An EEG is considered abnormal if it has findings known to be associated with a pathologic or disease state. As discussed in Chapter 8, “The Structure and Philosophy of the EEG Report,” this distinction is designed to prevent the interpreter from calling an EEG abnormal simply because it includes a finding that “looks unusual” or is uncommon, because uncommon findings may not be abnormal.

When deciding which types of studies should be labeled abnormal, it is useful to consider the referring physician’s purpose in ordering an EEG. Ideally, the clinician is confronted with a patient with a particular clinical picture and has formed a list of possible diagnoses, also called the “differential diagnosis,” that might explain the patient’s findings. If the result of an EEG could increase or decrease the chances that one or more entities from the differential diagnosis list is the correct diagnosis, then it may be reasonable to obtain the test. However, if the EEG is not likely to have an impact on the probability of any of the diagnoses on the list, then the test is probably not indicated. It is usually not a good idea to obtain an EEG just “to see what it looks like” without a specific question in mind that the EEG could potentially answer. A related concept is that an EEG is indicated if there is some likelihood that the result will affect the patient’s treatment. Simply reporting that the EEG looks “odd” or “unusual” will not likely be helpful to the referring physician. Rather, an EEG should only be considered abnormal if it contains a finding that has some association with a disease or an abnormal state.

The majority of EEG abnormalities do not specifically lead to a single diagnosis. Only a small minority of EEG abnormalities are associated with a short enough list of disease entities that they can be considered “specific” for one diagnosis or another. The majority of EEG findings are diagnostically nonspecific and are associated with a list of disease states that is lengthy and diverse, so much so that specific diagnoses usually cannot be suggested based on the EEG result. An example of a dramatically abnormal but nonspecific EEG abnormality is generalized delta slowing, a finding that is associated with so many types of abnormal states (e.g., coma, post-seizure state, meningitis, anesthesia) that the clinical implications of the finding can only be stated in the broadest terms in the EEG report. The utility of the EEG comes in combining the EEG result with the patient’s history and other findings to narrow the differential diagnosis.

The referring physician is usually best placed to put the EEG findings and the clinical story together to arrive at a clinical conclusion. Without the clinical history, the EEG is a considerably less powerful tool. Even when a clinical history has been submitted with the EEG request, the reader of the EEG should hesitate before suggesting specific clinical diagnoses. The submitted history may be incomplete and often lacks details such as the physical examination or certain laboratory or imaging findings. When reading the submitted history, the electroencephalographer usually cannot know whether that history is complete. For these reasons, the interpreting physician should resist the temptation to make specific diagnoses based on the EEG results. Rather, a list of diagnoses that have been associated with the observed abnormalities, a sort of “EEG differential diagnosis,” should be given at the end of the report, if feasible. The discussion given in the final clinical interpretation section of the report should take into account the clinical history provided, but the EEG differential diagnosis offered should not be limited by that history.

EEG abnormalities can be categorized in a variety of ways. Abnormalities may fall into the categories of 1) abnormal expressions of normally occurring rhythms (e.g., asymmetries of normal rhythms), 2) inherently abnormal rhythms (e.g., “slow” delta and theta rhythms in an adult who is awake), 3) certain repetitive or periodic patterns (e.g., burst-suppression patterns), 4) epileptiform abnormalities (spikes, sharp waves, etc.), and 5) abnormal “super-architecture” (e.g., abnormal sleep state cycling). These various abnormality types are discussed in the following sections. The first two abnormality families, abnormalities of normal rhythms and abnormal rhythms, are discussed by frequency range, starting with slow activity. Basic epileptiform abnormalities are discussed in this chapter. Various epilepsy syndromes and the EEG findings associated with them are discussed in Chapter 10, “The EEG in Epilepsy.”
ABNORMALITIES OF SLOW-WAVE (DELTA AND THETA) ACTIVITY

Descriptive Parameters for Slow Waves

Slow-wave abnormalities can be defined in multiple domains. The most obvious descriptor of a slow wave is its location. A slow wave may occur focally, such as in the left anterior temporal area or the right occipital area. Slowing may occur in broader regions, such as in the right posterior quadrant, in “anterior brain regions,” or over a whole hemisphere (see Figures 9-1 and 9-2). Such regional slowing can be considered a subset of focal slowing. Finally, slowing may also be diffuse or generalized.

Slow waves may be rhythmic or irregular (nonrhythmic). When there is a tendency to rhythmicity but the waves cannot be considered truly rhythmic, the intermediate term “semirhythmic” may be used (see Figure 9-3). Slow waves can be of varying amplitude. If slowing is only observed in a certain sleep stage, such as drowsiness or slow-wave sleep, this fact should be described. Slow waves may occur intermittently (in brief runs or bursts) or continuously (in long, continuous runs with few pauses). These different parameters are worth remembering and should usually be included in the written description of slow-wave activity.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythmicity</td>
<td>Irregular versus semirhythmic versus rhythmic</td>
</tr>
<tr>
<td>Amplitude</td>
<td>High voltage versus low voltage</td>
</tr>
<tr>
<td>Sleep Stage Specificity</td>
<td>Seen in wakefulness versus drowsiness versus sleep</td>
</tr>
<tr>
<td>Continuity</td>
<td>Intermittent versus continuous</td>
</tr>
<tr>
<td>Localization</td>
<td>Focal versus regional versus generalized</td>
</tr>
</tbody>
</table>

The parameters can be remembered using the mnemonic “RASCL.” Certain combinations of these parameters can define specific, well-described slow-wave abnormalities. For instance, a slow wave that is frontal, occurs intermittently, is rhythmic, and is seen during wakefulness has been given the specific name FIRDA (frontal intermittent rhythmic delta activity), discussed further later in the chapter.

