Figure 130.21  Hypnagogic hypersynchrony. This less common but well recognized normal variant of drowsiness is seen in children aged 3 months to 13 years. It consists of paroxysmal bursts (3–5 Hz) of high-voltage (as high as 350 μV) rhythmic waves. Note the normal alpha rhythm in the first two-thirds of the sample, which precedes drowsiness.

Figure 130.22  Stage 2: sleep spindles. This segment depicts typical short bursts (1–3 seconds) of sinusoidal 12–16 Hz central activity that waxes and wanes.
By strict sleep staging criteria on polysomnography, REM sleep is defined by (1) rapid eye movements, (2) muscle atonia, and (3) EEG “desynchronization” (compared to slow-wave sleep). Thus two of the three defining characteristics are not cerebral waves and theoretically require monitoring of eye movements (electro-oculogram (EOG)) and muscle tone (electromyogram (EMG)). Fortunately, muscle activity and eye movements can be evaluated on EEG; thus REM sleep is usually not difficult to identify (Figure 130.25). In addition to the three features already named, “sawtooth” waves also are seen in REM sleep.

- **EEG Desynchronization.** The EEG background activity changes from that seen in slow-wave sleep (stage 3 or 4) to faster and lower voltage activity (theta and beta), resembling wakefulness.

- **Rapid Eye Movements.** These are saccadic, predominantly horizontal, and occur in repetitive bursts.

- Despite the lack of a dedicated EMG channel, the muscle atonia that characterizes REM sleep is usually apparent as a general sense of “quiet” muscle activity compared to wakefulness.

- Sawtooth waves are a special type of central theta activity that has a notched morphology, resembling the blade of a saw, and usually occurs close to rapid eye movements (i.e., phasic REM). They are only rarely identifiable on EEG.

The duration of REM sleep increases progressively with each cycle and tends to predominate late in the sleep period into early morning.
Less Common Patterns and Normal Variants

Just like human anatomy, sizes and shapes vary somewhat among individuals, so too do brain waves. There is a wide range of variability, and it is important to read EEG “conservatively” and avoid overreading normal variants [9]. There are many normal variants that have been well described as benign and have no association with epilepsy. These include small sharp spikes (also called benign epileptiform transients of sleep, Figure 130.26), wicket spikes (Figure 130.27), 14 and 6 Hz positive spikes, phantom spike waves, rhythmic midtemporal theta of drowsiness (also called psychomotor variant), and subclinical rhythmic epileptiform discharges of adults. These can be overread as abnormal. Lambda waves (Figure 130.28) are occipital sharp transients that resemble POSTS but occur in wakefulness when subjects (especially children) scan the environment. Despite these, the most commonly overread patterns are “nameless” fluctuations of background activity [9].

THE ABNORMAL EEG

Like most neurophysiologic tests, EEG is a test of cerebral function, and as such is for the most part nonspecific as to etiology. Although earlier investigators have attempted to identify the reliability of EEG in differentiating types of lesions, this has clearly become a senseless and futile exer-
cise in the modern era of neuroimaging [10]. The exercise
to describe EEG abnormalities by pathology (stroke,
abscess, tumor, etc.), which was common in old EEG
texts [11], is clearly obsolete and will not be followed
here.

There are many different ways to classify EEG abnorm-
alities. This chapter will use a very practical classi-

cification developed at the Cleveland Clinic Foundation [12] and used
at many centers. The outline of the classification is shown
in Table 130.1.

Epileptiform Abnormalities

Types of Epileptiform Abnormalities EEG is useful in
epilepsy because it is the only test that gives direct evidence
for epileptogenicity. Most commonly, clinicians have to
rely on interictal abnormalities. Interictal epileptiform
abnormalities include spikes, sharp waves, and spike–
wave complexes. In addition to type and morphology, location
is very important and is typically either focal or gener-
ized. Generalized epileptiform discharges (i.e., spikes,
sharp waves, polyspikes, spike–wave complexes), as seen
in the primary generalized epilepsies, are usually maximal
in the frontal regions, with typical “phase reversals” at the
F3 and F4 electrodes or less commonly at F7 and F8.

