

# **EPISODIC DISORDERS**

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### Case 1

A slim 13-year-old boy is attending his grade school commencement, which is being held outdoors on a hot day. Near the end of the ceremony he feels nauseated, his vision fades, goes gray and he falls limply to the ground unconscious. His friends try to sit him up and his body shakes for several seconds. He awakens several seconds later and is seen to be pale and sweaty.

### Case 2

A 25-year-old law student is studying in the library. Suddenly her left hand begins to twitch. This is followed several seconds later by twitching of the left side of her face. She loses consciousness and falls stiffly to the floor, where her body and limbs shake rhythmically for one minute. She is incontinent, and confused for 15 minutes after she awakens.

### Case 3

A 65-year-old diabetic, hypertensive man is working in his garden when he experiences the sudden onset of weakness of the right arm and leg. He slumps to the ground. When his wife runs over to him he appears to be awake, but does not speak. The symptoms clear completely over the next 30 minutes.

The above are simple paradigms of episodic neurological disorders: **vaso-vagal syncope, seizure, and transient ischemic attack (TIA).**

In these simple cases, the combination of age, risk factors, circumstance and attack characteristics leaves little room for doubt.

Not all episodic neurological symptoms and signs suggest the diagnosis so easily, however. Two of the episodes above are characterized by temporary loss of consciousness and by convulsive movements, and two by acute, focal neurological symptoms and signs.

In syncope, seizures, and TIAs there may be overlapping aspects of history, presentation, symptomatology, but these disorders involve widely different pathophysiologies. They should rarely be confused. How are they reliably to be distinguished?

### Teaching points, or you need to know that:

1. Convulsive movements commonly accompany syncope.
2. The premonitory symptoms of syncope are usually diagnostic.
3. The alteration of neurological function such as that seen in Case 3 should suggest pathology involving a specific brain region, nucleus or tract, often correlating with an arterial perfusion area, and hence may straightaway be suggestive of the cause.
4. The symptoms at the start of many seizures are characteristic of the cerebral area of onset.
5. The 'postictal' state of a person recovering from (coming to after) a syncope differs markedly from that of a person recovering from a 'grand mal' seizure.

What are the features of the cases above which make the diagnosis obvious; and what are the common variants of these disorders, which may make their diagnosis more difficult, albeit ultimately achievable?

Let us start with the principle that the sudden onset of neurological symptoms (regional loss of function, focal symptoms, loss of consciousness) is generally due to an electrical (seizure) or vascular (TIA, stroke, syncope) event.

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### **Case 1: Syncope**

*Taking the first example, what features of the story lend credibility to the presumptive diagnosis of a syncopal event?*

Syncope or loss of consciousness is the direct result of a transiently inadequate supply of blood to the brain. Such reduction of cerebral perfusion may occur because of lowered cardiac output due to arrhythmias, and in such cases may be referred to as a Stokes-Adams attack. By far the most common form of syncope, however, is that associated with abnormal autonomic regulation of blood pressure and/or heart rate, and it is then called vaso-vagal syncope.

The diagnosis of syncope illustrates the paramount role of a careful history in patients with neurological disorders. Vaso-vagal syncope is almost always easily diagnosed if a careful history is taken from the patient presenting with an episode of loss of consciousness.

What features of the history may distinguish between the trivial, often single syncope and more serious disorder?

## **The Typical History**

### **Features of Epidemiology**

**Age.** Vaso-vagal syncope, the most common type of faint encountered in office practice, occurs most commonly in children, adolescents, and young adults.

**Triggers (provocative factors).** It is usually provoked by circumstances, which acutely lower blood pressure, the return of blood to the heart (pre-load), and hence reduce cardiac output.

Syncope may be triggered by a Valsalva maneuver such as that occurring with **micturition** or violent **coughing** ('cough syncope' in persons with chronic obstructive pulmonary disease), or **pooling of blood** in the veins of the legs in association with cutaneous vasodilatation as in troops standing on a hot parade ground.

Sudden rushes of sympathetic nerve activity such as those associated with a frightening experience (eg, **pain** or the sight of **blood**) may trigger a vagal reflex with bradycardia and/or lowered blood pressure. The doctor or dentist's office is a favorite site for such syncopes, and should always raise the possibility of that diagnosis.

Fainting shortly after arising from a sitting or lying position, especially in patients taking antihypertensive drugs, should always suggest **orthostatic hypotension**.

**Dehydration** may be a contributing factor, lowering the "safety factor" for adequate cerebral perfusion.

Severe **hemorrhage** as in menorrhagia may also lead to syncope.

**Circumstances.** The circumstances of the attack are often suggestive: The young man with micturition syncope may be dehydrated after an evening of heavy drinking; he gets up in the night to urinate due to alcoholic diuresis, stands at the toilet, and performs a Valsalva maneuver as he initiates urination. He typically awakens on the bathroom floor.

Another common scenario involves the adolescent who stands for an hour in the hot sun, blood pooling in his legs; he is wearing heavy graduation robes, sweating ineffectually, flushed with the heat. His heart rate increases, ventricular volume dropping until there is a sudden reflex vagal discharge with nausea, hypotension, and pallor. As cerebral perfusion falls, vision fades or is lost and unless he lies down or puts his head down he loses consciousness.

Orthostatic faints are particularly common upon arising first thing in the morning or during nocturnal trips to the bathroom, when blood pressure reflexes may be relatively inefficient. There may be a brief prodrome of faintness or dizziness, or loss of consciousness may be abrupt.

**Premonitory Symptoms.** Vaso-vagal syncope rarely happens without some premonitory symptoms: fading of vision, nausea, sweating, pallor, weakness, or a feeling of an impending faint are the most common. Such symptoms may last several seconds to a half-minute or so before consciousness is lost. Vomiting may occur on awakening.

The person with syncope usually slumps limply to the ground. Twitching or myoclonic movements of the limbs may then occur, and may suggest to on-lookers that a seizure is occurring. Unlike the clonic movements of a seizure, however, which lasts 30-60 seconds, in so-called “convulsive” syncope the jerks last only a few seconds.

## **Cardiac Syncope**

### **Age and Morbidity**

Cardiac syncope usually occurs in older patients with a history of other forms of heart disease such as coronary artery disease or angina. Here, a faint or syncope is generally due to a sudden arrhythmia, so that there may be little or no warning. The person simply awakens on the ground after few seconds. (Presumably those unfortunate enough to have a more prolonged reduction in cerebral perfusion do not survive.)