Focal Slowing

Focal slow waves are the classic sign of a lesion in the cerebral hemispheres. Before the era of modern neuroimaging, the electroencephalogram was an important tool for the localization of cerebral tumors. Today the
magnetic resonance imaging (MRI) scanner has moved
to the forefront in localizing tumors, but the EEG may
still play an important role in identifying certain types
of focal lesions that may or may not be evident on neu-
roimaging. The EEG has the advantage over imaging
studies of identifying areas of electrical abnormality
which may be “nonanatomical,” that is, functional abnor-
malities of the brain that may not be visible on an MRI
or computed tomography (CT) scan. Examples of
“nonanatomical” slowing may include postictal slowing
(see Figure 9-4), slowing from trauma that has not
caused an MRI lesion (see Figure 9-5), or even migraine
(discussed subsequently).

Focal slow waves may mark an area of previous,
rather than acute, injury. Brain lesions that cause
focal slowing in the absence of epileptiform activity
may not necessarily be prone to seizures. Figures 9-6
and 9-7 show a left temporal slow wave abnormality;
the perinatally acquired lesion causing the slow wave
is shown in Figure 9-8. Figure 9-9 shows a subtle right
occipital slow wave brought on by hyperventilation. If
asymmetric slowing is only seen during hyperventila-
tion, especially when the asymmetry is relatively mild
as seen in this example, it is less likely to be associated
with pathology than spontaneously occurring slow-
wave asymmetries. Slow-wave asymmetries that alter-
nate sides, whether spontaneous or elicited by hyper-
ventilation, are much less likely to be clinically
significant.

Focal slow-wave abnormalities have generally been
associated with deeper lesions located at the level of
the deep white matter (as opposed to more superficial
gray matter lesions), although exceptions to this rule
do occur. As discussed next, more superficial abnor-
malities of cerebral cortex are classically associated
with decreases in beta activity.

Figure 9-2  A mild but abnormal increase in slowing is seen in the left posterior quadrant (blue rectangles) compared with the right posterior
quadrant (gray rectangles) during drowsiness.
Figure 9-3  Although not perfectly rhythmic, the slow waves seen in the left occipital (O1) channels (arrows) are not completely irregular (dots). Some of the unpredictability of the appearance of these slow waves is due to intermixing with other rhythms. Waves of this intermediate degree of rhythmicity can be termed semirhythmic.

Figure 9-4  Significant slowing is evident in the top eight EEG channels (left hemisphere) compared with the bottom eight EEG channels (right hemisphere). The fact that this slowing represented a postictal change after a seizure was confirmed by demonstrating clearance of the slowing on a repeat EEG 2 months later. Depending on the nature of the patient and the intensity and duration of the seizure, postictal slowing may last from several seconds to as long as four weeks.
Practical Approach to Electroencephalography

Figure 9-5  Slowing and sharp waves are seen over the right hemisphere in this patient following a brain contusion. Note the sharp waves (arrows) over the right hemisphere. Normal alpha range activity with short wavelengths (A scale) is seen on the left compared with slower, theta range activity with correspondingly longer wavelengths (B scale) on the right.

Figure 9-6  Close comparison of the temporal areas (shaded areas) shows an asymmetry of slow activity with increased theta waves on the left as a result of an old perinatal injury to the left temporal lobe tip in a 17-year-old girl. The arrows indicate individual theta waves in the left temporal area. The patient's scan is shown in Figure 9-8. The lesion resulted in complex partial seizures.
Intermittent Rhythmic Delta Activity

Frontal Intermittent Rhythmic Delta Activity and Occipital Intermittent Rhythmic Delta Activity

The term intermittent rhythmic delta activity (IRDA) refers to rhythmic delta activity occurring in brief bursts, usually lasting no longer than a few seconds, typically located either frontally or occipitally (see Figure 9-10). In some examples the bursts can be more generalized (see Figure 9-11), and occasionally they can be asymmetrical. IRDA is a pattern typically seen in wakefulness and is usually associated with processes of mild to moderate severity. Keeping in mind that the patient generally must attain some level of wakefulness to manifest frontal (FIRDA) or occipital (OIRDA) IRDA, these patterns would not be expected in patients whose recordings are restricted to more deeply sedated or comatose states.

One important feature of FIRDA and OIRDA is that these abnormal patterns do not suggest a specific localization. Surprisingly, the tendency for IRDA to occur either frontally or occipitally is not dictated by an anterior or posterior location of the patient’s lesion but rather by the patient’s age. Up to approximately 10 years of age, IRDA tends to occur in the occipital areas. By the early teenage years, IRDA tends to occur frontally. One reason that it is important to correctly identify an example of rhythmic slowing as IRDA is to avoid being trapped into inappropriately using IRDA as a “falsely localizing sign.” As implied earlier, FIRDA is not particularly associated with frontal lesions, and OIRDA is not particularly associated with occipital lesions; either can be associated with anterior or posterior brain abnormalities. Indeed, the location of the IRDA tells us more about the age of the patient than the location of the lesion.

It is interesting to note that the predilection of the hyperventilation response for the occipital area in children and for the frontal area in adults parallels the age dependence of FIRDA and OIRDA. In older patients, the hyperventilation response may mimic the appearance of FIRDA, and in younger patients it mimics OIRDA, which is to say that younger children tend to manifest the intermittent rhythmic slow of hyperventilation in the occipital areas.

FIRDA and OIRDA are etiologically non-specific, and can be caused by a variety of toxic, metabolic, and other processes that affect the central nervous system. Their presence signals some type of cerebral disturbance, focal or diffuse.

Temporal Intermittent Rhythmic Delta Activity

Temporal IRDA (TIRDA) should be considered separately from FIRDA and OIRDA. The presence of intermittent, rhythmic trains of delta activity in either temporal lobe has been associated with temporal lobe epilepsy (see Figure 9-12). Furthermore, the side of the TIRDA, which is usually unilateral, indicates the side of the lesion if one is present. This localizing property of TIRDA distinguishes it from the major types of IRDA (FIRDA and OIRDA). Thus, unlike FIRDA and OIRDA, TIRDA is considered a potentially epileptogenic abnormality and has localizing value.