- **Spikes and sharp waves** are sharp transients that have a
very strong association with epilepsy. The two are
distinguished only by their duration (spikes <70 ms,
sharp waves 70–200 ms), but they have no differences
in terms of clinical significance. Several characteris-

tics help distinguish these from benign epileptiform
variants but this distinction can at times be difficult.
Helpful features that indicate pathologic discharges
include high amplitude, which makes them “stand
out” from ongoing background activity, and after-
going slow waves, which indicate “disruption” of
background activity (Figures 130.29 and 130.30).
The terms “spikes” and “sharp waves” should be
reserved for these abnormalities thought to be pathol-
ogical and indicative of epileptogenicity. If one wants to
use purely descriptive (uncommitted) terms, then
“generic” phrases should be used, such as “sharp
transients” or “sharply contoured waveforms.”

Figure 130.25 REM sleep. This segment shows rapid eye movements (3rd and 10th seconds). Note also the sawtooth waves: notched theta (~5 Hz) transients in the central regions.
Figure 130.26 Small sharp spikes (SSS) or benign epileptiform transients of sleep (BETS). Typical brief (<50 ms) and low-amplitude (<50 μV) sharp transients in the temporal region, typically in stage 1 or 2 sleep.

Figure 130.27 Wicket spikes. These are typically sharp transients seen in the temporal regions during wakefulness or stage 1 sleep. They have a symmetric upgoing and downgoing phase, typically arise from an ongoing background activity, and do not disrupt the background (i.e., no after-going slow wave). They are basically fluctuations of background activity.
TABLE 130.1 Cleveland Clinic EEG Classification

<table>
<thead>
<tr>
<th>Slow activity</th>
<th>Special patterns</th>
</tr>
</thead>
<tbody>
<tr>
<td>Background slow (BS)</td>
<td>Asymmetry</td>
</tr>
<tr>
<td>Intermittent slow (IS)\a</td>
<td>Excessive fast</td>
</tr>
<tr>
<td>Intermittent rhythmic slow (IRS)\a</td>
<td>Sleep-onset REM</td>
</tr>
<tr>
<td>Continuous slow (CS)\a</td>
<td>Periodic pattern</td>
</tr>
<tr>
<td>Epileptiform patterns\a</td>
<td>Triphasic waves</td>
</tr>
<tr>
<td>Spikes</td>
<td>PLEDs\a</td>
</tr>
<tr>
<td>Sharp waves</td>
<td>Background suppression\a</td>
</tr>
<tr>
<td>Benign epileptiform discharges of childhood (BEDC)</td>
<td>Special patterns used only in coma</td>
</tr>
<tr>
<td>Spike–wave complexes (SWCs)</td>
<td>Alpha coma</td>
</tr>
<tr>
<td>Slow spike–wave complexes (SSWCs)</td>
<td>Spindle coma</td>
</tr>
<tr>
<td>3 Hz spike–wave complexes (3 Hz SWCs)</td>
<td>Beta coma</td>
</tr>
<tr>
<td>Polyspikes</td>
<td>Theta coma</td>
</tr>
<tr>
<td>Hypsarrhythmia</td>
<td>Delta coma</td>
</tr>
<tr>
<td>Photoparoxysmal response (PPR)</td>
<td>ECI</td>
</tr>
<tr>
<td>EEG seizure pattern</td>
<td></td>
</tr>
<tr>
<td>Artifact obscured EEG</td>
<td></td>
</tr>
</tbody>
</table>

\a Abnormalities that require localization:
- Generalized
- Laterialized
- Multiregional
- Regional
- Multifocal
- Focal

Source: From [12].
Polyspikes are usually generalized (rarely focal), although focal spikes can at times have a multiphasic “polyspike-like” morphology. Polyspikes are multiple repetitive spikes occurring at about 20 Hz and are typically seen in the primary generalized epilepsy with myoclonic seizures (Figure 130.31).

Spike–wave complexes (SWCs) are the repetitive occurrence of a spike followed by a slow-wave. Since any significant spike or sharp wave usually is followed by a slow wave (see earlier), a run of 3 seconds is required to classify a record as SWC, as opposed to the categories already mentioned (spike or sharp wave). SWCs can be divided further into two more specific types, as follows:

3 Hz SWC. This pattern is characterized by a frequency of 2.5–4 Hz and a very monomorphic (“perfectly regular”) morphology (Figure 130.32).

