key is to investigate the underlying febrile illness. Lumbar puncture is indicated if there are signs and symptoms of meningitis (25% of children with bacterial meningitis present with seizures).[49] Febrile seizures often are self-limited, but there is risk for SE in up to 15% of cases.[50] If convulsive febrile seizures last longer than 5 minutes, initiate benzodiazepines followed by the standard protocol used for the management of SE.[51]

Epilepsy as a spectrum disorder

The higher prevalence of comorbid cognitive and psychiatric conditions in patients with epilepsy, affecting about half of patients,[52] suggests that seizures may constitute only one aspect of a multifaceted disease that otherwise should be considered a spectrum disorder. Among such conditions are memory deficits, depression, and anxiety. Conversely, epilepsy is more common in patients with depression than in those without.[52]

Social impact of epilepsy

Vehicle driving regulations. Patients with epilepsy are required to follow state law regarding driving restrictions. Different states have different rules and regulations about driving restrictions and reporting requirements (by patients or their physicians). Refer patients to the Department of Motor Vehicles (DMV) in their state of residence for up-to-date instructions.[53] The Epilepsy Foundation (epilepsy.com) can serve as a resource for each state's DMV website.

Employment assistance. Having epilepsy should not preclude patients from seeking employment and pursuing meaningful careers. The Americans with Disabilities Act (ADA) and the US Equal Employment Opportunity Commission (EEOC) forbid discrimination against qualified people with disabilities, including those with epilepsy, and require reasonable accommodations in the workplace (www.eeoc.gov/laws/guidance/epilepsy-workplace-and-ada).[54] JFP

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Having epilepsy should not preclude patients from seeking employment and pursuing meaningful careers.

WHAT'S NEW

Equivalence in pain and function scores

This trial showed equivalence in pain at 3 days' follow-up in children with distal radius torus fractures who were offered bandaging and then immediately discharged from the ED, compared with rigid immobilization and clinical follow-up. There were no significant differences in pain or function between groups during the 6 weeks following the initial injury. De-escalation of treatment offers an equivalent, resource-sparing alternative to traditional treatment of these fractures.

CAVEATS

Lack of masking likely introduced bias

There are no major caveats associated with managing distal radius torus fractures with a soft bandage and discharge from the ED, compared with the traditional treatment of rigid immobilization. However, bias was likely introduced in patient-reported outcomes due to the inability to mask patients and families to the treatment allocation. This may have led to overstating the severity of outcomes in the bandage group, given the strong preference for rigid immobilization, although equivalence was illustrated despite this potential bias.

CHALLENGES TO IMPLEMENTATION

Preferences may be difficult to change

Parents and clinicians demonstrated a preference for rigid immobilization, as shown in the imbalance in treatment crossovers, with 7% of children changing to the rigid immobilization group by the primary study end point of 3 days. The study authors hypothesized that crossovers may have been due to the perception by some parents that rigid immobilization is the gold standard of treatment, as well as clinicians' seeking to escalate care for patients returning for follow-up. Policy and guideline changes, as well as physician efforts to educate patients on outcomes with soft bandage treatment, are likely to improve these misconceptions. JFP

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