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How Do I Evaluate a First-Time Seizure?

Seeing a patient for a possible first seizure is an everyday consult in both outpatient and inpatient neurology. It is important to recognize that a first seizure is a time of high stress for the patient and their family. Essentially, they will look to you for five "answers" to the following questions:

1. Why did I have a seizure?
2. Will I have more seizures (i.e., do I have epilepsy)?
   a. If I have epilepsy, what kind of epilepsy do I have?
   b. If I do not have epilepsy, what is the diagnosis?
3. What kind of testing do I need to undergo?
4. Do I need to take antiseizure medicines (ASMs), and if so, for how long?
5. How will this affect my life?

To answer these questions, you will first need to answer several other questions for yourself through the history from the patient. To make an accurate diagnosis, the questions you will ask yourself are:

1. What history should I take to make a diagnosis?
2. If it is not a seizure, what are other possible diagnoses?
3. What further work up should I order?
4. Do I need to start an ASM?
5. How do I counsel the patient about their questions?

While ordering objective tests is a routine part of neurological care, *obtaining an excellent history is the most direct and impactful intervention you can make immediately on seeing the patient.* Therefore, this chapter focuses significantly

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on obtaining a specific epilepsy history that allows you to generate an accurate differential, proceed with an evidence-based evaluation, consider ASM therapy, and provide appropriate counseling to your patient.

Taking the Right History in an Efficient Manner

As with all medical care, obtaining an accurate diagnosis begins with a history. *An accurate history can lead to a specific epilepsy diagnosis nearly 50% of the time*, compared favorably to the diagnosis rate of electroencephalogram (EEG) at 30% [1]. An accurate seizure history can be obtained with essentially two critical pieces of information:

1. A thorough seizure semiology history
2. Assessment of epilepsy or seizure risk factors.

IMPROVING YOUR SEIZURE HISTORY TO EFFICIENTLY UTILIZE YOUR TIME

An accurate seizure semiology history can approximate the brain area that produces clinical symptoms during a patient's seizure, aka the symptomatogenic zone [2]. Seizure semiology is a description of the patient's subjective feelings as well as objective behaviors and movements during seizures. Thus, the first symptom in a patient's seizure description is often, although not always, quite close to where the seizure begins and is of fundamental interest. Therefore, the most important question you can ask a patient is:

What is the first thing that happens when you have a seizure?

When you ask this question, often a patient will begin at the end of the seizure, typically the tonic clonic portion, and likely the most traumatic part of the first seizure experience to patients and their loved ones alike.

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It is key to acknowledge the tonic clonic portion as you gently guide the patient back to the beginning of the seizure where often the most localizing history can be obtained.

After establishing the first seizure symptom, it is useful to proceed with questions like: *What happens next?* This question can be asked multiple times until the patient describes the seizure's end.

Once the patient's complete seizure recollection is obtained, one can ask the patient specific questions regarding typical seizure auras (i.e., Do you have strange tastes out of nowhere?) to elicit history of a potential gustatory aura) since many patients do not understand that those symptoms are part of their seizure until identified by you.

Moreover, a history of auras in isolation that precede a first bilateral tonic clonic seizure can establish an epilepsy diagnosis since the patient has already had more than two seizures. Indeed, nearly three-quarters of patients have had "small" seizures prior to a first generalized tonic clonic (GTC) seizure [3]. Identifying this crucial history makes the decision to start ASM therapy straightforward (discussed later in this chapter).

Lastly, clinical history from the period after a seizure concludes (the postictal period) can provide invaluable clues to the patient's epilepsy diagnosis. Here, questions can probe specific neurological dysfunction, the two most common being postictal weakness and aphasia.

Unilateral postictal weakness (Todd's paralysis) reliably lateralizes seizure onset to the brain hemisphere contralateral to the weakness. For example, left postictal weakness lateralizes to the right cerebral hemisphere. Postictal aphasia lateralizes to the language-dominant hemisphere, most commonly the left hemisphere [4].

In summary, a neurologist can divide a seizure into four possible phases as the history is obtained. Doing so can provide further organization that makes understanding a patient's seizure progression more intuitive.