It occurs in very discrete bursts, and between bursts the EEG is normal.

Slow SWC. This pattern is not only slower (<2.5 Hz) but also more irregular (less monomorphic) than the 3 Hz SWC. Bursts are less discrete than the 3 Hz SWC, and between bursts other abnormalities are seen in symptomatic/cryptogenic epilepsies of the Lennox–Gastaut type.

Hypsarrhythmia. This is characterized by continuous (during wakefulness) high-amplitude >200 μV (microvolts) generalized and polymorphic slowing with no organized background, and multifocal spikes (Figure 130.33). During NREM sleep, the pattern becomes discontinuous and fragmented, resembling a burst-suppression or pseudoperiodic pattern, while it tends to disappear in REM sleep.

- Electrographic Seizures (Ictal Patterns)
Focal seizures are discharges characterized by rhythmicity and evolution ("buildup") in frequency, amplitude, and distribution. The discharge can consist of rhythmic theta or delta activity, or repetitive spikes or sharp waves, but the most characteristic features of electrographic focal seizures are rhythmicity and evolution (Figure 130.34). Generalized seizures are also characterized by rhythmic discharges that evolve. They can be variable and there are rhythmic or periodic abnormalities that are not ictal (see later under status epilepticus).

Spike–wave complexes are both an interictal and an ictal pattern. The distinction is arbitrary and based on duration and the presence of detectable clinical symptoms (i.e., alteration of awareness). Electrodecrement consists of abrupt attenuation ("flattening") of background activity, often preceded by a high-amplitude transient, which can be sharply contoured or broad, or generalized paroxysmal fast activity (GPFA). This typically is associated with infantile spasms and tonic or atonic seizures (Figure 130.35).
Epileptiform discharges have to be differentiated from normal patterns and normal variants that can look like epileptiform, and this can at times be difficult. The overinterpretation of EEG is a common and underreported problem [9, 13, 14] and a major cause for the misdiagnosis of epilepsy. If read conservatively (being careful not to overinterpret normals), the specificity of EEG for epilepsy is very high, that is, greater than 90%. It is generally accepted that less than 3% of the general population has interictal epileptiform abnormalities, with a slightly higher percentage in children [15] (largely because of benign focal epilepsies in that age group, see later). Because of the relatively frequent misinterpretation (overreading) of benign EEG recordings, it is unfortunately true that “routine interictal EEG is one of the most abused investigations in clinical medicine and is unquestionably responsible for great human suffering” [16]. One of the important remedies to this pitfall is that when in doubt, one should err on the side of underreading. Clinical experience strongly supports the view that less harm will be done by underinterpreting an abnormality than by overinterpreting a normal pattern.

**Electrographic Status Epilepticus** Status epilepticus (SE) is typically an obvious clinical diagnosis, but in some situations an EEG is required to diagnose “nonobvious” SE. Electrographically, SE can take the form of either repetitive discrete seizures or, more commonly, a continued pattern of rhythmic or periodic discharges such as generalized periodic epileptiform discharges (GPEDs) [17, 18]. Unfortunately this pattern (Figure 130.36) is not specific for electrographic SE and can be seen in severe metabolic encephalopathies such as anoxic, uremic, or hepatic disturbances [19, 20, 21]. Thus the final answer as to whether it does or does not represent SE in a given patient often will depend on the clinical circumstances and the response to treatment [21].
Figure 130.32  3 Hz spike–wave complexes. This shows typical 3 Hz spike–wave complexes in a patient with an idiopathic (“primary”) generalized epilepsy. This patient likely has absence seizures. If tested with a clicker, a discharge of this duration (~4.5 seconds) is likely associated with a brief impairment of awareness. Classification: 3 Hz spike–wave complexes, generalized.