Occipital Intermittent Rhythmic Delta Activity in Childhood Absence Epilepsy

A specific type of occipital intermittent rhythmic delta activity is seen in children with childhood absence epilepsy. Intermittent rhythmic 3-Hz delta activity, similar to OIRDA, can be seen in what can be either brief or prolonged runs in the occipital area in children who have childhood absence epilepsy. This type of rhythmic delta activity distinguishes itself from typical examples of OIRDA in that the runs can be quite prolonged, lasting many seconds.

Figure 9-7 Close-up views of the left and right temporal channels taken from the shaded areas of the previous figure are shown. Note the mixture of slow waves with wider bases in the top channels compared with the faster alpha rhythms with narrower bases in the bottom channels.

Figure 9-8 This is the T2-weighted magnetic resonance imaging (MRI) scan of the patient whose EEG is shown in the previous two figures. In this MRI sequence, the cerebrospinal fluid appears white. Note the loss of volume in the left temporal pole (arrow), which is responsible for the EEG asymmetry noted in the previous figures.
Figure 9-9  A subtle increase in slow-wave activity is seen in the right occipital area compared with the left (compare shaded areas). The posterior rhythm appears against a flat baseline in the T5-O1 channel (upper shaded area), but the posterior rhythm rides up and down on a low voltage wave in the T6-O2 channel (lower shaded area). The same comparison can be made between the P3-O1 and P4-O2 channels.

Figure 9-10  Frontal intermittent rhythmic delta activity (FIRDA) consists of rhythmic runs of delta activity in the frontal areas of varying duration (arrows). Although FIRDA appears frontally, the finding does not necessarily imply a frontal pathology (see text).
**Generalized or Diffuse Slowing**

In general, theta and delta rhythms are not expected in the waking adult EEG. In younger patients, however, theta waves may be seen in the normal waking EEG. The range of normal posterior rhythms in childhood may serve as a useful reminder that theta frequencies are commonplace in the EEG of the awake child (see Table 2-2 in Chapter 2, “Visual Analysis of the EEG.”) For instance, in individuals young enough that a posterior rhythm of 7 Hz is considered normal, other 7-Hz rhythms may also be seen elsewhere in the normal waking EEG.

The determination that an adult EEG is abnormal simply because theta waves are present is made more difficult by the fact that it is normal for theta waves to appear with drowsiness. Therefore, in one way or another, the electroencephalographer must establish that a patient is not simply drowsy at the time that theta waves are seen to label them abnormal. Interpreting an EEG as abnormal on the basis of the presence of theta waves that are, in reality, related to drowsiness would constitute a significant error in interpretation. Indications that a patient is probably awake would include the presence of a posterior rhythm, temporalis or frontalis muscle artifact, eyeblink artifact, or conversation. The technologist can document (or force) alertness by having the patient count or perform some other task to exclude drowsiness as an explanation for observed slow-wave activity.

The list of pathologic states that can cause diffuse slow-wave activity is long and includes almost any abnormal state that can cause a diffuse cerebral disturbance. The most common causes are postictal (postseizure) states, postanoxic states, diffuse traumatic injuries,
Figure 9-12  Temporal intermittent rhythmic delta activity (TIRDA) is seen in a patient with temporal lobe epilepsy. This rhythmic delta activity is seen best in the left temporal area (arrows), although the left parasagittal area is involved as well. In this example, some of the theta waves are sharply contoured, which is not a necessary feature of TIRDA.

Infectious or inflammatory processes of the nervous system, toxic states related to drugs or other metabolic derangements (hepatic, renal, etc.), and a large number of other processes that have the potential to affect large portions of the cerebrum. Most of the many possible causes of coma are also on the list of possible causes of diffuse slowing in the EEG. For this reason, it is not feasible for the electroencephalographer to give a specific differential diagnosis for this pattern in the report. Often the interpretation will summarize the possible etiologies as a “diffuse cerebral disturbance” or a “diffuse encephalopathy.” The interpretation may also specifically discuss any clinical entities that have been questioned in the clinical history. EEG patterns in coma, many of which consist of slow-wave patterns, are discussed in further detail in Chapter 12, “Electroencephalographic Patterns in Stupor and Coma.”

Migraine

Because it is such a common disorder, the role of the EEG in migraine merits a separate discussion. A large number of abnormalities have been described in patients with common forms of migraine (“common” or “classic” migraine) between migraine attacks, so-called interictal EEG abnormalities in migraine. Many of these claims have been challenged because of the lack of well-controlled studies. The main problem is that the high frequency of migraine in the general population increases the chance that an individual who has any particular EEG abnormality may also incidentally have migraine. Some authors have claimed that focal slowing, and even epileptiform activity, are seen with increased frequency interictally in migraine patients. The “high-frequency photic response” (the ability to maintain a photic driving response at particularly high flash stimulation frequencies) has also been described in migraine patients. None of these phenomena, however, has been clearly proved to have an incidence in migraine patients above that seen in the general population. Because there are no EEG abnormalities that have proved to be useful in helping to diagnose uncomplicated migraine patients between attacks, EEG is not recommended as part of the routine evaluation of migraine patients. The EEG may be indicated, however, in the rare cases in which there is a question that a headache symptom may represent an epileptic seizure phenomenon.
Complicated Migraine

Focal slowing, either rhythmic or irregular, may be seen during the course of complicated migraine attacks. Figure 9-13 shows an example of high-voltage irregular slowing over the left hemisphere during an attack of hemiplegic migraine (a rare type of complicated migraine) in a 12-year-old girl. Confusional migraine, another rare type of complicated migraine that occurs predominantly in children, may cause bilateral slowing in the EEG. In some cases, migraine with visual aura can cause slowing during an attack, particularly in the posterior quadrants, although in many cases the EEG is not particularly remarkable during attacks.

In summary, EEG abnormalities, usually in the form of slowing, can be recorded in some patients during migraine attacks (particularly during complex migraine attacks). Whether certain abnormalities are characteristic of the EEG between attacks in migraine patients remains an unsettled question.

