Recognizing syncope usually only requires the best of one’s skills in history taking. Simply making the correct diagnosis, transmitting it to the family, and understanding their feelings may be of major help, not least because it allows the family to contact a parent support group. When difficulties arise, the dearth of evidence-based treatment options argues for a generally conservative approach.

What Is Syncope?

Syncope happens when the brain is abruptly deprived of energy. This occurs either because blood flow ceases, the brain’s oxygen supply fails, or some combination of the two. Not surprisingly, it is commonly a dramatic event, not in the sense of a histrionic swoon but owing to the appearance of the syncopal convulsion. It is important to realize that although some faints are synapses, synapses do not resemble the popular conception of fainting. The stereotype—in which the patient sighs, sinks to the floor, lies motionless with eyes closed, and finally recovers and says “Where am I?”—is wrong in every particular way. Knowing this gives the pediatrician a good start in arriving at the correct diagnosis.

Terminology

Using words with precision is central to making the correct diagnosis, especially in patients whose symptoms are complex. This particularly applies to those words used to label paroxysmal events. If this seems to be an attack on North American speech, it is not. The problem addressed here is universal, at least among the English-speaking peoples.

Seizure

In an epileptic seizure, changes in behavior—objective or subjective—are associated with a sudden change in the electrical activity of the brain, commonly as a hypersynchronous discharge of many neurons, usually neurons in the cerebral cortex. Nonepileptic seizures are sudden changes in objective or subjective behavior that do not have at their root a sudden cerebral discharge.

There are two main varieties of nonepileptic seizure. The most important is the anoxic seizure, which results from syncope. In an anoxic seizure, cerebral activity is extinguished. In contrast to both epileptic and anoxic seizures, there is no massive change in cerebral activity in the psychological type of nonepileptic seizure. Pediatricians who believe that they confine the term “seizure” to epileptic seizure may reflect for a moment on febrile seizures. Although most febrile seizures are epileptic in mechanism (albeit the child usually does not have epilepsy), it is certain that some febrile seizures are anoxic seizures.

Convulsion

What has just been said about seizure applies equally to the term “convulsion,” except that in a convulsive seizure the child convulses—that is, has some combination of jerks, spasms, and stiffenings. In contrast to epileptic seizures (eg, absence seizure), anoxic seizures are generally convulsive.

The term “convulsive syncope” implies, first, that there is a peculiar variety of syncope with clonic or tonic muscle activity and, second, that there may be an epileptic mechanism involved. Both of these presumptions are wrong: nonepileptic syncope is naturally convulsive.
Fit

“Fit” may not be a popular term for a convulsion in North America, but in Scotland, this author has found it a useful one, particularly in discussing with families the difference between a fainting fit (anoxic seizure) and an epileptic fit.

Attack, Event, Episode, Turn, Blackout, and Transient Loss of Consciousness

*Attack*, *event*, *episode*, and *turn* are nonspecific terms for something paroxysmal. *Blackout* and *transient loss of consciousness* (TLOC) have been used for episodes involving a loss of awareness whose cause is not clear.

Seizure Disorder and Convulsive Disorder

Some use these terms as if they were synonymous with epilepsy. Such use is hazardous, for it allows the difference between syncopal disorders and epileptic disorders to become blurred. Insofar as clarity of thought leads to better patient management, these terms should be jettisoned for good.

Words Used for Synapses

Typing syncope into the search window of PubMed or Ovid will retrieve articles on pallid, neurally mediated, neurocardiogenic, and vasovagal syncope but not breath holding, reflex anoxic seizures, or most of the items discussed below under “Dangerous, Rare, but Treatable Synapses.”

How to Take a History

It is sometimes possible to arrive at a diagnosis of syncope in a minute or two, but beware the knee-jerk diagnosis of epilepsy or psychological attacks. When the diagnosis is difficult, there are many steps to the conclusion.

Prolonged Interrogation

In the world of paroxysmal events, the diagnosis is as good as the history. The objective is to build a moving picture of the event from before it started until after it ended. The result will be better than a video recording because it will also include the mental experiences of the patient before and after the loss of consciousness that led to the consultation.

History from Both Patient and Witness

Obviously, there is a lower age limit for eliciting anything useful, but even a young child may reveal information not yet known to the parents. Not surprisingly, it is necessary to track down the witness who was present at the onset. Because of the situational context of most synapses, they are likely to occur at the nursery or at school. Reflex anoxic seizure or reflex asystolic syncope (RAS) is an exception: episodes tend to occur when the child is “off guard” at home. When the story is of a seizure, convulsion, or fit just in school, vasovagal syncope is the likely diagnosis (the child was probably standing and reading to the class), but one needs to check details with the teacher directly.