Figure 130.33  Hyparrhythmia. These recordings are from two different patients. Note on both the lack of any reactive background, the very high-amplitude polymorphic delta activity, and the multifocal spikes. Classification: hyparrhythmia.
Figure 130.34  Focal seizure. Left temporal seizure (singular), showing the typical rhythmic activity that evolves (build up). Classification: EEG seizure, regional left temporal.

Figure 130.35  Generalized paroxysmal fast activity followed by attenuation (electrodecrement) in a patient with a symptomatic generalized epilepsy of the Lennox–Gastaut type. This could be an interictal (asymptomatic) discharge but could also be an ictal pattern associated with a tonic or atonic seizure. Classification: EEG seizure, generalized.
EEG-Video Monitoring and the Differential Diagnosis of Epilepsy

Prolonged EEG-video monitoring is indicated when seizures do not respond to medications [4]. About 20–30% of patients referred for refractory seizures do not have epilepsy but have psychogenic nonepileptic seizures (PNESs) instead [3, 13]. A small percentage have syncope [14, 22]. Occasionally other paroxysmal conditions can be misdiagnosed as epilepsy, but PNESs are by far the most common condition, followed by syncope. Unfortunately, the current average delay in the diagnosis of PNESs is over 7–9 years and 80% of patients with PNESs have received antiepileptic drugs [23]. This means that EEG-video monitoring is probably underutilized and the “established” diagnosis of seizures is not verified often enough. Once the diagnosis of PNESs is suspected clinically [7], it is usually easily confirmed by EEG-video monitoring, which can even be performed as an outpatient. In the hands of experienced epileptologists, the combined electroclinical analysis of both the clinical semiology of the “ictus” and the ictal EEG findings allows a definitive diagnosis in nearly all cases. The second reason why medications may fail is that 30% of epilepsies are intractable and require nonpharmacologic treatments [4].

Encephalopathic Patterns

These patterns are associated with diffuse (generalized) brain dysfunction, that is, diffuse encephalopathies. Again, in general, they are completely nonspecific in terms of etiology. In order of increasing severity, they include:

- **Background Slowing (Classification as “Background slow”).** There is a posterior dominant background that is reactive, but the frequency is too slow for age (<8 Hz after the age of 8) (Figure 130.37).
- **Intermittent Generalized Slowing (Classification as “Intermittent Slow, Generalized”).** There are intermittent bursts of generalized slowing, in the theta or (more commonly) delta range (Figure 130.38), which are not attributable to normal circumstances such as hyperventilation or sleep. A subtype of intermittent generalized slowing is intermittent rhythmic slowing, which is usually frontally predominant and thus often referred as “FIRDA.”
- **Continuous Generalized Slowing (Classification as “Continuous Slow, Generalized”).** Here the slowing is continuous or nearly continuous (>80% of the...
Figure 130.37  Background slowing. Note that there is a posterior dominant background, which is even normally reactive (attenuation with eye opening in the last 2 seconds). However, the frequency is only 6 Hz, which is too slow (for a subject older than 3 years) and thus is evidence for a mild diffuse encephalopathy. Classification: background slow.

Figure 130.38  Intermittent generalized slowing. Note the brief (~4 sec) burst of polymorphic delta (~2 Hz) activity. When this is intermittent or reactive, it is indicative of a mild to moderate diffuse encephalopathy. Classification: intermittent slow, generalized.
record) and is unreactive. Unreactive means that it does not change with external stimulation, and that there are no state changes such as evidence for drowsiness, sleep, or alerting responses (Figure 130.39).

- Periodic patterns are generalized discharges that occur with periodicity (i.e., at regular intervals) and are often referred to as generalized periodic epileptiform discharges (GPEDs) (Figure 130.35). The discharges are often sharply contoured, and the periodicity is most often 1–3 seconds. The etiology is diverse and includes nonconvulsive status epilepticus [17, 18] as well as severe metabolic encephalopathies such as anoxic, uremic, or hepatic disturbances [19, 20, 21]. Another cause of generalized periodic pattern is Creutzfeldt–Jakob disease.

Burst-suppression (Figure 130.40) is a subtype of periodic patterns where the activity between complexes is suppressed (i.e., <10 μV). This is indicative of an extremely severe degree of encephalopathy, which in fact immediately precedes electrocerebral inactivity. It is typically caused by either drugs (e.g., anesthetic agents), in which case it is reversible, or anoxia.