“Sharp Slow”

At times, a hybrid between sharp waves and delta or theta waves is seen consisting of sharply-contoured slow waves. An example of these slow waves with sharpened peaks is shown in Figure 9-14. The informal term for this type of wave is sharp slow. Whether such sharply contoured slow waves represent a true epileptiform abnormality or simply a variant of slow waves is unclear; some may be truly epileptiform and some may not. A variety of claims have been made regarding the significance of sharply contoured slow waves, including their association with vascular abnormalities such as small strokes, although none of these associations has been definitely proven.

Abnormalities of Alpha Activity

Alpha range frequencies make their most dramatic appearance in the EEG in the form of the posterior rhythm. Asymmetries of the posterior rhythm are common, and certain asymmetries are even expected: the posterior rhythm is usually of higher voltage over the right, non-dominant hemisphere (see Figure 9-15). However, complete absence of the posterior rhythm may occur in a minority of otherwise normal individuals. It is also true that the posterior rhythm may be absent in individuals with brain injuries or other abnormalities, but in such cases the EEG usually shows other abnormal features. Therefore, an EEG whose only remarkable feature is absence of the posterior rhythm should be considered normal. Asymmetries of the posterior rhythm are discussed in more detail in Chapter 2, “Visual Analysis of the EEG.”

In some patients, alpha rhythms take up much of the posterior quadrants of the EEG during wakefulness. This occurs either when the field of the posterior rhythm extends far forward or when the posterior rhythm blends with mu rhythms, a normal variant rhythm seen in the central areas that may also be in the alpha range. Mu rhythms are discussed in more detail in Chapter 11, “Normal Variants in the EEG.”

ABNORMALITIES OF BETA ACTIVITY OR “FAST ACTIVITY”

Excess Fast Activity

Unusually high voltage or plentiful beta activity is the most frequently encountered abnormality of fast activity in the EEG. By far, the most common explanation
Figure 9-14  Sharp theta waves are seen in each temporal area (arrows). Note the varying degree of sharpness with some waves in the series appearing sharper and others appearing more rounded.

Figure 9-15  This EEG shows the typical asymmetry of the posterior rhythm. Higher voltages are seen in the right occipital area (P4-O2 and T6-O2 channels) compared with the left (T5-O1 and P3-O1 channels).
for excess fast activity in the waking EEG is pharmacologic effect. Benzodiazepines (such as diazepam, lorazepam, and clonazepam) and barbiturates (such as phenobarbital and pentobarbital) are the most common causes of increased fast activity in the EEG, although other categories of medication may cause this effect less frequently. Because these two classes of drugs are frequently used in both the acute and chronic management of seizures, it is common to see increased fast activity as a pharmacologic effect in the EEG. Drug-related increases in beta activity are usually diffuse but may also be frontally predominant (see Figures 9-16 and 9-17). The increased beta activity seen as a normal finding in many patients at onset of drowsiness usually subsides with deepening Stage II sleep.

Whether excess fast activity in the EEG as a drug effect should be considered an abnormal finding is, to some extent, a question of semantics. According to the definition used in this text—that a finding is abnormal only if it is associated with a pathologic or disease state—excess fast activity caused by drug administration would not represent a true abnormality; having received phenobarbital or diazepam should not be considered, in itself, a disease state. (A normal volunteer receiving these medications would manifest an increase in fast activity in the absence of a pathologic state.) Many readers note the presence of the fast activity in the interpretation without calling the tracing abnormal solely based on the finding of increased fast activity.

A relatively rare explanation for dramatically increased fast activity is developmental delay or mental retardation, especially when associated with a dysgenetic lesion of the brain (e.g., lissencephaly). Increased fast activity in the absence of other explanation should not prompt the reader to jump to the conclusion that a dysgenetic brain lesion is present. Often the medication list is incomplete, or, alternatively, barbiturates may have been administered previously and subsequently discontinued but may not yet have been cleared from the bloodstream. Fortunately, the increased beta activity that is seen with dysgenetic lesions usually occurs in the context of an otherwise significantly abnormal EEG and is easy to recognize. Also, in these cases the beta activity is often of a slower frequency than is seen in normal individuals.

“Slowed” or Absent Fast Activity

Although cortical fast activity recorded during wakefulness is usually within the range of 18 to 30 Hz, a reduction in the frequency of fast activity may also represent an abnormality. “Slow” fast activity is most commonly encountered in cases of diffuse cortical injury, as may be seen after an anoxic episode. In other cases, decreased frequency of fast activity may represent the pharmacologic effect of certain sedative or anesthetic medications, such as those used for pharmacologic induction of coma.

![Figure 9-16](image_url)

Figure 9-16 A large amount of beta activity is distributed across all channels. Scanning for low-voltage spike activity is made considerably more difficult by the large amount of fast activity.
A relative absence of fast activity in the EEG may also represent an abnormality. Absent fast activity may be seen in the setting of severe, diffuse cortical injuries (in which case, other abnormalities are typically present), although it must be appreciated that some patients have less fast activity in the EEG than others, and apparently decreased amounts of fast activity in the waking EEG may represent a normal variant in some individuals.

In assessing the amount of fast activity in an EEG, the reader should not be led astray by the situation of a tracing that is displayed with low amplifier gains. Tracings with large amounts of high-voltage slow activity are typically displayed with low amplifier gains so that adjacent channels will not collide and cross. At these lower gains, the lower voltage fast activity, although present in normal amounts, may not be visible in the display. This effect is discussed in more detail in Chapter 2, “Visual Analysis of the EEG,” and illustrated in Figure 2-24.

**ASYMMETRIC RHYTHMIC ACTIVITY**

In cases in which not just a single frequency range but all frequencies of electrical activity are depressed in a certain area, the shorthand term “decreased rhythmic activity” can be used to refer to a decrease in activity of all frequencies (see Figure 9-19). There are many possible causes for decreased rhythmic activity in a particular region, and care should be taken not to suggest a list of possible causes that is too narrow because even non-cerebral causes are on the list.