History from a Second Witness

When syncope in an infant always occurs in the mother’s presence but is witnessed by others, it is necessary to check whether those witnesses saw the actual onset.

Mime, Video, and That’s It!

The use of mime by the physician and by the parent is an extension of the verbal history in the diagnosis of motor seizures, including syncope. Showing parents a variety of video recorded events in other children and asking which, if any, resemble the episodes in their own child is also helpful. “That’s it!” indicates a hit. This technique is becoming easier now that CDs showing multiple examples of seizures and syncope are more widely available (see for example the episodes on the CD associated with Stephenson, 2001, and Stephenson et al, 2004).

Camcorder in the Home

The capturing of a natural event on film extends the clinical history to its limits. A difficulty is the time it takes from the onset until the camcorder is running, but the diagnosis of longer events, such as anoxic-epileptic seizures, may be confirmed in this way (see the CD with Stephenson et al, 2004).

Recognizing Syncope

Settings, Provocations, Precipitants, Stimuli, and Triggers

No part of the history is more important than this. Synapses occur in particular settings—in church, when reading in class, when blood is being drawn, in pregnancy—and are provoked by stimuli. Not every syncope has a discernible provocation, but good history taking will unearth provocations in some, maybe 90%, certainly some of the time. For the practitioner, the safest rule is to assume that if some events have typical precipitants, then all the events are synapses. In toddlers, a common precipitant is an unexpected blow to the head; in adolescents, it is more likely to be the sight of blood. How provocations may change with age is illustrated in the following example.

Case 1

This girl suffered her first episode of syncope at the age of 10 months after a slight bump to her head. The presentation was similar from then on, except that the episodes became more “severe” as she got older. When she was old enough to
talk, she would say “Oh Mum, I’ve hurt myself” and look pale. After 10 or 20 seconds, she would fall rigidly, making a gurgling noise, her hands and feet turning in and her back arcing. Her arms might jerk a little and her legs move as if she were pedaling a bike, but sometimes this motor activity was more violent. She would then “look like death” and not know what had happened for a couple of minutes before wanting to lie down and sleep.

From the age of 7 years she began to describe an aura. She would hear a noise, like a high-pitched screaming and something like a voice, though without words. Sometimes she would see red, a color she does not like, and sometimes she would have hallucinations, such as a train rushing toward her.

Her mother described how the triggers that would precipitate a syncope changed over the years. After the first episode after the head bump, all the syncopes in early childhood followed slight but unexpected pains, such as her finger being bent back. Later, she began to experience the same reaction on seeing a minor injury, such as a scab that had come off a wound. Then she began to experience the same reaction on seeing a minor injury, such as a scab that had come off a wound. Then she developed inevitable syncope at the sight of blood. Finally, even the thought of self-injury was enough to bring on an attack. On the day before this author saw her in consultation at the age of 13 years, she was warned—wrongly!—that her eyeballs would be pressed into her head, and within 2 minutes she was in a violent syncope, stiff and snorting.

In this girl, virtually every syncope had a recognizable provocation, but even if only a proportion of her convulsive attacks had been provoked, the diagnosis of (vasovagal) syncope would have been entirely secure. The range of unpleasant syncopagenic stimuli is enormous, but, to an extent, each affected individual has “preferred” stimuli. Whether there are important differences in the syncopes induced by different precipitants, such as pain and disgust, is not known.

Some precipitants, such as hair grooming, are not, as far as one can see, unpleasant. Fortunately for the pediatrician, the story that the convulsion began during a hair-cutting or blow-drying session immediately secures the diagnosis of syncope.

The question of exercise as a trigger will be discussed under “Dangerous, Rare, but Treatable Syncopes.”

Aura, Warning, or Prodrome

Many neurologists and pediatricians imagine that an aura means an epileptic seizure. However, subjective premonitory symptoms occur before most syncopes. The same rule applies as with provocations: if some events, attacks, seizures, convulsions, or fits begin with a typical presyncopal experience, then all these events are likely to be syncopal. The best-known symptoms are blurring of vision with blacking out, together with an alteration in hearing with fading of sounds and some sort of tinnitus, followed by loss of consciousness. This combination always implies syncope. Other symptoms, such as a sensation of dread, may be less specific, but clinical studies are unable to adequately test this point. In some individuals, abdominal pain is a common precursor. Clearly, there is a lower age limit for recognizing a syncopal aura.