1. A beginning portion where the patient is aware (auras; Table 1.1)
2. A portion where the patient is unaware (Tables 1.2 and 1.3)

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Table 1.1 Sensory seizures[a]

Common patient descriptors	Seizure Semiology Classification [5]	2017 seizure classification [6,7]
Déjà vu, panic, anxiety, hallucinations, fear, unease	Psychic aura	Focal aware cognitive seizure *or* Focal aware emotional seizure
Foul smell like rotten eggs, sulfur	Olfactory aura	Focal aware sensory seizure
Sometimes foul or metallic taste	Gustatory aura	Focal aware sensory seizure
Rising or flipping sensation of the stomach, mild nausea	Abdominal aura	Focal aware sensory seizure *or* Focal aware autonomic seizure
Flushing, intense nausea, choking sensation, palpitations, hair standing on end	Autonomic aura	Focal aware autonomic seizure
Tingling, prickling; less commonly numbness	Somatosensory aura	Focal aware sensory seizure
Tones, clicks, basic sounds; less commonly complex sounds like music	Auditory aura	Focal aware sensory seizure
Flashing or swirling lights	Visual aura	Focal aware sensory seizure

Symptoms and the 1998 seizure name should include a laterality modifier when appropriate with options including left, right, axial, or generalized. The 2017 seizure classification does not include laterality modifiers.

[a] Also commonly called auras (focal aware seizures)

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Table 1.2 Seizures primarily affecting consciousness/behavior

Common patient descriptors	Seizure Semiology Classification [5]	2017 seizure classification [6, 7]
Unresponsiveness, incorrect answers to questions, space out, blank out, repetitive, or simple responses (yeah, no, etc.)	Dialeptic seizure	Focal behavior arrest seizure *or* Unknown onset behavior arrest seizure
Unresponsiveness, space out, blank out, repetitive, or simple responses (yeah, no, etc.)	Absence seizure	Generalized absence seizure
Lip smacking, "acting weird," repetitive hand/finger movements, drooling, repetitive swallowing	Automotor seizure	Focal automatisms seizure
Flailing, running, kicking, boxing, punching, screaming, "crazy" movements	Hypermotor seizure	Focal hyperkinetic seizure
Laughing, giggling, "creepy" laugh	Gelastic seizure	Focal emotional seizure
Being unable to speak as the primary seizure manifestation with retained awareness	Aphasic seizure	Focal cognitive seizure

All 2017 seizure classification seizures should have either "aware" or "impaired awareness" following focal depending on patient awareness of symptoms.

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3. A portion where the seizure propagates throughout the entire brain (secondary generalization with bilateral tonic clonic seizure; Table 1.3)
4. The postictal period (the portion after the seizure concludes).

Table 1.3 Seizures with primary motor manifestations

Common patient descriptors	Seizure Semiology Classification [5]	2017 seizure classification [6, 7]
Stiffen	Tonic	Tonic[a]
Shake	Clonic	Clonic[a]
Head turn, eye turn	Versive	Versive[a]
Muscle jerk	Myoclonic	Myoclonic[a]
Stiffen and shake all over, begin with a loud yell (ictal cry)	Tonic clonic	Tonic clonic
Fall, "just drop," head drop	Atonic	Atonic[a]
Arms stiffen and then hunching over, most commonly in the truncal areas	Epileptic spasm	Generalized onset epileptic spasm *or* Unknown onset epileptic spasm
Can have any variety of previously described seizure types that evolve to a bilateral tonic clonic seizure	n/a[b]	Focal to bilateral tonic clonic seizure

[a] The 2017 seizure classification should be preceded by either focal onset or generalized onset as appropriately determined by other testing except for "tonic clonic" and "epileptic spasm," which can be either generalized or unknown onset while "behavior arrest" can be focal onset or unknown onset.

[b] The 1998 classification does not have a correlate to "focal to bilateral tonic clonic seizure" as the 1998 classification lists all relevant seizure types from which it evolves. Common patient descriptors and 1998 seizure name should include laterality modifiers when appropriate with options including left, right, axial, or generalized. The 2017 seizure classification does not include laterality modifiers.

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SEIZURE CLASSIFICATION SYSTEMS

After obtaining the seizure semiology history, you should have a clear narrative of how the patient and bystanders perceived the seizure from beginning to end. You then "translate" the described movements/behaviors of a seizure into specific seizure types from which you can more easily make a localization.