Cardiac syncope may be due to potentially fatal arrhythmias such as ventricular tachycardia, so that accurate diagnosis is vital.

### **Other Risk Factors/Circumstances**

Orthostatic syncope is also typical of faints due to loss of normal blood pressure regulation, as found in diabetic and other peripheral neuropathies.

### Teaching Points:

1. Loss of consciousness and falling characterize syncope.
2. Brief convulsive movements due to cerebral hypoxia may occur in 70-90% of syncopal episodes.
3. Incontinence occasionally may occur.
4. Patients usually are fully oriented to their surroundings on awakening.

*How may the convulsive movements associated so commonly with syncope be distinguished from those associated with epilepsy?*

1. Epileptic seizures occur in any circumstances, while syncope is due to common precipitants - see history above.
2. Postictal confusion most adequately distinguishes epilepsy from syncope.
3. In syncope, muscle movements are usually of small amplitude, and may be repeated only a few times over several seconds.
4. Muscle movements during a tonic-clonic (grand mal) seizure are more violent and last 30 to 60 seconds. Syncopal myoclonus is probably due to brainstem ischemia and has been misleadingly called "convulsive syncope" or "syncopal convulsion." It is a very frequent cause of misdiagnosis. A careful history, however, will reveal the typical circumstances or prodromal symptoms of vaso-vagal syncope and prevent this serious diagnostic mistake.

### Teaching Points:

1. The convulsive movements of epilepsy last longer and are more violent than those seen in syncope.
2. The prodromal symptoms of vaso-vagal syncope (nausea, faintness, weakness, diaphoresis, fading of vision) are highly suggestive, and usually well recalled by the patient. Take time to elicit the history of how the patient felt before losing consciousness.
3. In all three conditions with which you are dealing, the account of an observer (or observers) should be sought. They may describe aspects of the attack unknown to the unconscious patient.

*What should you expect to find on examination?*

**The physical examination.** The physical examination in vaso-vagal syncope rarely reveals an abnormality. Routine blood pressure measurements are usually normal.

If orthostatic syncope is suspected it is crucial to measure the blood pressure after keeping the patient supine for 10 minutes. The pressure is retaken immediately after having the patient stand up, and again three minutes later. A systolic drop of 15 mm, or a diastolic drop of more than 10 mm is abnormal; even a smaller change may be significant if it reproduces the patient's prodromal symptoms.

The patient with cardiogenic syncope may have physical signs of heart disease such as cardiomegaly, irregular heart rate, or murmur. Massaging the carotid sinus, the physiologic blood pressure detector in the neck, to look for the rare condition of carotid sinus sensitivity is a

controversial maneuver, which has been blamed for strokes and life-threatening changes in blood pressure or heart rhythm. The procedure is best avoided in the office.

*What diagnostic tests may be useful?*

**Diagnostic tests.** No diagnostic tests are necessary in a case of vaso-vagal syncope. If cardiac syncope is suspected, a standard electrocardiogram and rhythm strip can often be immediately performed in the office. If not, a 12-lead EKG (\$65 to \$75) followed by a 24-hour Holter monitor (\$300 to \$600) is indicated. An (M-mode 2D) echocardiogram (\$600) will look for valvular disease such as aortic stenosis. If all of these tests are normal, it is unlikely that further tests will be required because the diagnosis of cardiogenic syncope is extremely unlikely. Recurrent episodes should provoke another careful history and a repeat Holter.

Tilt-table testing (\$3500 to \$4000) does not usually contribute to the diagnosis of vaso-vagal syncope, and is indicated only in cases when disease of the autonomic nervous system causing orthostatic hypotension is suspected. Such conditions may be accompanied by other autonomic symptoms or signs, and if they are suspected neurological consultation would be appropriate.

### **Teaching Point**

In the face of a typical history of vaso-vagal syncope, even one associated with a few myoclonic jerks, an EEG is unnecessary and may even be counter-productive, since the number of falsely positive "epileptic" EEG abnormalities emerging from some laboratories is distressingly high.

As in all of these episodic disorders, the history is the paramount diagnostic instrument. If the diagnosis is unclear after a careful history is taken from the patient and a witness of the attack(s), the answer very often eludes other simple diagnostic procedures.

*What treatment strategies are available?*

**Treatment** of syncope depends upon the cause.

Cardiac disorders are treated in the usual fashion. Adjustment or discontinuation of drugs causing orthostatic hypotension may be necessary.

Treatment of orthostatic hypotension due to peripheral or autonomic neuropathies centers around measures designed to increase water retention. Nocturnal elevation of the head of the bed is the simplest initial step. If this fails, fludrocortisone may be tried.

Vaso-vagal syncope usually requires no treatment other than explanation to the patient. Understanding the benign nature of the attacks, being aware of the kind of circumstances which put him or her at risk, and being prepared to lie down or put the head down at the first sign of a syncopal prodrome is generally enough.

*When may a specialist be useful?*

When a diagnosis of cardiac syncope is probable (eg, signs of severe aortic stenosis, serious cardiac arrhythmias), a cardiologist should be consulted. If cardiac syncope is strongly suspected clinically but unproven, the cardiologist may recommend specialized tests of cardiac conduction.

When the clinical history is ambiguous, or when an imprudently ordered EEG shows unexpected "epilepsy" in the patient with classical symptoms of vaso-vagal syncope, the neurologist may help reinforce the correct diagnosis and avoid years of inappropriate antiepileptic treatment. Most neurologists may also provide a treatment plan for severe orthostatic hypotensive syncope due to neuropathies.

### **To Sum Up**

1. *The excessive work-up of simple syncope usually means that the episode has been misinterpreted. It is wasteful of resources and frequently confusing (eg, the false-'positive' EEG or tilt-table test). Vaso-vagal syncope should almost always be diagnosable by the primary care physician.*
  2. *Serious causes of syncope require thorough investigation, and sometimes consultation.*
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### **Case 2. Seizures and Epilepsy**

*Approaching the second case, what are the features that lend credibility to a presumptive diagnosis of epilepsy?*

Seizures occurring in the context of an acute illness such as meningitis, eclampsia, alcohol withdrawal, or high fever (in infants) do not constitute epilepsy, and generally are not an indication for chronic treatment.

In contrast, **epilepsy** consists of recurrent seizures appearing at unpredictable intervals. It is most logically regarded as a symptom rather than as a disease *per se*. Just as the acute seizure raises the immediate question, "What is the cause?" similarly chronic epilepsy should prompt the question, "What is causing it?"