When rhythmic activity is decreased over a discrete area, an anatomical injury such as loss of brain parenchyma (e.g., a stroke or encephalomalacia) is an initial consideration. Abnormalities in the various anatomical spaces should also be considered, including the intraparenchymal space (the brain tissue), the extra-axial space (including the space between the brain and the skull), and the extracranial space (the scalp). Orderly consideration should be given to the possibility of fluid collections in the subdural, subarachnoid, or epidural spaces, which may consist of blood, pus, or cerebrospinal fluid. Individuals with large but asymmetrical subgaleal

---

**Figure 9-17** The amount of fast activity in this patient has been markedly increased by the administration of lorazepam. In this example, the fast activity is most prominent in the anterior quadrants (arrows).
Figure 9-18  The waking EEG of this patient who has suffered a left middle cerebral artery infarct shows decreased fast activity over the area of the stroke. The close-ups emphasize the lower voltage of fast activity over the left parasagittal area compared with the right.

Figure 9-19  This patient, who has experienced a left hemispheric stroke, shows a more marked asymmetry of rhythmic activity (i.e., for waves in multiple frequency ranges) in the highlighted areas. Whereas in the previous figure the asymmetry was mostly restricted to fast frequencies, in this example all frequencies are relatively diminished over the left central and temporal areas (shaded rectangles), giving them a relatively flat appearance. Compare with the right central and temporal areas (unshaded rectangles). Incidental note is made of left occipital spike-wave discharges.
ASSESSMENT OF VOLTAGE ASYMMETRIES

Hemorrhages or other scalp swelling such as a caput succedaneum (a type of scalp swelling seen in the newborn soon after delivery) can cause dramatic voltage asymmetries even in the absence of cerebral abnormalities. It would be undesirable to make the error of interpreting the EEG of a patient who has been in a car accident as indicating a brain injury in the left hemisphere simply because of a large hemorrhage in the left scalp. Therefore, the whole gamut of intra-axial and extra-axial/extracranial abnormalities should be considered in the case of an EEG tracing with a voltage asymmetry that includes a wide band of frequencies. Figures 9-20 and 9-21 show a dramatic asymmetry of rhythmic activity as an aftereffect of surgical removal of the right hemisphere for refractory epilepsy.

ASYMMETRIES OF SLEEP ELEMENTS

In the case of asymmetric sleep spindles or vertex waves, the magnitude and persistence of the asymmetry are important in deciding whether or not to label the asymmetry as abnormal (see Figure 9-22). Mild asymmetries in the amplitude of sleep spindles are occasionally seen and may be of little consequence. Spindle voltage asymmetries greater than one third to one half bring up the question of a true functional brain asymmetry (see Figure 9-23). In certain patients, occasional
asymmetry of vertex waves is seen and may not be significant. Mild or nonpersisting shifts of vertex wave maxima to the left or right may not be clinically important. More persistent or dramatic asymmetries in the appearance of spindles or vertex waves may, however, reflect an asymmetry of the brain structures that generate these waves and therefore may be clinically significant. Asymmetry of sleep elements should be confirmed in a referential montage whenever possible.

**Extreme Spindles**

“Extreme spindles” is an exaggerated spindle pattern in sleep and represents a rare abnormality. These abnormally prolonged and persistent spindles have been described in mentally retarded children (see Figure 9-24). Rarely, this abnormal spindle pattern may even intrude into wakefulness.

**EPILEPTIFORM ACTIVITY**

In the 1974 glossary of EEG terms (Chatrian et al., 1974), epileptiform activity was defined as “distinctive waves or complexes, including spikes and sharp waves, that are distinct from background activity and resemble those recorded in a proportion of human subjects suffering from epileptic disorders.” The word *epileptiform* itself suggests that these are waves that appear in the “form” of epilepsy but do not constitute an example of epilepsy (i.e., epileptic seizures) itself. By definition, epileptiform activity refers to abnormal discharges that occur between seizures—the formal definition specifically excludes epileptic seizure discharges themselves. Many epileptiform discharges (i.e., discharges that occur between seizures) do resemble the component discharges of actual epileptic seizures. The single interictal spike seen in a patient’s EEG may resemble the component parts of the train of rapid spikes that constitutes that patient’s epileptic seizure discharge, although this is not always the case.

Occasionally, it may be difficult to distinguish interictal activity from electrographic seizure activity (discussed in more detail in Chapter 10, “The EEG in Epilepsy”). The electroencephalographer must always be conscious of the distinction between discharges that take place during the seizure or “ictus,” referred to as ictal activity, and activity that takes place between seizures, interictal activity. The definition of epileptiform activity has been extended by some to include certain paroxysmal bursts of slow-wave activity, reasoning that these bursts are the indicator of a decreased seizure threshold in certain patients (see the discussions of OIRDA associated with childhood absence and TIRDA earlier in the chapter). Keeping in mind that approximately 1% of asymptomatic adults and 3% to 4% of asymptomatic children will have epileptiform activity in the EEG, it is important to avoid the error of equating epileptiform activity in the EEG with the diagnosis of epilepsy.
Persistent asymmetry of sleep spindles is considered abnormal. Note that the spindle visible in the right central region (lower shaded area) and the midline can barely be seen during this interval in the left central region (upper shaded area).

An example of extreme spindles is shown in a referential montage. Note that spindle voltages are highest in the C3 and C4, F3 and F4, and Fz and Cz electrodes, as expected.
Electrographic Versus Electroclinical Seizures

By definition, a seizure is an event during which a clinical change in the patient is accompanied by a characteristic abnormal change in brain waves. The clinical change may either occur outwardly and be observable by those surrounding the patient or the change may be experienced subjectively by the patient. The outward clinical change may be as obvious as generalized tonic-clonic seizure activity or more subtle, such as a mild slumping of the shoulders or slowed responsiveness. Internally experienced seizure manifestations, such as olfactory hallucinations or the psychic experience of fear, may be invisible to outside observers. Because most seizure events do not occur while a patient is being recorded on EEG, the brain wave portion of the definition implies a presumed change in the EEG at the time of the clinical event had the patient been recorded by an appropriate technique at the time.