There are two seizure classification systems for seizure semiology that over time have increased in similarity [5, 6]. The Seizure Semiology Classification (SSC) uses semiology on its own without reference to imaging or electrophysiologic data. In essence, the SSC recognizes semiology as its own discrete data point with significant potential to inform a specific epilepsy diagnosis [5].

The International League Against Epilepsy (ILAE) system by contrast combines the seizure type with an electrophysiologic implication (generalized versus focal) as part of the classification itself [6]. Still, removing the electrophysiologic implication from the ILAE seizure classification yields a similar description to the SSC; hence why overlap is increasing.

The SSC additionally benefits from emphasizing the progression of seizure symptoms [8]. For instance, people with temporal lobe epilepsy can have a classic progression of metallic taste (gustatory aura) to unresponsiveness with lip smacking and pill-rolling finger movements (automotor seizure) to a bilateral tonic clonic seizure (aka grand mal seizure, GTC). By contrast, the ILAE system defines each seizure separately without reference to progression, potentially obscuring key information that informs a specific epilepsy diagnosis. The earlier described seizure would be termed a focal to bilateral tonic clonic seizure, unfortunately losing some of the rich description that allows for the localization process so familiar to all neurologists.

Accordingly, a third key difference is that the SSC encourages establishment of a specific anatomic localization whereas the ILAE

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system, by definition, limits to one of three localizations: focal, generalized, and unknown [6].

All of those factors reviewed, the authors assert that epilepsy patients benefit from a specific and actionable localization or syndrome diagnosis, similar to any neurological diagnosis. Since the SSC urges the neurologist to establish a localization, we will primarily discuss that classification. However, we do appropriately reference the most current ILAE system in Tables 1.1–1.5 recognize that many neurologists use the ILAE system in everyday practice. As noted, it is worth being conversant in both classifications in case patients or other physicians alike use those seizure types. In the end, *what is most important is that the patient receives a specific and actionable diagnosis*, whether epilepsy, nonepileptic events, or a nonneurological diagnosis like syncope.

EVOLUTION OF ILAE SEIZURE TERMINOLOGY

Before further discussing the SSC, it is worth reviewing the evolution of the ILAE seizure classification since previous terminology continues in widespread use. Perhaps the best-known ILAE classification remains the 1981 version [9], which introduced the terms "simple partial" and "complex partial." The 2017 version changes verbiage without substantive change in meaning (Table 1.4). Thus, it is worth being fluent in the 1981 and 2017 ILAE systems since both are used.

Table 1.4 1981 and 2017 ILAE terminology

1981 terms [9]	2017 terms [6]
Partial	Focal
Simple	Aware
Complex	Impaired awareness or unaware
Secondary generalization	Focal to bilateral tonic clonic

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SEIZURE SEMIOLOGY CLASSIFICATION SYSTEM

The SSC utilizes common localization elements from other neurological diagnoses like stroke while expanding on localizations specific to epilepsy [5]. It is useful to break down seizures into four main categories, with the fourth category being less common than the first three:

1. Sensory – commonly called auras; these are subjective experiences of which only the patient is aware (Table 1.1)
2. Consciousness – changes in patient behavior or responsiveness that can be objectively observed and, at times, variably noted by the patient. One novel term of note, dialeptic, is introduced by the SSC and means altered awareness/consciousness as the only manifestation of the seizure. An intuitive alternate term to use here is dyscognitive (Table 1.2)
3. Motor – specific stereotyped movements that can be observed and, at times, noted by the patient (Table 1.3)
4. Autonomic – a less common seizure type where symptoms affect the autonomic nervous system, whether subjective (considered an aura, i.e., palpitations) or objective (then considered a seizure, i.e., tachycardia).

For each seizure type, one should describe the localization of the behavior/movement as well as the laterality, if applicable. We provide four examples of how you would translate a patient history into a seizure type with its commensurate localization.