Such epileptogenic disorders include genetic epilepsies, fixed or progressive structural brain lesions, and metabolic or degenerative neurological syndromes of which epilepsy is only one manifestation. In many cases no obvious cause of the epilepsy can be found (idiopathic).

*What do you understand to be going on?*

Seizures reflect paroxysmal, abnormal neuronal activity, occurring in the cerebral cortex. Thalamic, basal ganglia, and other gray matter structures may participate in the ictal paroxysms, but the cortex is crucial to seizure generation. Lack of normal neuronal inhibition by gamma-amino butyric acid (GABA), excessive glutamate (excitatory) activity, and abnormal calcium channel regulation have all been proposed as possible pathophysiologic mechanisms.

## Epidemiology

### Age and Incidence

Acute, symptomatic seizures are very common, with a lifetime incidence of about 10%. Chronic epilepsy has a prevalence of about 0.5% across all age groups. The incidence of epilepsy is highest in the first year of life, falls slowly until a plateau is reached beginning about 10 years of age, and rises again in the elderly.

Risk factors include intracranial lesions (tumor, hemorrhage), a family history of epilepsy, a history of acute symptomatic seizures, or a history of meningitis or encephalitis. Minor head trauma does not increase the risk of epilepsy, but a history of loss of consciousness for more than 30 minutes, focal neurological signs, skull fracture, or seizures at the time of the trauma do so. Abuse of cocaine, amphetamine, or PCP, and therapeutic doses of some antidepressants, particularly newer serotonin-sparing drugs, can provoke seizures. "Birth injury" is probably over-rated as a cause of epilepsy. In at least half the patients with adult-onset epilepsy, no cause can be found.

### Circumstances and Premonitory Symptoms

Seizures can occur at any time, in any circumstances. The "day after" is a common time for alcohol-provoked seizures. Patients with known epilepsy often report an increase in seizures near the time of menses, during acute viral infections, or with stress.

Patients with focal seizures may report a stereotyped feeling (the aura) or focal motor movement at the beginning of an attack.

### Seizure Types

The most common seizures are focal or partial, that is, they begin (as in **Case 2**) in a localized cortical area. The clinical appearance of the seizure depends upon the function of the cortical area involved, eg, motor cortex gives rise to contralateral twitching, visual cortex produces flashing lights, limbic cortex causes the experience of nausea, fear, or memories. The subjective "warning" of a seizure or aura reported by some patients actually represents the start of the attack, and is an indication of its focal origin.

*What does the focality tell you?*

Seizures which stop after such experiences are called simple partial seizures, while attacks which proceed to altered consciousness or start with it are called **complex partial** or **psychomotor** seizures. Complex partial seizures are the most common type of focal seizure. Patients usually remain seated or standing, stare or look around, and often smack their lips or fiddle with their buttons or rub their bodies. These movements are called **automatisms** and are the same in each seizure, in each patient.

If the focal seizure activity spreads throughout the brain, the result is a **tonic-clonic seizure**, also known as a **grand mal** or **generalized** seizure. The tonic-clonic seizure starts with stiffening of the body and limbs ("tonic"), followed within half a minute by bilaterally synchronous jerking ("clonic") of the limbs and face. Respirations are halted or ineffective, and cyanosis is common. The clonus usually slows, then stops abruptly. This part of the episode

usually lasts one to two minutes. Reactive hyperventilation and excessive salivation (the postictal state) are often seen at this point ("frothing at the mouth") resolving over the next 10-30 minutes.

Consciousness is lost during a tonic-clonic seizure, with amnesia for the tonic-clonic movements, and disorientation and sleepiness during recovery.

**Nonfocal seizures.** **Absence** or **petit mal seizures** consist of abrupt immobility and loss of awareness (in effect, loss of continuity of consciousness, usually lasting no more than 15 seconds, with immediate recovery of mental functions after the attack. Patients themselves may be unaware of these brief seizures. Absence attacks occur mainly in children.

Less common brief, nonfocal seizure types are myoclonic seizures, which consist of sudden, bilateral jerks of the arms, and atonic seizures, in which the patient falls to the ground without warning or recollection. Like absences, they last only a few seconds, and have no warning. Atonic seizures are characteristic of children (and less commonly adults) with psychomotor retardation or other clinical signs of serious, fixed cerebral pathology.

*What are we dealing with?*

### **Common syndromes**

Febrile seizures are common, age-specific, symptomatic seizures with a high recurrence rate in the individual and the family. Seen mainly between 18 months and 2 years, they are usually brief, tonic-clonic seizures triggered by rapid, high temperature rises such as those of roseola infantum. Chronic antiepileptic drugs are generally not prescribed after the first febrile seizure. The parents should be instructed to use cool baths at the first sign of fever. Rectal diazepam, for home use, is sometimes prescribed after recurrent febrile seizures.

Simple febrile seizures do not occur after the age of 5 years.

**Infantile spasms** or salaam attacks are brief motor seizures consisting of sudden flexion or extension of the neck, trunk, or limbs. This is an age-related seizure type, usually limited to the ages of 6 months to 2 years. Infantile spasms usually occur many times daily. Although they may occur in healthy infants, many children with infantile spasms have pre-existing cerebral damage, and most of them show developmental delay with or without chronic epilepsy as they mature. Neurological consultation is generally indicated.

**Benign Rolandic epilepsy** is a common epileptic syndrome beginning between 5 and 10 years of age. Typically, a healthy child has infrequent nocturnal tonic-clonic seizures, or seizures beginning with facial twitching. A family history of similar attacks in a sibling is frequent, as is one of febrile seizures. A correct diagnosis is important, both for appropriate treatment (see below) and because the prognosis of complete remission by the end of puberty may be given to the parents with confidence.

**Juvenile myoclonic epilepsy** is one of the most common seizure disorders, also frequently seen in more than one family member. Beginning in childhood with bilateral myoclonic jerks of the arms, it usually comes to the attention of the physician when a tonic-clonic seizure occurs in the early to late teens. Most attacks happen shortly after arising in the morning, often provoked by lack of sleep or alcohol ingestion. Although usually easily controlled by treatment (see below), juvenile myoclonic epilepsy is a lifelong condition.

**Childhood absence epilepsy** is a benign genetic disorder, which usually begins between 5 and 10 years of age with petit mal seizures. Tonic-clonic seizures may occur several years later. The great majority of cases go into permanent remission by young adulthood, when medication may be stopped.

**Alcohol-related seizures** are most common after cessation or reduction of alcohol. They are usually nonfocal, tonic-clonic seizures which are symptomatic in nature, and do not represent chronic epilepsy.