The question may arise as to how to classify an EEG seizure discharge that is not accompanied by a known clinical change in the patient. EEG seizure discharges during which the patient’s clinical status is unknown or apparently unchanged are termed electrographic seizures. To distinguish electrographic seizures from those seizures that meet the classic seizure definition of concurrent clinical seizure behavior and EEG seizure discharge, the term electroclinical seizure can be used. Imagine a patient who has 10 seizure discharges recorded in the EEG. Five are recorded during wakefulness and are associated with behavioral arrest, and the other five are electrically identical discharges recorded during sleep, a state during which behavioral arrest cannot be observed. The electroencephalographer may then report that five electroclinical seizures and five electrographic seizures were recorded during the study.

Whether a seizure is truly “electrographic” as opposed to “electroclinical” can sometimes approach a philosophical question. How does one deal with an electrographic seizure discharge during sleep as described in the preceding paragraph or in a child too young to describe experience? Similar questions arise when an individual recorded on video has an electrical seizure discharge but is not in the midst of an activity. In such a case, had the patient been speaking or carrying out an activity, perhaps a behavioral arrest or slowed response time would have been observed. Often such questions regarding individual events may not be answerable and the convention is to label any seizure discharge as an electrographic seizure if no behavioral or experiential change can be ascertained. Likewise, in some patients, it can be difficult or even impossible to determine whether a run of discharges represents an interictal discharge or an electrographic seizure discharge. This is a particular problem in patients with slow spike-wave discharges in which the ictal and interictal discharges can look quite similar.

Descriptions and definitions of epileptiform spikes, sharp waves, and spike-wave discharges are given in Chapter 3, “Introduction to Commonly Used Terms in Electroencephalography.” Because the large majority of epileptic patients do not experience an actual epileptic seizure during routine EEG recording in the EEG laboratory, it is the interictal epileptiform discharges that are the main tool in further defining an individual’s specific epilepsy diagnosis (see Figure 9-25).

Limitations to the Localization Value of Interictal Epileptiform Activity

Interictal discharges can be valuable in localizing the epileptogenic zone in epileptic patients, but there are instances when their localization value may be limited. Certain cerebral regions are particularly difficult to localize using scalp recordings. Some types of discharge may even be “falsely localizing,” suggesting a localization that is different from their scalp source. This is especially true of discharges that arise from areas of cortex that are not adjacent to the scalp, such as the interhemispheric fissure, orbital surfaces, mesial temporal lobe, basal surfaces of the occipital lobes, or insular cortex.

A second potential problem is that the location of an epileptiform discharge, even if well ascertained, may not always indicate the seizure onset zone. Interictal discharges often arise from the seizure focus, but this is not true 100% of the time. Keep in mind that some individuals with interictal discharges may never have a seizure, which implies that at least some interictal discharges never give rise to seizure activity. Also, there are some patients with seizures who never have interictal discharges. Acknowledging these two situations, if it is possible for an area of the brain to generate interictal discharges but never originate a seizure and another area may generate seizures but be interictally quiet, it is easy to imagine an occasional circumstance in which an interictal discharge could occasionally “point to the wrong place” in some patients with seizures. Some individuals may have two separate foci of epileptiform activity, although seizures may only arise from one of the regions. In such a patient, the location of one of the discharges may suggest an incorrect localization. It is not hard to imagine how this could happen. Imagine a person who has experienced a patchy injury to the brain, perhaps from trauma or infection. Two areas are damaged, one in such a way that it generates abnormal epileptiform discharges but, by chance, not in a way that generates actual seizures. The second area is damaged in such a way that it generates both epileptiform activity and seizures. In such a patient, only one of the spike localizations is pertinent to understanding from where the seizures arise. Therefore, a spike localization often, but not always, corresponds to the area of seizure onset. Other factors such as the history, imaging results, and seizure semiology should be used to confirm localization.

A secondary use for localizing epileptiform activity, even in the absence of epileptic seizures, is to localize different types of lesions in the brain. In the past, electroencephalography was among the most useful tools for localizing brain tumors, but modern neuroimaging has supplanted electroencephalography.
Practical Approach to Electroencephalography

Figure 9-25 A run of spike-wave discharges is seen across the left temporal chain (black arrow) in a 15-year-old girl with complex partial seizures. The sharp slow waves seen at the end of the page (gray arrow) suggest a hybrid between slow waves and sharp waves, sharing the characteristics of both.

as a primary tumor localization tool. Still, despite the primacy of MRI in imaging tumors, epileptiform activity in the EEG may sometimes give the first clue to the presence of a tumor, as occurred in the patient discussed in the paragraph that follows on secondary bilateral synchrony.

Secondary Bilateral Synchrony

Secondary bilateral synchrony refers to the phenomenon of generalized discharges arising from a unilateral cortical focus. Typically we think of focal discharges in the EEG as associated with focal lesions and generalized discharges as associated with generalized processes or generalized epilepsies. Apparently generalized discharges in the EEG are not always associated with true generalized seizure onsets, however. Occasionally, a unilateral focal lesion can originate a discharge that spreads rapidly and synchronously across both hemispheres. Such discharges, although arising from a single focus, may spread throughout the brain so rapidly that they are virtually indistinguishable from a primary generalized discharge. Figure 9-26 shows an example of apparent generalized spike-wave discharges. Figures 9-27, 9-28, and 9-29 are taken from other pages of the same patient's EEG and give clues to the phenomenon of secondary bilateral synchrony. The patient's causative lesion is shown in Figure 9-30. Because of this phenomenon, it is good practice to consider the possibility that any generalized discharge might be an example of secondary bilateral synchrony and to look for clues to the latter. One such clue is voltage asymmetry of the discharge—the discharge may be of persistently higher voltage over one hemisphere as compared with the other. An even more compelling finding is a consistent “lead-in” of the discharge from one hemisphere, the hallmark finding of secondary bilateral synchrony. In these cases, the discharge is seen to originate from one hemisphere followed by rapid generalization. Because close analysis of genuine generalized discharges may disclose an apparent onset that is several milliseconds ahead on one side compared with the other, the most compelling “lead-ins” are those of 2 seconds’ or more duration that always occur on the same side. In some patients, it is not uncommon for genuine generalized discharges to alternate the
apparent side of onset with brief “lead-ins” seen in one hemisphere, then the other. When the side of the lead-in alternates, this decreases the chances that the generalized appearance of the discharge is due to secondary bilateral synchrony.