1. A patient tells you their right arm tingles at seizure onset. You would note a right arm somatosensory seizure, which concisely localizes to the left parietal lobe (Tables 1.1 and 1.5).
2. A patient describes flashing lights in the left peripheral vision. You "translate" to a left visual aura with a most likely localization to the right occipital lobe (Tables 1.1 and 1.5).
3. You observe a seizure with stereotyped posture of the left arm extended and the right arm raised/flexed. This seizure type is concisely described as a left asymmetric tonic seizure (since the left arm is

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Table 1.5 Typical lateralization and localizations of seizure types and postictal symptoms

Common patient descriptors	Seizure Semiology Classification [5]	Localization [4]
Déjà vu, panic, anxiety, hallucinations, fear, unease	Psychic aura	Temporal lobe; can consider parietal or frontal lobe
Foul smell like rotten eggs, sulfur	Olfactory aura	Temporal lobe; can consider orbitofrontal lobe
Sometimes foul or metallic taste	Gustatory aura	Temporal lobe
Rising or flipping sensation of the stomach; mild nausea	Abdominal aura	Temporal lobe
Flushing, intense nausea, choking sensation, palpitations, hair standing on end	Autonomic aura	Insula
Tingling, prickling; less commonly numbness, far less commonly painful	Somatosensory aura	Contralateral parietal lobe; can consider insula, particularly if painful
Tones, clicks, basic sounds; less commonly complex sounds like music	Auditory aura	Temporal lobe
Flashing or swirling lights	Visual aura	Contralateral occipital lobe
Unresponsiveness, incorrect answers to questions, space out, blank out, repetitive, or simple responses (yeah, no, etc.)	Dialeptic seizure	Not particularly localizing; should consider absence epilepsy in a child
Lip smacking, "acting weird," repetitive hand/finger movements, drooling, repetitive swallowing	Automotor seizure	Temporal lobe is most likely, although this seizure type has been seen in all localizations

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Table 1.5 (cont.)

Common patient descriptors	Seizure Semiology Classification [5]	Localization [4]
Flailing, running, kicking, boxing, punching, screaming, "crazy" movements	Hypermotor seizure	Frontal lobe, although can be from alternate localizations; if dystonia is present, it will localize contralateral to the dystonia
Laughing, giggling, "creepy" laugh	Gelastic seizure	Hypothalamic hamartoma; frontal lobe if mechanical laugh; temporal lobe if "emotional"
Stiffen	Tonic seizure	Contralateral motor cortex or supplemental motor area
Shake	Clonic seizure	Contralateral motor cortex
Head turn, eye turn	Versive seizure	Frontal lobe (frontal eye fields)
Muscle jerk	Myoclonic seizure	Generalized
Stiffen and shake all over, begin with a loud yell (ictal cry)	Tonic clonic seizure	If primary seizure type, generalized epilepsy Otherwise, does not localize
Fall, "just drop," head drop	Atonic seizure	Generalized
Arms stiffen and then hunching over, most commonly in the truncal areas	Epileptic spasm	Can be generalized or focal

Symptoms and 1998 seizure name should include laterality modifier when appropriate with options including left, right, axial, or generalized.

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extended), which often localizes to the right frontal lobe, but at the least lateralizes to the right hemisphere (Tables 1.3 and 1.5).

4. A patient is observed to have bilateral stiffening of their arms and legs followed by rhythmic shaking of the arms and legs. This would be classified as a GTC seizure or bilateral tonic clonic seizure. It would not have localizing or lateralizing value (Tables 1.3 and 1.5).

ADDITIONAL HISTORY VITAL IN THE CONTEXT OF A POTENTIAL FIRST SEIZURE

While the semiology history is the bulk of seizure-specific history, there are multiple risk factors that you should assess. In essence, these risk factors revolve around ascertaining if your patient has had any brain trauma, whether acquired or developmental. Straightforward questions you can ask the patient include [10]:

1. Was there any difficulty with your birth?
2. Did you develop normally as a child?
3. Have you had any brain or spine infections?
4. Have you had a head injury with loss of consciousness (LOC) and, if so, how long were you unconscious?
5. Is there anyone in your family with epilepsy or seizures?

Infections can predispose to seizures and epilepsy. Early seizures can be seen in 22% of viral encephalitis cases and 10% of patients present with seizures later. Likewise, early seizures can be seen in 13% of patients with bacterial meningitis and 2.4% of patients on a more chronic basis [11].

Head injury can be classified as mild, moderate, or severe. These are determined by length of amnesia or LOC. Mild is <30 minutes, moderate is 0.5–24 hours, and severe is >24 hours. Rate of epilepsy after mild head injury was 1.5 and not statistically significant compared to 2.9 and 17 times more likely for moderate and severe, respectively [12]. It is worth remembering that people with nonepileptic events more commonly had mild head injury [13].