Most **adult-onset epilepsies** consist of focal seizures, and many are symptomatic of significant focal cerebral pathology. Of these focal seizures the most common are complex partial seizures.

Although certain metabolic disorders such as uremia, hypoglycemia, and hypocalcemia can cause nonfocal tonic-clonic seizures, all new tonic-clonic seizures should be considered focal until proven otherwise, and a focal etiology should be sought.

*Diagnosis. Taking a careful history of the attack from the patient and a witness is the key to an accurate diagnosis of seizures and epilepsy.*

**Teaching Points:** Important seizure characteristics are:

1. Time course: 1-2 minutes for focal and tonic-clonic seizures, 5-15 seconds for absence, myoclonic, and atonic seizures;
2. Stereotypy: although seizures and their auras may take many forms, in the single patient seizures do not vary. Infrequently a patient may have 2 or even 3 different seizure types, even in such cases each type repeats itself over and over.
3. Simplicity: the patient who reports 5 or 6 "auras" such as fear, and an abnormal taste, and sweating, and chest pain is more likely to be having panic attacks than seizures.
4. Testimony of a witness, as with syncope, may be vital: does the witnessed episode conform to the person's usual type of 'seizure'? How many different types does the person manifest. Briefly interviewing a witness of an ambiguous attack, even by phone, is often decisive. For example, the patient may be unaware of loss of responsiveness and consciousness during a complex partial seizure. He or she may not remember feeling the focal onset of a tonic-clonic seizure, but may have alerted a witness that an attack was starting.

## Diagnostic Pitfalls

Episodes of loss of consciousness without loss of postural tone are almost always complex partial seizures in adults, but are commonly absences in children. Misdiagnoses are common, but a careful history is almost always clearly diagnostic:

**Table 1. Differential features of complex partial and absence seizures**

	<b>Complex Partial</b>	<b>Absence</b>
Aura	Sometimes	Never
Duration	0.5 - 2 minutes	< 15 seconds
Behavior	Motionless or Automatisms	Motionless, stare
Postictal	Confused	Alert

Syncope with accompanying twitching of the limbs is often misdiagnosed as epilepsy. Children and adolescents are at highest risk, and attacks are situational. A **history** of the usual presyncopal symptoms usually makes the diagnosis clear.

**Breath-holding attacks** in children are easily diagnosed with a good history. They usually occur around 2 to 3 years of age, and usually follows angry crying. The child suddenly stops, becomes cyanotic and apneic, and then slumps unconscious. There is also a **pallid form**, often triggered by minor head trauma. The child turns suddenly pale and becomes briefly unconscious and apneic. Recovery from both forms is rapid.

**Panic attacks** are usually accompanied by typical emotional and autonomic symptoms: some combination of fear, a feeling of impending death, chest pain, palpitation, shortness of breath, tingling in the hands or lips, weakness, tachycardia or sweating. Attacks usually last a few minutes but often recur continuously or repetitively for hours.

*What should you expect to find on examination?*

Answer: The examination in the patient with epilepsy is often normal, but sometimes may reveal manifestations of many types of acute or chronic, focal or diffuse, cerebral disease. The neurological examination and developmental profile in most of the common genetic epilepsies, for example, are normal.

Adults with new onset seizures should be carefully examined for signs of focal cerebral dysfunction. Children should be scrutinized for skin changes typical of neurocutaneous syndromes such as Sturge-Weber disease, neurofibromatosis, or tuberous sclerosis.  
What diagnostic tests may be useful?

## Diagnostic Tests

If the diagnosis is not clear after a thorough history is taken, other investigations may prove unhelpful or even misleading.

The **electroencephalogram** (EEG, \$250 to 400) records electrical potentials generated by neurons in the cerebral cortex, and in patients with epilepsy often shows interictal cerebral discharges called spikes.

The EEG is best used to:

1. Confirm a diagnosis of the type of seizure disorder. (When positive, for example, it clearly distinguishes between absence and complex partial seizures. Myoclonic epilepsy and benign Rolandic epilepsy also produce characteristic EEG patterns.)
2. Assess the efficacy of AED therapy in patients with absences. Other types of seizure disorders continue to show spikes, despite clinically adequate treatment.
3. Assess the prognosis for withdrawal of antiepileptic drugs. (See below.)

Although traditionally used in diagnosis, the EEG does not meet modern criteria for an accurate diagnostic test for epilepsy. It is diagnostically insensitive in many types of epilepsy, giving false negative results in 30% to 50% of cases of focal seizure disorders. Diagnostic sensitivity for absence and myoclonic epilepsies is better, reaching 80% to 90%. The diagnostic specificity of EEG abnormalities is also unsatisfactory: inaccurate, "over-read" EEG readings are distressingly common, especially in children. In addition, some children or adolescents who have never had a seizures but who have a positive family history of epilepsy have true epileptiform spikes on their EEGs. One to three percent of adults without epilepsy may also have EEG spikes. Because an incorrect diagnosis of epilepsy may have profound effects on self-esteem, employment, medical insurance availability, driving, and long-term use of pharmaceuticals, such false positive rates make the EEG inappropriate as the sole or major basis for a diagnosis.

The **24-hour ambulatory EEG** (\$1000) is not indicated in most epilepsies. The test is most appropriately used in the diagnosis of frequent (every day or two), episodic, possibly ictal symptoms which need to be "caught" on the EEG. The usefulness and reliability of the computerized EEG called "brain mapping" (\$1000-\$3000) is controversial, and is currently recommended by the American Academy of Neurology only as a research tool.

**Brain imaging** is indicated for every newly diagnosed seizure disorder, with the exception of those known to be genetic and unassociated with detectable cerebral pathology, eg, benign Rolandic epilepsy, childhood absence or petit mal, juvenile myoclonic epilepsy. Enhanced **magnetic resonance imaging** (MRI, \$1000 to \$2000) is the technique of choice, since it is more sensitive than CT for certain neoplasms, tuberous sclerosis, small vascular malformations, and cortical migrational anomalies.

When there is reason to suspect that the patient's episodes may be psychogenic in origin ("pseudoseizures"), a serum prolactin level drawn within 15 minutes of an attack may be useful. Serum prolactin rises sharply, to 2 to 3 times normal, after all tonic-clonic seizures and after many complex partial seizures.

## **Treatment**

Many seizure disorders can be successfully treated by primary care doctors. In most cases this includes initiation of treatment.

In contemplating this, the following points may be useful.

An accurate diagnosis of the type of seizure is essential, since it dictates the appropriate choice of antiepileptic drug (AED).