Complex Aura

Although hallucinations, misperceptions, and out-of-body experiences (see below) commonly occur at the end of a syncope during the recovery phase, sometimes something of the sort is experienced at the onset, as described in Case 1 above.

Falls

One might imagine that falling is an inevitable component of syncope, but, of course, that is not so in the very young nor in those who are unable to walk for other reasons. It used to be thought and taught that in syncope, the fall was limp and flaccid. It is now clear this is not necessarily so: a stiff (tonic) fall occurs just as often.

Injury

It should come as no surprise that the abrupt falls of syncope may injure the patient.

Convulsions

Some sort of convulsion is the rule rather than the exception in “pure” syncope. The movements involved include myoclonus, varying from subtle twitches to a storm of violent jerks involving the whole body, together with spasms and tonic extensions. The spasms have some resemblance to the better-known epileptic infantile spasms. The tonic extensions may be severe enough to induce opisthotonus but may also be subtle. Asymmetry of all these features may be prominent, with an appearance akin to the asymmetrical tonic reflex of young infants. Although these syncopal convulsions may be dramatic, they do not include prolonged regular rhythmic clonic activity.

Eye Movements

Eye movements in syncope include upward deviation, down-beat nystagmus, and brief conjugate lateral deviation.

Vocalizations

The emitting of some sort of noise is common during the unconscious phase of a syncope. These sounds have been described as growling, moaning, cackling, barking, groaning, snorting, and gurgling (see Case 1). Often, there is a single brief inspiratory groan, particularly near the end of the syncope in a breath-holding spell.

Tongue Biting

Although very rare, the presence of some tongue biting does not exclude syncope.
Urinary Incontinence
This is common in syncope and of no diagnostic value. Obviously, it is extremely embarrassing for the sufferer, long before adolescence.

Automatisms
Complex movements during a syncope are not unusual but are usually very brief. Even if they are prolonged for minutes, the diagnosis of syncope should not be discarded as long as the other characteristic features of syncope—precipitants and prodrome—are present.

Hallucinations and Out-of-Body Experiences
If the child is old enough, it is likely that hallucinations are the rule rather than the exception, but such symptoms, including out-of-body experiences, will not be volunteered without sensitive questioning. These symptoms commonly occur as the individual is beginning to regain consciousness. Descriptions include visual and auditory experiences but not formed speech. One patient of this author described a dream in which firemen with a fire engine were spraying blood out of a hose.

Out-of-body experiences are also not rare, even though the feeling of floating up to the ceiling and looking down on one's body is unusual. More commonly, there is a feeling of passing into a dark tunnel and being hurled through space toward a bright light. A child who is coming round from a syncope in the school classroom may imagine that he is at home in bed and dreaming that he is at school. There may be no sense of personal identity, including not being aware of the existence of one's body or limbs, even if recovery is associated (as it sometimes is) with excruciating unlocalized pain.

Postictal Phenomena
It is said that confusion after syncope lasts less than 30 seconds, but this is not always so (see Case 1). Once again, one must accept that if the onset was as in syncope, then syncope is the diagnosis. Great fatigue, intense desire to sleep, pains, difficulty speaking, and wanting to remain on the ground may last a long time. An experience that everyone else is rushing about and talking too fast and too loudly is common and may last for days. In young children, nighttime sleep disturbance is upsetting for families.

Syncope versus Epilepsy
In the previous section, it was discussed how the pediatrician will know when a patient has syncope because most of management comes from getting the diagnosis right. The major importance of this is to avoid the erroneous diagnosis of epilepsy. Such an error is extremely common. Such an error leads to inappropriate alterations in lifestyle with unnecessary restrictions, completely unnecessary side effects from antiepileptic drugs, and, when a female patient grows older and becomes pregnant, inexcusable hazard to the fetus. Bear in mind that a “family history of epilepsy” is frequently wrong, maybe in 50% of cases.

Epileptic tonic-clonic seizures differ from synapses in these ways:
• They are never precipitated, provoked, or triggered, or, at any rate, never provoked by the stimuli that provoke syncope.
• The convulsion is always long—one minute or more.
• The convulsion is always rhythmic—fast generalized twitching that slows down.

If there is doubt, it is syncope!
A good example of the erroneous diagnosis of epilepsy follows, in this case by a neurologist who was not a pediatric specialist.