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There is a hereditary nature to some epilepsy. Generalized epilepsy can be passed from either the mother or father. For focal epilepsy, the risk is limited more specifically to the mother [14]. Still, in general, most people with epilepsy do not inherit it.

Besides risk factors intrinsic to the patient, you should assess external causes of a seizure, otherwise known as a provoked seizure. Some medications (bupropion, tramadol, cefepime, benzodiazepine rapid/acute withdrawal) are well known to cause isolated seizures [15, 16].

Is It Really a Seizure? The Differential Diagnosis of a First-Time Seizure

Patients who present for evaluation of a possible first seizure most typically have alteration of consciousness. Alternate symptoms can include specific transient neurological dysfunctions such as numbness, visual disturbance, weakness, or difficulty speaking. When discussing the differential of a first seizure, it bears recalling typical features of seizures as discussed earlier. This will help to compare and contrast with alternate diagnostic possibilities.

DIFFERENTIAL #1: NONEPILEPTIC EVENTS

Nonepileptic events are covered extensively in Chapter 10 of this manual. In brief, these are paroxysmal episodes with some resemblance to epilepsy, but with some key differences. Patients with nonepileptic events are more likely [10, 17]:

- To have asymmetric movements
- To have a start/stop quality to the events themselves

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- To have events that last longer than seizures (often >5 minutes compared to the <2 minutes for seizures)
- To have their eyes closed during the episodes
- Suggestible to the event starting and stopping
- To have awareness during whole body movements.

DIFFERENTIAL #2: SYNCOPE

Syncope is an acute LOC or near LOC. The key difference is that syncope is due to globally decreased cerebral blood flow whereas a seizure is due to abnormal coordinated brain electrical activity [10].

A common confounder is when people have convulsive movements during a seizure. This is by no means uncommon as myoclonic movements are noted in 60% of patients in one well-documented cohort [18]. Nearly all patients had their eyes open with syncope [18] in similarity to epilepsy but in contrast to nonepileptic events.

From a historical perspective, syncope patients expectedly have autonomic symptoms such as pallor, change in heart rhythm, or sweating [18, 19]. As discussed previously, autonomic symptoms can be present in epilepsy, but are less common compared to syncope in general.

DIFFERENTIAL #3: TRANSIENT ISCHEMIC ATTACK

A focal acute loss of cerebral perfusion can cause a transient ischemic attack (TIA). Crucially, the initial symptom is more likely to be a *loss of function* due to hypoactivity of the brain compared to the relative "gain of function" due to electrical hyperactivation of the brain region during a seizure. One well-documented exception is the limb-shaking TIA. This is seen as episodic limb-shaking episodes contralateral to the occluded carotid artery [20]. Prompt consideration of limb-shaking TIAs in the work up of "EEG negative" focal seizures is indicated.

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DIFFERENTIAL #4: MIGRAINE WITH AURA OR BASILAR MIGRAINE

Migraine with aura can have a variety of neurological symptoms including most commonly visual aura as well as less commonly numbness, speech difficulty, weakness, and vertigo. Compared to epilepsy, migraine symptoms are more prolonged and there is of course the subsequent headaches in the majority of migraine patients [10].

DIFFERENTIAL #5: METABOLIC DERANGEMENTS

Alteration of consciousness, encephalopathy, and provoked seizures are commonly seen in periods of either hepatic or uremic encephalopathy [15]. Acute hypoglycemia with blood sugars below 20 can cause GTC seizures and is a particularly relevant consideration in patients with diabetes.

Do I Need to Order Labs, EEG, or Imaging?

While the previously mentioned semiology and epilepsy-specific history is the most accessible way to make a specific diagnosis in a first seizure patient, additional testing is indicated.

LAB WORK

Lab work in general is also covered in Chapter 6. In brief here, routine lab work such as complete metabolic profile (CMP) or complete blood count (CBC) should be obtained to assess for any infection or metabolic derangement. Urinalysis with or without urine toxicology may also

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be obtained as clinically appropriate. Many first-time seizure patients are otherwise healthy so the routine lab work may be more useful if considering ASM initiation [17].

An ammonia level >80 μmol/L drawn ≤60 minutes after the seizure can correctly classify 80% of episodes between a generalized seizure and focal seizure or nonepileptic event [21]. Given the specific timing requirements, ammonia levels would be of limited utility and not recommended unless drawn within 1 hour of the seizure.