For first-line therapy:

**Simple partial, complex partial, tonic-clonic**

carbamazepine (CBZ, Tegretol<sup>®</sup>)

phenytoin (PHT, Dilantin<sup>®</sup>)

valproate (VPA, Depakote<sup>®</sup>)

**Absence, myoclonic, atonic**

valproate

ethosuximide (ESX, Zarontin<sup>®</sup>)

lamotrigine

Other AEDs have been approved over the past several years. They are generally used as second-line therapy, and are probably most appropriately recommended by a consultant (neurologist).

These are: lamotrigine (LAM, Lamictal<sup>®</sup>), gabapentin (GP, Neurontin<sup>®</sup>), topiramate (Topamax<sup>®</sup>), tiagabine (Gabitril<sup>®</sup>), felbamate (Felbatol<sup>®</sup>), oxcarbazepine (Trileptol<sup>®</sup>), zonisamide (Zonegran<sup>®</sup>), and levetiracetam (Keppra<sup>®</sup>).

**Other Principles**

**Phenobarbital** is appropriate in the neonatal period, but is *best avoided for chronic use*.

**Valproate**, especially in combination with other AEDs, *is generally not used in infants under 2 years of age* because of potential hepatic toxicity.

**Phenytoin** *is better used in adults than children*, in whom it can have significant unwanted effects such as acne, gum hypertrophy, hirsutism, and possibly coarsening of facial features.

**Teaching Points**

1. In general it is good to aim for the lower therapeutic level of the AED that you choose.
2. If seizures recur, slowly increase the dose until seizures are controlled or the patient reports dose-related side effects (**Table 4**).
3. If the first AED fails, slowly add a second and follow the same dosing strategy.
4. **Monotherapy is preferred to polytherapy**; if the second drug is effective, taper the ineffective one.
5. Use "**therapeutic blood levels**" **only as guides**; some patients are well controlled at lower levels, others are comfortable at higher levels, and need the higher dosage for seizure control.

Because their therapeutic doses are very close to their toxic doses, and because patient compliance is vital to successful therapy, management of AEDs requires careful attention to drug kinetics and interactions. Dosing frequency, for example, should be based on the drug's half-life (**Table 2**), avoiding more than three times per day dosing.

**Table 2: Average half-lives of common AEDs**

carbamazepine	8 hours
phenytoin	24
valproate	8
lamotrigine	12 with inducers 20 with inducer + valproate 70 with valproate
ethosuximide	6
gabapentin	6
phenobarbital	60

Most AEDs have effects on the hepatic P450 enzyme system, which metabolizes AEDs and other drugs, so interactions are common and often significant. Those which induce more enzyme activity may reduce the levels of other drugs, and those which inhibit them may raise levels. Enzyme-inducing AEDs, for example, may impair the effectiveness of oral contraceptives.

**Table 3: Effects of AEDs on P450 enzymes**

Inducers:	carbamazepine phenytoin phenobarbital primidone
Inhibitors:	valproate
No effect:	gabapentin lamotrigine ethosuximide topiramate zonisamide

Dose related side effects of the AEDs are predictable, occur in nearly everyone who takes a high enough dose, and reversible on lowering the dose. Idiosyncratic side effects are uncommon to rare, unpredictable, unrelated to dose, and sometimes serious. Some are listed in **Table 4**. All AEDs can cause allergic skin rash.

**Table 4.**

<b>AED</b>	<b>Dose Related</b>	<b>Idiosyncratic</b>
Carbamazepine (CBZ)	Diplopia, nausea Blurred vision	Neutropenia Hyponatemia
Phenytoin (PHT)	Ataxia, dizziness,	Gingival hyperplasia acne, hirsutism
Lamotrigine (LAM)	Blurred vision, insomnia	Skin rash
Valproic acid (VPA)	Fine tremor	Weight gain, alopecia, hepatitis*
Gabapentin (GP)	Drowsiness	Weight gain
Topiramate (TOP)	Cognitive changes	numb face, hands weight loss
Zonisamide (ZM)	Cognitive changes	

\*rare, mainly seen in children under 2 years of age on polytherapy

Many epilepsies remit in time. The rate varies between 80-90% (for childhood absence) and as low as 0% (for juvenile myoclonic epilepsy, with most focal epilepsies somewhere in between. Except in juvenile myoclonic epilepsy, after 2 years of successful therapy in children, and after 3 to 5 years in adults, discontinuation of AEDs should be considered.

*When may a neurologist be useful?*

1. If there is any doubt about the initial diagnosis, or about the specific type of epilepsy, a diagnostic consultation may be appropriate, and inappropriate tests avoided.

2. Most patients or parents facing a new diagnosis of seizure disorder have questions about the implications of the condition, treatment, genetics, driving, and prognosis, which may best be addressed by the specialist. Most neurologists will give the patient information about the Epilepsy Foundation of America, where group and individual counseling, help with insurance and employment problems, and other supports are free.

3. If both the first-line and the second-line AED fail to control the seizures, a neurologist can review the diagnosis and offer a further treatment plan. In some patients with medically intractable focal epilepsy, surgical treatment can prevent irreparable educational, vocational, and psychosocial damage.

4. All of the AEDs are thought to increase the risk of fetal malformations or developmental delay if used during pregnancy. How or whether to take AEDs during pregnancy should be discussed with every woman or girl of childbearing potential prior to pregnancy. The neurologist can usually provide the facts, perspective, and reassurance that will enable the patient to make informed choices.

5. Stopping AEDs after several years of successful treatment can be fearsome for the patient and the physician. Since an unsuccessful taper can have negative implications for the patient's job, driving, and safety, a thorough discussion of the relative risks and benefits of stopping AEDs can be provided in a single visit.

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## Strokes And Transient Ischemic Attacks

### Pathophysiology

Most strokes are ischemic, that is, they are due either to blockage of a cerebral artery from a thrombus, which develops at a site of atherosclerotic narrowing or from a fatty deposit (plaque), or to platelet emboli, which aggregate at such a site, then block a smaller artery downstream. Such ischemic strokes can involve the territory of a major artery such as the middle cerebral (the most common such site), or may be small, deeply located, and even asymptomatic. The latter can cause significant clinical symptoms, eg, hemiplegia if they involve a vital area such as the internal capsule. Many such tiny strokes are one of the major causes of chronic, progressive dementia, accounting for 30% in some studies.