Case 2
The consultant neurologist wrote to the general practitioner: “Thank you for asking me to see this 7-year-old young man. As a toddler, he began to have attacks of loss of awareness, rigidity, and eye rolling, which would be induced by minor knocks. This has continued, and recently, he had an undoubted tonic-clonic seizure with incontinence of urine. Curiously, as far as I can tell from the mother’s account, every attack has been triggered by a minor bump on the head, and he has never had an attack out of the blue…It seems to me that this boy is having some sort of reflex epileptic seizure [and should continue] with sodium valproate…Even though two [electroencephalograms] EEGs have been normal…he should be on treatment for at least a couple of years free from attacks.”

The neurologist failed to recognize that a triggered seizure is a syncope, in this case RAS, as discussed in the next section.

Vasovagal and Similar Syncopes
The major problem—recognizing syncope—has now been solved by the clinical history alone. It remains to establish the underlying “cause.” Most childhood syncopes are likely variants of vasovagal syncope, otherwise called neurally mediated or neurocardiogenic syncope. Various other labels have been applied, including breath-holding spells (see Chapter 55, “Breath-Holding Spells”).

Vasovagal Syncope
This is the name given to the most frequent syncope of the older child, adolescent, and adult. It involves acute changes in the sympathetic and vagal outflows, but critical analysis indicates that the pathogenesis is still not understood. When such syncopes are severe, the mecha-
nism is likely to include prolonged cardiac asystole (20 to 30 seconds or more).

Reflex Asystolic Syncope
The term RAS has come to be used for the short-latency dramatic syncopes of younger children, as illustrated in Case 2. There may be nothing fundamentally different between this syncope and vasovagal syncope (see Case 1). Although there are superficial resemblances between RAS and (cyanotic) breath-holding spells—such that they have been called pallid breath-holding spells—the mechanism of RAS is vagal-mediated asystole. Hence, some now use the term RAS to mean reflex asystolic syncope.

Induced Syncopes
Sometimes syncopes are self-induced and sometimes induced by others.

Compulsive Valsalva
Some children with learning difficulties, particularly those with autism, seem to enjoy hyperventilating and then undertaking a powerful Valsalva’s maneuver. In this context, it is easy to mistake the resultant anoxic seizure for an epileptic seizure; sometimes, anoxic-epileptic seizures occur in this situation (see end of chapter).

Stretch Syncope
Healthy adolescents may unintentionally induce syncope by stretching, with neck extended.

Imposed Upper Airway Obstruction
In this dangerous manifestation of Munchausen syndrome (or factitious disorder) by proxy, the syncopes always begin in the presence of the mother, but she shows the syncopal infant to another adult—a relative or hospital staff—before recovery. Episodes cease immediately if the mother is no longer allowed unsupervised access.

Poisoning
The list is limitless, but a confusing situation arises as a manifestation of Munchausen syndrome by proxy when a mother feeds her child a tricyclic antidepressant. Such a drug may induce syncopes secondary to acquired long QT syndrome and independent epileptic seizures.

Dangerous, Rare, but Treatable Syncopes
The apparently life-threatening events induced by imposed upper airway obstruction in Munchausen syndrome by proxy, described above, are dangerous, rare, but treatable syncopes. More “organic” examples are described below.

Long QT and Other Cardiac Syncopes
This is an increasingly difficult area. One is taught—and one teaches—that syncope occurring during exercise may be cardiac, as in long QT syndrome. Difficulties include the facts that (1) all those carrying a long QT-associated ion channel mutation do not have long QT on a standard electrocardiogram (ECG) and (2) most of those who have exercise-related syncope may have vasovagal syncope with no cardiac disorder. However, if a patient experiences emotionally charged exercise that induces syncope (such as being chased by a dog) or syncope induced by a sudden sound or syncope during sleep (in which one has neither the obvious trigger nor the prodrome), then a long QT syndrome or similar tendency to ventricular tachyarrhythmia is highly likely. If there is doubt, an ECG should be ordered and the patient referred to a cardiologist. Recently, short QT syndrome has been described, associated with a high rate of familial mortality.

Hyperekplexia
In the neonatal variety of startle disease, slight stimuli induce a nonepileptic convulsion that may be tonic or clonic or a mixture of the two. A quivering vocalization precedes the silence when a profound syncope ensues, with subsequent anoxic seizure. The clinical diagnosis is made by the nose-tap test: percussion of the tip of the nose induces an obvious startle. Ictal treatment is by repeatedly flexing the baby (face to knee); further episodes are prevented by clonazepam or clobazam.