Prolactin is a desired biomarker for seizures, but is only useful if there is a baseline prolactin level >6 hours prior to the episode. In that rare scenario, an elevated prolactin level can distinguish between a generalized and focal seizure, but crucially not between a seizure and syncope or a seizure and a nonepileptic event [10]. As these are the two more common clinical questions being assessed as well as the rare presence of a prolactin level >6 hours prior, we do not recommend prolactin measurement.

ELECTROENCEPHALOGRAM

Patients with a first seizure should have EEG performed. Data do support an increased epileptiform yield if done within 24 hours of the first seizure [1]. When strongly suspecting an epilepsy diagnosis, data also indicate that a >1 hour study will increase the likelihood of capturing an epileptiform finding. Moreover, the same study increased capture of events (epileptic or nonepileptic) after 30 minutes of recording [22]. The utilization of EEG in epilepsy care is covered expansively in Chapter 4.

IMAGING

The utility of imaging in a first seizure is twofold: rule out a neurological emergency and potentially identify the cause of the patient's seizure. To rule

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out a neurological emergency such as stroke or intracranial hemorrhage, computed tomography (CT) of the head is adequate. To identify more subtle causes of epilepsy like focal cortical dysplasia, hippocampal sclerosis, and so on, a 3 tesla (3T) magnetic resonance imaging (MRI) with an epilepsy protocol has shown to be superior [23, 24]. Thus, if considering MRI, it is worth verifying your facility performs 3T with an epilepsy protocol to enhance the diagnosis rate of the MRI study. Imaging in new onset and chronic epilepsy is covered extensively in Chapter 5.

Do I Need to Start an ASM?

The decision to start an ASM can be more accurately stated "Do I think the patient has epilepsy?" If you do not think that the patient has epilepsy, an ASM will have no clinical benefit. For instance, if a patient has had recurrent seizures due to alcohol withdrawal, the best treatment would be to address the alcoholism. Epilepsy is defined as one of the three following conditions [25]:

1. At least two unprovoked seizures >24 hours apart
2. One unprovoked seizure with a >60% chance of seizure recurrence within 10 years
3. A recognized epilepsy syndrome.

For condition #1, it is critical to consider all of the patient's seizure types. For instance, the patient has had multiple episodes of déjà vu over the past months and comes to you after a first tonic clonic seizure; that patient has had more than two seizures. The epilepsy definition does not require multiple tonic clonic seizures.

Condition #2 arose to better manage patients in clinical situations such as a first seizure and neuroimaging demonstrating a brain tumor concordant with semiology history. Logically, one should not withhold ASM treatment for the patient to have a second seizure just to fulfill the criteria for condition #1.

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When you have decided that the patient does have epilepsy, you should start an ASM. While the topic of which ASM and what dose to use will be covered extensively in Chapter 3, data indicate that lamotrigine, zonisamide, and carbamazepine are reasonable options for focal epilepsy whereas valproate is the most effective choice for generalized epilepsy [26, 27]. However, valproate's teratogenic risk and side effect profile certainly influences the selection of valproate as initial therapy [28, 29].

In general, patients who are seizure free tend to be so at lower doses, so you do not need to target the highest dose to achieve a seizure-free outcome [30].

How Do I Counsel Patients after a First Seizure?

This brings us back to the initial questions that patients will ask you in consultation of a first seizure.

WHY DID I HAVE A SEIZURE?

Ideally a combination of history, lab work, and imaging can help provide this answer. You first should establish that the patient really did have a seizure and then provide an etiology if it is known at that time. If you do not know an etiology, it is perfectly acceptable to tell the patient that the cause of the seizure is not determined at this point.

WILL I HAVE MORE SEIZURES?

Again, you should be able to clearly tell the patient if they have epilepsy. If you do think that they have epilepsy, you are likely starting a seizure

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medicine and you can tell them that around 50% of people with epilepsy are seizure free with their first ASM [31]. For patients with a clear first-time seizure, a metaanalysis of five prospective studies has indicated that the risk is 40% for seizure recurrence [32] – coincidentally why most patients with an isolated first seizure are not started on an ASM.

WHAT KIND OF TESTING DO I NEED TO UNDERGO?

You can counsel patients that they should have a thorough history, an EEG (ideally within 24 hours if feasible), and a 3T MRI with an epilepsy protocol.