The pathophysiology of cerebrovascular disease (CVD) has been found to vary by race. Small, multiple infarcts (lacunes) are more common in African-Americans. The cause may be a higher incidence of hypertension, but differing socio-economic status and rates of diabetes may also play a role. On the other hand, large infarctions caused by atherosclerosis of the carotid or large arteries in the brain (eg, middle cerebral) are more common in Caucasians.

### Risk Factors

Cerebrovascular accident (CVA) is the **third most common cause of death** in the United States and a common cause of physical and mental disability. The clinical approach to CVD has changed over the past 10 years because of advances in two directions:

First, a clearer delineation of risk factors for stroke, some of them modifiable, has enabled techniques of effective primary and secondary prevention to be better defined and carried out.

Second, effective acute treatments of stroke are beginning to be used and tested, making prompt recognition of the premonitory symptoms of stroke vital.

**Although the most obvious risk factor for stroke is advancing age, strokes can occur in young adults or even in children.** In the young, additional risk factors include migraines, intravenous drug use or the use of cocaine or amphetamines by any route, AIDS, and pregnancy.

**Hypertension is the most important modifiable risk factor for stroke.** In large population studies, even small reductions in blood pressure produce detectable reductions in stroke incidence. This also includes lowering of systolic hypertension (>140 ) in the elderly. Careful, skillful management of hypertension is the single most important contribution to stroke "management" that the primary care physician can make. Hypertension is also an important risk factor for multi-infarct dementia, which, once established, is untreatable.

**Atrial fibrillation**, whether or not it is due to rheumatic heart disease, has been found to be an independent risk factor for embolic stroke. If the heart rhythm cannot be normalized, anticoagulation can lower the incidence of stroke (see below).

**Cigarette smoking roughly doubles a person's risk of stroke**, an increase that is reversible within five years if smoking is stopped. The combination of hypertension and

cigarette smoking is associated with a stroke risk that is greater than the sum of the individual risks.

Other risk factors include a **family history of stroke, occurrence of a previous stroke, high cholesterol level, heavy alcohol use (particularly for intracerebral hemorrhage), sedentary lifestyle, hyper-coagulable states, and anticardiolipin antibodies.** Obviously some of these conditions are also modifiable through advice and other interventions.

In the short term, transient ischemic attacks (TIAs) are a "risk factor" for stroke. For this reason, diagnosis and proper preventive treatment are important.

### **Clinical Appearance**

Whether transient, ischemic or hemorrhagic, a stroke produces an abrupt focal neurological deficit. The symptoms of a TIA appear over seconds to a few minutes, and are generally gone within five to 30 minutes. By definition, the patient must return to normal within 24 hours. Reversible ischemic neurological dysfunction (or RIND), is a similar event which resolves within three weeks. The effects of a CVA, by definition, last more than three weeks. The clinical appearance of all three is identical at onset.

*Essential features of a cerebrovascular event are:*

1. abrupt onset, within seconds to minutes;
2. a focal neurological pattern such as hemiparesis or aphasia;
3. a clinical pattern conforming to a vascular territory.

When such a diagnosis is considered, the next question should be: "What part of the brain is affected?" If this question cannot be answered, then other diagnoses may be more likely. The following question should be: "*What has caused it?*" And the final question is: "*What can be done about it, or about the next one?*"

Common neurological features of cerebrovascular events involving the anterior (carotid) or vertebro-basilar (posterior) circulation are found in **Table 5**. When all of the patient's abnormal neurological findings "fit" onto one side of the cerebrum (eg, right-sided hemiparesis, right-facial paresis, aphasia), the CVA involves the anterior circulation. When the findings are "crossed," that is, they involve both sides of the body, the brainstem is usually the site of the event and the posterior circulation is involved (eg, numbness of the right side of the face and the left side of the body).

**Table 5: A guide to localization**

<p><b>Carotid circulation</b> hemiparesis hemi-sensory loss or numbness homonymous hemianopsia aphasia apraxia conjugate eye deviation</p> <p><b>Vertebral-basilar circulation</b> diplopia vertigo ataxia limb tremor/incoordination dysarthria "crossed" sensori-motor loss* homonymous hemianopsia nausea</p>
--

Loss or depression of consciousness can occur in the context of a stroke involving the brainstem or most of the left hemisphere, but as an isolated sign (without flaccid right hemiplegia, asymmetric unreactive pupils) or as a transient phenomenon it is generally not suggestive of a vascular event.

### **Transient Ischemic Events**

Most TIAs are repetitive (over days to weeks), fairly stereotyped, and last from several minutes to half an hour. TIAs involving the carotid circulation usually produce hemiparesis, numbness on one side of the body (or part of it), partial or complete ipsilateral monocular visual loss, or loss of speech. Vertebro-basilar TIAs usually produce loss of balance, vertigo, nausea or vomiting, or diplopia.

Transient neurological symptoms which are literally momentary or fleeting, drop attacks without other symptoms, transient loss of consciousness, and vertigo or dizziness which is related to movement or head position are *not* usually TIAs.

The diagnosis of TIAs is important because they often herald a more lasting cerebrovascular event, at a time when appropriate therapy may limit or prevent further CNS damage.

*What should you expect to find on examination?*

The physical examination of the patient with suspected TIAs should include a careful cardiovascular exam (?murmurs, arrhythmias, signs of peripheral vascular disease), and neurological exam (?limb weakness, facial asymmetry, sensory deficit, monocular or homonymous visual loss, carotid bruits).

*What diagnostic tests may be useful?*

Like syncopes and seizures, TIAs are a clinical diagnosis best made by a good history. Diagnostic tests can only confirm the presence and severity of cerebrovascular disease. Once the diagnosis has been made, brain imaging by MRI scan can show whether strokes have already occurred, and whether other causes of transient ischemia are present (eg, neoplasm, chronic subdural hematoma).

A CBC, SMA-18, prothrombin and thromboplastin times, fasting lipid profile, and EKG should be done. If cardiac embolism is suspected on the grounds of previous myocardial infarct or valvular disease, a cardiac echo exam (\$600) is indicated. When large vessel disease is suspected, a carotid ultrasound (Doppler) examination (\$600) should also be done, along with a transcranial Doppler (\$700). The latter are noninvasive tests which measure the presence and speed of bloodflow in the carotid, vertebral, and large intracranial arteries. Magnetic resonance angiography gives somewhat more detailed information about these and some of the smaller arteries, as well as the intracranial venous circulation. It is noninvasive, requires no contrast agent, and can be done at the time of a routine MRI scan.