The criteria for a definite diagnosis of secondary bilateral synchrony are fairly strict and include a definite “lead-in” of the discharge from one side, a possible voltage asymmetry, or possible focal interictal epileptiform activity seen at other times in the record arising from...
Practical Approach to Electroencephalography

The same page of EEG as was shown in the previous figure is displayed in a referential montage, confirming the presence of a voltage asymmetry.

Another page of EEG is taken from the same tracing as shown in the previous three figures. This example of the patient’s generalized spike-wave discharge now shows a clear and prolonged lead-in from the right hemisphere (arrows). An asymmetrical lead-in, especially if prolonged, is one of the most convincing clues to the presence of secondary bilateral synchrony.

The same side as the lead-in. The threshold for doubting that a generalized discharge is truly generalized (i.e., suspecting the diagnosis of secondary bilateral synchrony) is considerably lower. At times, focal lesions may cause discharges to generalize so rapidly that a lead-in is not identifiable. Therefore, every generalized discharge should be scrutinized for the possibility that it has a hidden focal onset, even if not meeting strict criteria for secondary bilateral synchrony. When other signs of a primary generalized epilepsy are not present
in the EEG, the reader may choose to warn of the possibility of a focal onset, even when generalized spike-wave or other generalized discharges are seen. If appropriate, the clinical correlation paragraph can include wording to the effect that “the generalized discharges present in this EEG are potentially consistent with a primary generalized epilepsy, although the possibility of a focal onset with rapid secondary generalization cannot be completely excluded.”

PERIODIC PATTERNS

The term periodic pattern refers to the presence of a periodic waveform in the EEG. The term periodic implies repetitive recurrence of the waveform with an approximately fixed interval between the waveforms. The term periodic may be compared with the term rhythmic, which implies a more fixed and regular interval. In fact, periodic patterns may sometimes be so regular as to appear rhythmic, but the term periodic implies the possibility of somewhat more variation in the intervals between waveforms.

A particularly useful classification system for periodic EEG patterns was described by Brenner and Schaul (1990). In this system, a periodic pattern is classified according to whether it occurs over a single hemisphere or both hemispheres (unilateral vs. bilateral), whether it occurs over both hemispheres in a synchronous or asynchronous (bilaterally independent) fashion, and whether the intervals between waves are less than or greater than 4 seconds (short interval vs. long interval). In practice, combinations of these three parameters result in four categories of patterns: 1) periodic lateralized epileptiform discharges (PLEDs), 2) bilateral periodic lateralized epileptiform discharges (BiPLEDs), 3) periodic short interval diffuse discharges (PSIDDs), and 4) periodic long interval diffuse discharges (PLIDDs). (See Table 9-1.)

PLEDs

Of the periodic patterns, the best known is PLEDs (see Figure 9-31). Of the four letters in this abbreviation, the “L” for lateralized is the most useful to keep in mind. The word lateralized is used in place of the word focal, reflecting the fact that, rather than being confined to a small area, the field of a PLED spreads across a whole hemisphere. If an apparent periodic discharge is focal rather than lateralized, the diagnosis of PLEDs should be questioned.

PLED waveforms, apart from being lateralized to a single hemisphere, may be simple, complex, or polymorphic. Typically, although not always, the PLED waveform contains sharp or spike-like features. PLEDs occur repetitively in the tracing approximately every 1 to 4 seconds. In general, when PLEDs are present, they are present for the entirety of the tracing; they do not tend to appear and disappear within a tracing. If a periodic waveform is seen only intermittently, the diagnosis of PLEDs should be questioned.

Etiologies associated with PLEDs include a list of acute and subacute processes with stroke, infection,
and tumor at the top of that list. Classically, the finding of PLEDs has a particularly well-known association with herpes simplex virus (HSV) encephalitis (although the finding is not pathognomonic). In HSV encephalitis, PLEDs may either be unilateral or bilateral but independent (BiPLEDs, discussed in the next subsection).

Occasionally, the distinction between PLEDs and seizure activity can be difficult. For instance, when PLEDs are associated with contralateral jerking movements of the body, the diagnosis of epilepsia partialis continua (EPC) is suggested. Unless there is such a contralateral motor response in association with the discharges, the electrical patterns associated with EPC can be indistinguishable from PLEDs. In general, however, PLEDs are not considered to represent an electrographic seizure discharge but rather an interictal pattern. This is borne out by the fact that PLEDs are usually resistant to treatment with antiseizure medications. Rare exceptions to this concept are seen in the form of confusional states associated with PLEDs that have cleared with the administration of antiseizure medications.

PLEDs may be seen less commonly after a prolonged seizure discharge, so-called postictal PLEDs. After the clinical seizure has ceased, periodic afterdischarges identical to PLEDs may be seen over a single hemisphere, eventually burning out with time. Except in unusual cases, PLEDs are a temporary phenomenon and do clear with the passage of time.

**BiPLEDs**

PLEDs that occur over both hemispheres simultaneously but in an asynchronous or independent fashion are termed bilateral periodic lateralized epileptiform discharges (BiPLEDs). The possible causes of BiPLEDs is similar to the causes of PLEDs but favor those processes that are more likely to be bilateral. Therefore, a tumor or a discrete, focal stroke, both of which tend to be unilateral processes, are not expected as causes of BiPLEDs, but anoxic encephalopathy (the equivalent of bilateral, diffuse strokes) is a more common cause. As mentioned earlier, BiPLEDs are particularly characteristic of HSV encephalitis which is usually a bilateral process.