Familial Rectal Pain
In this rare disorder, episodes begin in the neonatal period. Stimuli include perineal cleansing and defecation. The presyncopal appearance has resemblances to hyperekplexia, but the clinical clue is harlequin color change of the face or of the trunk. Although this disorder is not a form of epilepsy, carbamazepine is usually helpful.

“Fainting” and Cerebral Syncope
The Hollywood stereotype of the swooning faint is likely to be a psychological (psychiatric) disorder. Head-up tilt test studies (see below) have shown that these “pseudosyncopes” may be easily induced. There have been recent descriptions of apparent syncopes without change in heart rate or blood pressure but with isolated reductions in cerebral blood flow shown by transcranial Doppler or near-infrared spectroscopy. Such findings have prompted the label cerebral syncope. But the possibility of concurrent hyperventilation with consequent hypocapnia has not been excluded in these studies. In any such cases, the pediatrician must think deeply and choose a referral physician with much care. Postural orthostatic tachycardia syndrome
is predominantly a feature of women of childbearing age; see <http://www.potsplace.com> for more information.

**Useless and Useful Investigations**

This author does not favor unnecessary tests. Seemingly innocent investigations may have adverse consequences.

**Electroencephalography**

It is difficult to think of any good reason for ordering a standard EEG in a child with a history of syncope. The main danger is that an irrelevant abnormality may lead to serious mismanagement. The next case illustrates this.

**Case 3**

A schoolboy began to have convulsions. The pediatrician thought that these must be syncopal because they were provoked—as when running—and there was an aura of dizziness, but he ordered an EEG nonetheless. Numerous centrotemporal spike discharges were seen, and because another episode had occurred in sleep, the diagnosis was changed to benign rolandic epilepsy. The boy had no episodes while on sodium valproate. At the age of 12 years, he collapsed and could not be resuscitated from his ventricular tachyarrhythmia. Review of the ECG strip on the original EEG record showed unequivocal prolongation of the QT interval. He had inherited long QT syndrome from one of his parents.

This case also illustrates the danger of the use of the “therapeutic trial” (here sodium valproate) in deciding if the diagnosis is epilepsy or syncope.

**Electrocardiography**

It is perhaps reasonable to ask for a standard ECG in any child with syncope, but if the history includes episodes during emotionally laden exercise and in sleep, cardiac consultation is mandatory. Missing the diagnosis of cardiogenic syncope is far more dangerous than missing the diagnosis of epilepsy.

**Ocular Compression**

Before the days of cardiac event monitors, this was a useful test when it was important to the parents for the doctor to see the RAS that resulted from prolonged asystole. Almost the only remaining indication now is when a young child with RAS is suspected of having absence status (and perhaps other types of epileptic seizure) induced by the syncope (see “Anoxic-Epileptic Seizures”).

**Head-Up Tilt**

There is a huge amount of literature on head-up tilt testing, much of it empiric. We now know that false-positives—syncope in control volunteers—are frequent. If tilting is used, the end point must be the exact reproduction of the natural attack, whether that is a syncope, a syncope with hyperventilation, or a psychological pseudosyncope. This is technically complex, in that it may involve continuous pCO₂ and EEG monitoring, and should inhibit unnecessary tilt testing.

**Home Event Monitoring**

The advent of small, lightweight ECG recorders that may be worn for weeks and the more recent introduction of implantable cardiac event recorders have simplified the diagnosis of the type of syncope and the confirmation of asystole in RAS in particular.

**Prolactin**

Prolactin levels increase after syncope as readily as after epileptic seizures and so are of no value in that differential diagnosis.

**Management**

**Diagnostic Imperative**

In the management of syncope, making the correct diagnosis is of such overwhelming importance that guidance on honing one’s diagnostic skills dominates this chapter. Just imagine a patient—a female child with seizures who grows up and by the time of her first pregnancy is taking two antiepileptic drugs because the first drug did not help—presenting with her mentally retarded, malformed offspring and asking why she had been prescribed these antiepileptic drugs, as she has discovered that she has had vasovagal syncope all along? It is just as important not to misdiagnose syncope as psychological or hysterical.

High-quality randomized, placebo-controlled, double-blind trials do not feature highly in any category of management. Some treatments may seem helpful only because many syncope tend to improve.