### **Treatment**

Many TIAs are thought to be caused by clumps of platelets, which embolize from areas of stenosis in the carotid artery. Carotid endarterectomy has been shown to be more effective than medical therapy when tight carotid stenosis is present. When vertebro-basilar TIAs are occurring or no significant stenosis is found in patients with anterior circulation TIAs, medical therapy is indicated.

*What preventive strategies ought to be introduced?*

Aspirin can reduce the risk of stroke in patients with TIAs. The optimal dose has not been determined; successful trials have used 80 mg to 700 mg per day. Some stroke experts recommend a combination of dipyridamole and aspirin. When TIAs persist despite the use of aspirin, or if aspirin cannot be tolerated, ticlopidine or clopidogrel (Plavix<sup>®</sup>) can be used.

When a cardiac source of thrombotic embolization is found, anticoagulation is indicated. Coumadin producing an INR of 2 to 3 has been found to reduce the incidence of stroke in patients with atrial fibrillation, even when TIAs have not occurred.

For the long term, modification of risk factors should be stressed.

This is an area, which is evolving quickly. If available, consultation with a neurologist or stroke specialist may help the patient receive the most effective treatment for his or her specific type of vascular disease.

### **Stroke**

Most strokes present to an emergency room, or should be sent there immediately. The possibility of acute therapy makes it particularly important that the patient with an apparent stroke be evaluated quickly.

A typical history is vital but not always available. Risk factors should be assessed.

*What should you expect to find on examination?*

Focal neurological abnormalities consistent with cerebral or brainstem dysfunction are characteristic of acute stroke (see above). As in the patient with TIA, the cardiovascular examination and blood pressure measurements are particularly important.

If stroke is the working diagnosis at this point the case is a neurological emergency, and immediate consultation with a neurologist or Stroke Service may lead to a recommendation for acute treatment aimed at reversing or limiting the neurological damage. (See below and Chapter 10.)

**Initial diagnostic tests.** Meanwhile initial blood tests should be sent, including a complete blood count with platelet count, chemistry panel, prothrombin time, partial thromboplastin time, an echocardiogram, and syphilis serology. An electrocardiogram should be done quickly, 20% of strokes are associated with myocardial infarction. If the EKG is abnormal, cardiac enzymes should be drawn.

A plain computed tomograph (CT) of the head should be done immediately, to look for hemorrhage or neoplasm. An acute ischemic stroke may not be seen well, or at all, on an early CT scan.

At that point the physician will know whether the stroke is ischemic or a hemorrhagic. If it is ischemic, the physician may have a good working hypothesis about its cause (eg, cardiac embolism).

**Initial management.** At this point nearly all patients with an apparent CVA or possible RIND will be admitted to a hospital. The presence or absence of acute bleeding will determine whether the initial intervention can be done, often suggests the cause of the stroke, and in some cases forecasts the prognosis. For example, small, basal ganglia hemorrhage is often associated with chronic hypertension, can cause acute hemiplegia, but may have a relatively good outcome. A large hemispherical ischemic infarct which is seen on an initial CT scan may be followed within 48 hours by cerebral edema causing worsening focal signs, coma, and death. A large hemispherical or cerebellar hemorrhage may cause death immediately or within hours from intracranial hypertension or pressure on the brainstem. In the case of the latter, neurosurgical intervention, although infrequently done, can be life saving and should be considered.

Until it becomes clear whether or not an acute cerebrovascular event is a RIND or a CVA, the initial management is that of a CVA. The best goal for control of blood pressure in the chronic hypertensive is a contentious issue. Reduction of *malignant* hypertension (>200/120) is vital, because it can cause focal neurological symptoms and signs, which usually resolve when blood pressure is reduced. But overly aggressive lowering of blood pressure can reduce cerebral perfusion and cause worsening of the neurological symptoms, particularly in the context of

significant cerebral atherosclerosis and abnormal autoregulation of arterial blood flow caused by a stroke.

If the patient is known to be hypertensive, and malignant hypertension is not present, urgent lowering of blood pressure is probably not necessary.

Neither physical (hyperventilation) nor pharmacologic (mannitol, steroids) efforts to reduce increases in intracranial pressure in ischemic stroke are helpful in reversing the symptoms and signs of stroke. Occasionally hyperventilation and mannitol may be useful as interim measures when neurosurgical relief of intracranial pressure is contemplated.

If intracranial hemorrhage seems unlikely or has been ruled out by CT, 300 mg aspirin should be given. Anticoagulation in the face of an acute stroke may be indicated or contraindicated, depending upon the cause and the risk of subsequent stroke, and is best considered with the aid of a neurologist. The acute use of heparin or warfarin (Coumadin<sup>®</sup>) has been associated with hemorrhage into large ischemic cerebral infarcts. On the other hand, anticoagulation may safely prevent subsequent strokes when an embolic source has been demonstrated and the acute CVA is small.

Tissue plasminogen activator or tPA has been approved for the treatment of acute stroke within the first three hours of onset, and if certain other clinical indicators are present. The risk of hemorrhage, including cerebral hemorrhage, is significant, and tPA is best used in consultation with a neurologist.

Hyperglycemia has been associated with worse stroke outcomes, and should be corrected. For the same reason fever should be lowered. Proper hydration should be managed carefully.

*Consultation with a stroke service has been found to be associated with better outcome, and can be cost-effective.*

### **Subsequent Diagnostics**

Unless another cause of the stroke is found, a carotid ultrasound examination or magnetic resonance angiogram, another noninvasive test, should be done to look for evidence of carotid artery occlusion or stenosis. This is not merely to seek or confirm the cause of the current stroke, but to look for a preventable cause of the next one. In some patients with stenosis of the internal carotid artery near the bifurcation who have a stroke, carotid endarterectomy has been proven to reduce the risk of subsequent stroke. Cerebral angiography is not indicated unless the carotid ultrasound is suggestive of significant carotid stenosis and surgery is contemplated. A normal carotid Doppler examination makes surgically treatable carotid stenosis extremely unlikely.

If the diagnosis of a cerebrovascular event or its etiology remains in doubt, magnetic resonance imaging (MRI) of the brain with infusion may be helpful in revealing the typical vascular distribution of a cortical embolism, for example, or the vascular pattern of an arteriovenous malformation or tumor. Small, deep lacunar infarctions are also much better seen on MRI than on CT.

## Teaching Points

1. An acute stroke is a neurological emergency. Immediate evaluation of the patient and formulation of a working diagnosis are essential.
2. CT scans can usually be done on a STAT basis and can rule out or display acute cerebral hemorrhage.
3. Malignant hypertension should be treated as an emergency. Less dramatic increases in blood pressure should not be treated.
4. Acute treatment of cerebral ischemia may reverse patient's symptoms, or reduce the eventual neurological deficit or disability.