**PSIDDs**

Periodic short interval diffuse discharges (PSIDDs) are periodically occurring waveforms that may consist of sharp waves, spikes, spike-wave discharges, or triphasic waves. As the name implies, the field is spread diffusely over the brain, and the interdischarge interval is 4 seconds or less (see Figure 9-32). The most common clinical association of PSIDDs is anoxic encephalopathy. Each discharge may or may not be associated with a body jerk (myoclonus). In general, the presence of PSIDDs in anoxic encephalopathy suggests a poor neurologic prognosis. PSIDDs may also occur as a seizure phenomenon (see the section on nonconvulsive status epilepticus later in the chapter).
Triphasic Waves

Triphasic waves may be considered a subset of PSIDDs. Triphasic waves are medium- to high-voltage complexes with a characteristic triphasic pattern, often with a sequence of negative–positive–negative deflections, repeating at rate of 1.5 to 2.5 Hz (see Figure 9-33). The first phase of the deflection has been said to resemble a blunted spike and the whole complex may resemble a blunted spike-wave discharge, although usually the initial deflection is too rounded for the complex to be considered a true spike-wave discharge. Triphasic waves usually occur diffusely and symmetrically, often with a frontal predominance. The most frequent clinical association is with hepatic encephalopathy or hepatic coma, but other metabolic derangements (such as renal failure) may also be associated with triphasic waves. Triphasic waves may also show the unusual manifestation of an anterior-to-posterior time lag in which the wave is seen frontally up to 200 ms before it is seen posteriorly.

Nonconvulsive Status Epilepticus

Generalized nonconvulsive status epilepticus (NCSE) may also technically fulfill the criteria for PSIDDs in that this pattern may consist of repetitive, diffuse sharp discharges (see Figure 9-34). Both the EEG appearance and the clinical context may aid in distinguishing between NCSE and other causes of PSIDDs, although at times the distinction is difficult. Because NCSE is an epileptic seizure pattern, its presence requires a different treatment approach aimed at terminating the seizure activity, usually with aggressive use of antiepileptic medications.

Creutzfeldt-Jakob Disease

Creutzfeldt-Jakob disease (CJD) is a rare disorder that consists of a progressive dementia characteristically associated with repetitive myoclonus. The disease may be transmitted by infectious particles called prions. CJD is often associated with the finding of PSIDDs in the form of sharp wave complexes, often with triphasic morphology (see Figure 9-35). The myoclonus typically associated with this disease may occur synchronously with the PSIDDs. The findings of PSIDDs and myoclonus alone cannot establish the diagnosis of CJD. Rather, the distinctive clinical picture of CJD should be considered supported by finding this characteristic pattern in the EEG.

PLIDDs

Periodic long-interval diffuse discharges, or PLIDDs, refer to repetitive waveforms that are separated by intervals of four to 20 seconds’ duration. PLIDDs are the hallmark finding of subacute sclerosing panencephalitis (SSPE), a rare disorder related to slow measles infection of the central nervous system. Measles
An example of triphasic waves is shown in a patient with hepatic failure. The waves are displayed in a referential montage using a reference that consists of the average of both earlobes, A1 and A2. The three separate phases of the triphasic waves ("up," "down," and "up") are labeled "1," "2," and "3," respectively. The characteristic time shift of triphasic waves is highlighted by the gray arrow. Note that the trough of the wave begins earlier in the frontal area and is delayed by several milliseconds in each successively more posterior channel. Although a classic feature of triphasic waves, this back-to-front delay is not seen in all cases of triphasic waves.

immunization has almost completely wiped out this disease in many parts of the world, although a low rate of SSPE persists even in the immunized population; a possible relationship between the slow virus infection and the vaccine is unclear. This fatal disorder begins with mental status changes and behavioral deterioration followed by dementia and myoclonic seizures that correlate with the periodic discharges. Over weeks or months, the disorder inexorably progresses to a vegetative state and death. The presence of such discharges in the appropriate clinical context is considered highly characteristic of SSPE.

Burst suppression patterns related to anoxia or drugs (including pharmacologic agents used in the intensive care unit) may fall into the category of PLIDDS (see Figure 9-36). Because the interval between bursts in burst suppression may be both less than or greater than 4 seconds in duration, burst-suppression patterns may fall into the category of either PLIDDS or PSIDDS. Burst suppression patterns are discussed in more detail in Chapter 12, “EEG Patterns in Stupor and Coma.”

ABNORMAL SLEEP STRUCTURE

Abnormalities of sleep structure fall into a variety of categories, including sleep disorders that are beyond the scope of this text. Abnormal sleep structure, including the absence of normal sleep elements or the abnormal ordering of sleep staging, may be the result of an abnormal central nervous system, a disease process, or a pharmacologic effect.

Occasionally, patients are seen who lack the expected synchrony seen in normally occurring sleep elements. This type of abnormality has been associated with agenesis of the corpus callosum (see Figures 9-37, 9-38, and 9-39). Not all patients with agenesis of the corpus callosum manifest this finding, however.
Figure 9-34 During the episode of nonconvulsive status epilepticus shown in this figure, this 19-month-old girl was motionless and unresponsive. This seizure pattern was eventually terminated with anticonvulsant medications.

Figure 9-35 Periodic short interval diffuse discharges (PSIDDs) ranging from 30 to 50 µV are seen in a patient with Creutzfeld-Jakob disease (dots). Note that the field of the discharge is diffuse, although this patient’s variant of the disorder is posteriorly predominant. (Image courtesy of Dr. Edward Bromfield and Dr. Barbara Dworetzky.)
This burst-suppression pattern consists of 2- to 3-second bursts of mixed activity that includes sharp elements, separated by several seconds of diffuse suppression. Technically, this example falls into the category of periodic long interval diffuse discharges because of the long interburst interval, but the term *burst-suppression* is much more commonly used for this type of pattern.

Both vertex waves (gray arrows) and spindles (blue arrows) are asynchronous in this 3-year-old boy with agenesis of the corpus callosum. Although it seems logical that vertex waves and spindles might not be synchronous in individuals who lack a corpus callosum, some patients with agenesis of the corpus callosum still do manifest vertex wave and spindle synchrony. Likewise, the presence of spindle and vertex wave asynchrony does not always indicate absence of the corpus callosum.
SUGGESTED READINGS