DO I NEED TO TAKE ASMs, AND IF SO, FOR HOW LONG?

This question is essentially asking: "Will I have more seizures, or do I have epilepsy?" If you think that they have epilepsy, your patient should take ASMs. The length of ASM treatment depends on your certainty that they have epilepsy and the specific diagnosis that you have made. For instance, childhood absence epilepsy is a common diagnosis in children that most commonly resolves, so you could counsel the patient that they will likely be able to come off medications. Alternately, a patient who has had a stroke and a year later has a first seizure consistent with the area of brain injury, you would counsel that it is less likely that they will come off medications due to the static nature of the injury.

HOW WILL THIS AFFECT MY LIFE?

Answering this question requires sensitivity coupled with straightforwardness, no doubt a delicate balance.

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The first specific thing that patients often want to know about is driving. Laws regarding driving after a seizure differ between countries and states. Commonly, patients may resume driving after being 6 months seizure free, although this length may be longer or shorter depending on location. You should ascertain your local laws. You can discuss that seizure freedom at 6 months, 12 months, and 18 months are associated with a relapse rate of 44%, 32%, and 17%, respectively [33]. This can help some patients understand the local laws who are understandably frustrated about the revocation of their driving privileges.

A straightforward way to discuss precautions taken at the time of first seizure are to note that they are made to prevent injury. Thus, in addition to driving, patients are not recommended to take a bath or swim alone (risk of drowning), use power tools (risk of bodily injury), or cook with open flames (risk of fire and burns) until they are 6 months seizure free. Scuba diving and sky diving are commonly restricted completely in the event of an epilepsy diagnosis [34].

Life events that can be encouraged include having a family [35]. In the United States, epilepsy is a protected disability by the American with Disabilities Act and workplace accommodations should be made for these patients [36].

Works Cited

1. MA King, MR Newton, GD Jackson et al. Epileptology of the first-seizure presentation: A clinical, electroencephalographic, and magnetic resonance imaging study of 300 consecutive patients. *Lancet*. 1998;352(9133):1007–11.
2. F Rosenow and H Lüders. Presurgical evaluation of epilepsy. *Brain*. 2001;124(Pt. 9):1683–700.
3. WA Hauser, VE Anderson, RB Loewenson, and SM McRoberts. Seizure recurrence after a first unprovoked seizure. *N Engl J Med*. 1982;307(9):522–8.
4. N Foldvary-Schaefer and K Unnwongse. Localizing and lateralizing features of auras and seizures. *Epilepsy Behav*. 2011;20(2):160–6.
5. H Luders, J Acharya, C Baumgartner et al. Semiological seizure classification. *Epilepsia*. 1998;39(9):1006–13.

https://doi.org/10.1017/9781009264976.001 Published online by Cambridge University Press

6. RS Fisher, JH Cross, JA French et al. Operational classification of seizure types by the International League Against Epilepsy: Position paper of the ILAE Commission for Classification and Terminology. *Epilepsia*. 2017;58(4):522–30.
7. RS Fisher, JH Cross, C D'Souza et al. Instruction manual for the ILAE 2017 operational classification of seizure types. *Epilepsia*. 2017;58(4):531–42.
8. H Lüders, N Akamatsu, S Amina et al. Critique of the 2017 epileptic seizure and epilepsy classifications. *Epilepsia*. 2019;60(6):1032–9.
9. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. From the Commission on Classification and Terminology of the International League Against Epilepsy. *Epilepsia*. 1981;22(4):489–501.
10. JR Gavvala and SU Schuele. New-onset seizure in adults and adolescents: A review. *JAMA*. 2016;316(24):2657–68.
11. JF Annegers, WA Hauser, E Beghi, A Nicolosi, and LT Kurland. The risk of unprovoked seizures after encephalitis and meningitis. *Neurology*. 1988;38(9):1407–10.
12. JF Annegers, WA Hauser, SP Coan, and WA Rocca. A population-based study of seizures after traumatic brain injuries. *N Engl J Med*. 1998;338(1):20–4.
13. E Barry, A Krumholz, GK Bergey et al. Nonepileptic posttraumatic seizures. *Epilepsia*. 1998;39(4):427–31.
14. AL Peljto, C Barker-Cummings, VM Vasoli et al. Familial risk of epilepsy: A population-based study. *Brain*. 2014;137(Pt. 3):795–805.
15. P Beleza. Acute symptomatic seizures: A clinically oriented review. *Neurologist*. 2012;18(3):109–19.
16. R Sutter, S Rüegg, and S Tschudin-Sutter. Seizures as adverse events of antibiotic drugs: A systematic review. *Neurology*. 2015;85(15):1332–41.
17. JA French and TA Pedley. Clinical practice: Initial management of epilepsy. *N Engl J Med*. 2008;359(2):166–76.
18. JG van Dijk, RD Thijs, E van Zwet et al. The semiology of tilt-induced reflex syncope in relation to electroencephalographic changes. *Brain*. 2014;137(Pt. 2):576–85.
19. A McKeon, C Vaughan, and N Delanty. Seizure versus syncope. *Lancet Neurol*. 2006;5(2):171–80.
20. T Yanagihara, DG Piepgras, and DW Klass. Repetitive involuntary movement associated with episodic cerebral ischemia. *Ann Neurol*. 1985;18(2):244–50.
21. R Albadareen, G Gronseth, P Landazuri et al. Postictal ammonia as a biomarker for electrographic convulsive seizures: A prospective study. *Epilepsia*. 2016;57(8):1221–7.
22. DB Burkholder, JW Britton, V Rajasekaran et al. Routine vs extended outpatient EEG for the detection of interictal epileptiform discharges. *Neurology*. 2016;86(16):1524–30.