**Subsequent management.** After admission to the hospital, attention to physical care is important, so that intercurrent complications do not hinder whatever neurological recovery will occur.

### Table 6: Initial management

1. The disabled patient should be turned frequently.
2. A shoulder sling for the paralyzed arm may prevent humero-clavicular separation.
3. Mobilization should be encouraged as soon as possible.
4. TED (support) hose should be used in immobile patients.
5. A swallowing evaluation should be done in all brainstem or large hemispherical strokes, and appropriate modifications in diet made in order to prevent aspiration.
6. Aphasic patients should be seen by a speech therapist while still in the hospital.
7. Post-stroke depression is common, especially with left hemispherical CVA's, and should be promptly recognized and treated.
8. Early consultation with occupational and physical therapists will both expedite the patient's return to a prepared home and establish long-term connections for rehabilitation. Most occupational therapists will visit the patient's home and suggest relatively inexpensive but important modifications, especially in the bathroom and stairways, which can facilitate a disabled person's daily activities.
9. Gait training should start as soon as the patient is alert and medically stable.

Early consultation with a physical medicine and rehabilitation specialist will enable plans for rehabilitation to begin and expedite transfer to a rehabilitation ward or center.

Both the patient and the family need information about what has happened, and continuing reports about the patient's progress or lack of it. Usually no clear prognosis can be provided during the first 24 hours, unless massive cerebral hemorrhage has occurred.

In the case of a completed stroke, the pace of recovery quickly slows over the subsequent days and weeks, and in most cases there will be no significant functional changes after three months. Aggressive physical rehabilitation is appropriate during that time, but very gradual improvement may occasionally occur after 6 to 12 months.

### **Long-Term Management**

Modification of risk factors may lower the risk of further strokes and at the same time lower the risk of symptomatic coronary artery disease. The most common cause of death in patients who survive stroke is myocardial infarction.

### **Some Special Cases**

Strokes can occur in young adults and even in children. Risk factors include migraines, pregnancy, renal failure, lupus, AIDS, intravenous drug use or the use of amphetamines and cocaine by any route. The diagnostic workup of the patient under 50 should include serum for HIV, antiphospholipid antibodies, proteins S and C, antithrombin III, hemoglobin electrophoresis, and an autoimmune profile. A four-vessel angiogram should be considered for the young person who has an intracerebral hemorrhage.

## **Patient History Checklist**

### **Case X: Topic**

What features of the story lend credibility to a diagnosis of -----?

What do you understand to be going on (pathophysiology)

Each case: stress importance of good accurate detailed history taking

### **Epidemiology:**

Any data on national health costs and costs to patients?

Ages

### **Risk factors**

### **Triggers/provocative agents**

### **Circumstances**

### **Premonitory symptoms**

**What should you expect to find on examination?**

**What diagnostic tests may be useful?**

**What treatment strategies are available?**

**When may a specialist be useful?**

## Self-Assessment

1. The most important diagnostic aid in the diagnosis of epilepsy is the:
  - A. electroencephalogram
  - B. history of the attacks
  - C. MRI head scan
  - D. family history
  - E. B and C
  
2. Shaking or twitching in an attack of loss of consciousness:
  - A. may occur during vaso-vagal syncope
  - B. is diagnostic for epilepsy
  - C. occurs in all seizure types
  - D. rules out a diagnosis of pseudoseizures
  - E. A and C
  
3. Syncope caused by cardiac arrhythmias is characterized by:
  - A. vertigo at the start of an attack
  - B. sweating and nausea at the start of an attack
  - C. loss of consciousness lasting 1 to 2 hours
  - D. sudden loss of consciousness
  - E. may be associated with all of the above
  
4. Complex partial seizures:
  - A. are the most common focal seizure type
  - B. may included stereotyped movements of the lips or body
  - C. usually last 30 seconds to 2 minutes
  - D. may be caused by tumors or other focal brain lesions
  - E. all of the above
  
5. Early morning jerks of the limbs, tonic-clonic seizures, and good therapeutic response to valproate are characteristic of:
  - A. juvenile myoclonic epilepsy
  - B. salaam attacks
  - C. atonic seizures
  - D. many focal epilepsies
  - E. any of the above
  
6. In suspected epilepsy, the electroencephalogram:
  - A. may help confirm the type of seizure disorder
  - B. is rarely useful
  - C. should be repeated every two years
  - D. should be run for at least 24 hours
  - E. A and D

7. A newly diagnosed focal epilepsy:
- A. means a lifetime of medication
  - B. should be followed by an MRI scan of the head
  - C. may be treated by ethosuximide
  - D. requires a search for other cases in the family
  - E. none of the above
8. Effective drug treatment for tonic-clonic seizures may include:
- A. carbamazepine
  - B. lamotrigine
  - C. clonazepam
  - D. valproate
  - E. all but one of the above
9. Symptoms occurring at the start of a syncopal episode include:
- A. nausea
  - B. sweating
  - C. graying of vision
  - D. none of the above
  - E. all of the above
10. Risk factors for stroke include:
- A. older age, hypertension, and HIV
  - B. older age, seizures, and atrial fibrillation
  - C. hypertension, use of phenytoin, and migraine
  - D. all of the above
  - E. none of the above
11. Common symptoms of an anterior circulation (carotid artery territory) stroke include:
- A. hemiparesis
  - B. aphasia
  - C. loss of sensation on one side of the body
  - D. two of the above
  - E. all of the above
12. The diagnosis of syncope, seizure, or transient ischemic attack is best made by:
- A. a 24 hour electroencephalogram
  - B. an MRI of the brain
  - C. a careful neurological examination
  - D. a detailed history
  - E. none of the above

13. The risk of stroke in patients with atrial fibrillation can be best reduced by:
- A. aspirin
  - B. Coumadin
  - C. either
  - D. neither
14. An EKG in the patient with an acute stroke:
- A. is not cost-effective
  - B. often reveals a myocardial infarction
  - C. is indicated only if the pulse is irregular
  - D. is irrelevant
  - E. should not be done until the second day
15. An immediate CT scan of the head in the patient with a new stroke can identify:
- A. carotid stenosis
  - B. intracerebral hemorrhage
  - C. an acute lacunar infarction
  - D. none of the above
  - E. all of the above

## Answers

1. B
2. A
3. D
4. E
5. A
6. A
7. B
8. E.
9. E.
10. A.
11. E.
12. D.
13. B
14. B
15. B