**Appreciation of Patients’ and Parents’ Perceptions**

Severe syncope are highly unpleasant convulsive events. To dismiss concerns by saying “It’s only a simple faint” is as heartless as saying “It’s only a simple fit” at the onset of epilepsy. The feelings of the individual who suffers syncope need to be sensitively explored. Having a severe syncope is unpleasant, both immediately before and for a long time afterward. Then there is the problem of how to deal with onlookers, friends, and family, not to mention unsympathetic emergency room staff.

Chapter 55, “Breath-Holding Spells,” deals with breath-holding spells, but parents of children with RAS do not like that label. They translate breath holding into temper tantrums due to bad parenting, although they know their
child cannot help it and is not in control. It is a reflex—a reflex arrest of the heart, beyond the child’s control.

Advice on Posture
Those who have already had a syncope will be on the ground and will not want to be moved. Those who have postural hypotension might like an alternative to lying down during the aura, that is, raising the arms above the head. Squatting on the spot is an excellent emergency measure. Of proven value in controlling or aborting vasovagal syncope is crossing the legs combined with muscle tensing (see Suggested Readings).

Dietary Advice
Adequate salt and plenty of fluid (water) are advocated. Ingesting a mug of water may be helpful when a syncope is expected, if that is not a contradiction in terms.

Medical Therapy
Many pharmacologic agents have been employed for vasovagal syncope and for RAS, including beta-blockers, atropine, mineralcorticoids, iron, and piracetam. There is not a strong evidence base for the use of any of them.

Psychological Approaches
Although synapses are “organic” disorders in the same sense that epileptic seizures are “organic,” psychological methods may help synapses, as in the case of epilepsies. Two examples may suffice.

The psychological term for the basis of vasovagal syncope triggered by “blood and gore” is blood-illness-injury phobia. One reportedly successful psychological approach is to teach the use of anger directed at the triggering stimulus.

One study reported the abolition of anoxic-epileptic seizures (in the form of clonic status epilepticus triggered by breath-holding spells) after “psychotherapy for the mother and daughter,” once it was found that a severely disturbed mother-daughter relationship was the trigger for the spells.

Cardiac Pacing
One double-blind study has shown that cardiac pacing may prevent RAS. This trial was undertaken, with ethical approval and informed consent, on children who had frequent severe synapses with RAS and prolonged asystole recorded on cardiac event monitors. Thus, it is known that pacing is effective in preventing RAS (Stephenson and McLeod, 2000), but the possible long-term harm of a pacing system in a growing child means that this should only be considered in very exceptional circumstances, such as when frequent severe attacks disrupt family, social, and educational life or when anoxic-epileptic seizures are frequent, and antiepileptic medication is not acceptable. Asystole of at least 10 seconds (probably at least 20 seconds) should have been demonstrated in at least two natural attacks. If possible, it should be demonstrated by ictal EEG or ECG recordings that the duration of isoelectric EEG is entirely accounted for by the duration of cardiac asystole.

Patient Support Groups
Several years ago, the author persuaded a mother from England to start a family support group to help those afflicted with RAS. Her organization (Syncope Trust And Reflex anoxic Seizures) now covers the whole range of synapses, and families around the world find such contacts extremely helpful. Severe synapses may be so strange that sufferers find it reassuring to contact others with the same experiences.

Anoxic-Epileptic Seizures
In childhood particularly, synapses—RAS, prolonged expiratory apnea, or compulsive Valsalva’s maneuvers—may be followed not only by the usual anoxic seizure but also by “true” epileptic seizures. The epileptic component is usually clonic—often running into epilepticus status—or an absence seizure, also often long. The epileptic component may also resemble a myoclonic absence. Most such children do not have epilepsy in the usual sense of recurrent unprovoked epileptic seizures: the epileptic seizures occur only after the syncope. The existence of anoxic-epileptic seizures should inhibit the casual use of the term “seizure disorder.”

Conclusion
Inevitably, only the surface of the vast subject of syncope has been skimmed. However, if this chapter has whetted an appetite for more knowledge and helped to make the diagnosis and management of syncope easier and more sympathetic, then it will have served its purpose.

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Suggested Readings


Practitioner and Patient Resources

Syncope Trust And Reflex anoxic Seizures (STARS)
P.O. Box 175
Stratford Upon Avon
Warwickshire, UK CV37 8YD
http://www.stars.org.uk
Patient and family support group for all types of syncope.

Dysautonomia Information Network
PO Box 55
Brooklyn MI 49230
http://www.potsplace.com

National Institute of Neurological Disorders and Stroke
Syncope information page.