https://doi.org/10.1017/9781009264976.001 Published online by Cambridge University Press

23. S Knake, C Triantafyllou, LL Wald et al. 3T phased array MRI improves the presurgical evaluation in focal epilepsies: A prospective study. *Neurology*. 2005;65(7):1026–31.
24. J von Oertzen, H Urbach, S Jungbluth et al. Standard magnetic resonance imaging is inadequate for patients with refractory focal epilepsy. *J Neurol Neurosurg Psychiatry*. 2002;73(6):643–7.
25. RS Fisher, C Acevedo, A Arzimanoglou et al. ILAE official report: A practical clinical definition of epilepsy. *Epilepsia*. 2014;55(4):475–82.
26. A Marson, G Burnside, R Appleton et al. 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*. 2021;397(10282):1363–74.
27. A Marson, G Burnside, R Appleton et al. The SANAD II study of the effectiveness and cost-effectiveness of valproate versus levetiracetam for newly diagnosed generalised and unclassifiable epilepsy: An open-label, non-inferiority, multicentre, phase 4, randomised controlled trial. *Lancet*. 2021;397(10282):1375–86.
28. E Perucca and T Tomson. The pharmacological treatment of epilepsy in adults. *Lancet Neurol*. 2011;10(5):446–56.
29. T Tomson, D Battino, E Bonizzoni et al. Comparative risk of major congenital malformations with eight different antiepileptic drugs: A prospective cohort study of the EURAP registry. *Lancet Neurol*. 2018;17(6):530–8.
30. P Kwan and MJ Brodie. Effectiveness of first antiepileptic drug. *Epilepsia*. 2001;42(10):1255–60.
31. Z Chen, MJ Brodie, D Liew, and P Kwan. Treatment outcomes in patients with newly diagnosed epilepsy treated with established and new antiepileptic drugs: A 30-year longitudinal cohort study. *JAMA Neurol*. 2018;75(3):279–86.
32. AT Berg and S Shinnar. The risk of seizure recurrence following a first unprovoked seizure: A quantitative review. *Neurology*. 1991;41(7):965–72.
33. YM Hart, JW Sander, AL Johnson, and SD Shorvon. National General Practice Study of Epilepsy: Recurrence after a first seizure. *Lancet*. 1990;336(8726):1271–4.
34. NB Fountain and AC May. Epilepsy and athletics. *Clin Sports Med*. 2003;22(3):605–16, x–xi.
35. PB Pennell, JA French, CL Harden et al. Fertility and birth outcomes in women with epilepsy seeking pregnancy. *JAMA Neurol*. 2018;75(8):962–9.
36. A Krumholz, JL Hopp, and AM Sanchez. Counseling epilepsy patients on driving and employment. *Neurol Clin*. 2016;34(2):427–42, ix.

https://doi.org/10.1017/9781009264976.001 Published online by Cambridge University Press