Triphasic waves (Figure 130.41) can also be viewed as a subtype of periodic patterns. These are high-amplitude surface-positive sharp transients preceded and followed by lower amplitude negative components (thus “triphasic”) and occur semi-periodically at 1–3 seconds. They are typically seen in metabolic encephalopathies, especially hepatic or renal.

- Background suppression is the absence of any cerebral activity greater than 10 μV, including with attempts at activating/stimulating the patient (unreactive). This is often used for severely “flat” EEG recordings that do not meet the criteria for electrocerebral inactivity (see later).

- Electrocerebral inactivity (ECI) (Figure 130.42) is the EEG pattern of brain death. The criteria are precisely defined [24], but it should be remembered that, at least in the United States, brain death is a clinical diagnosis and EEG is a supportive test (i.e., not required) for the diagnosis. The main criteria for ECI include no

**Figure 130.39** Continuous generalized slowing. Note the polymorphic delta (~2 Hz) activity. When this is continuous (>80% of the record) and unreactive, it is indicative of a severe diffuse encephalopathy. Classification: continuous slow, generalized.
Figure 130.40  Burst-suppression. This sample depicts the typical periods of suppression (activity $< 10 \mu V$), which in this example last 3–5 seconds, interrupted by "bursts" of activity. Classification: burst-suppression.

Figure 130.41  Triphasic waves. Note the quasiperiodicity, triphasic morphology with a prominent frontal (anterior) positivity, and the (mild) anteroposterior lag shown by the vertical bold line. Classification: triphasic waves.
activity greater than 2 μV with double interelectrode distances, complete lack of reactivity, and exclusion of confounding factors such as sedative drugs and hypothermia.

Patterns of Focal Abnormalities

**Slow Activity** Abnormal slow activity is by far the most common EEG manifestation of focal brain dysfunction. Like generalized slow activity, focal slow activity can be either intermittent (classification as “Intermittent slow, regional ___”) or continuous (classification as “Continuous slow, regional ___”). Continuous focal slowing is typically in the delta range and polymorphic and is also known as polymorphic delta activity (Figure 130.43). This is a very strong finding, highly associated with the presence of a structural lesion (but completely nonspecific as to the nature of the lesion). Continuous focal slow activity is the only nonepileptiform abnormality that can unequivocally be interpreted as abnormal as an isolated finding. By contrast, intermittent focal slowing (Figure 130.44) is a weak and nonspecific finding, which can even be normal (e.g., “temporal slowing of the elderly”).

As outlined above, focal slowing is completely nonspecific as to etiology, and in the era of imaging the EEG has no role in diagnosing the nature of a lesion. Focal slowing is the most common abnormality associated with focal lesions of any type, including (but not limited to) neoplastic, vascular, subdural collections, traumatic, and infectious [10].

**Amplitude Asymmetry** In this discussion, the term asymmetry refers to an asymmetry of amplitude and refers to normal rhythms. By contrast, a focal frequency asymmetry would be classified as focal slow.

Destructive lesions can attenuate normal rhythms. However, normal rhythms are never perfectly symmetric in amplitude, so when to consider asymmetries significant is not always clear. A good rule of thumb is that, with very few exceptions, significant focal asymmetries will be associated with slowing. In general, as with other types of focal EEG abnormalities such as slowing, asymmetry is nonspecific as to etiology.

Although asymmetry in amplitude is usually indicative of dysfunction on the side of depressed amplitude, one very common exception to this rule is the so-called breach rhythm [25] (Figure 130.45). This is caused by a skull

Figure 130.42  Electroencephalogram (EEG) showing electrocerebral inactivity. There is no cerebral activity greater than 2 μV. Because this is recorded at a sensitivity of 2 μV/mm, there is prominent EKG and 60 Hz artifact. Classification: electrocerebral inactivity.
Intermittent lateralized slowing in the left hemisphere. Note the brief (~2 second) burst of delta (~2 Hz) activity over the left hemisphere. This type of focal slowing, unlike that showed in Figure 130.42, is indicative of mild dysfunction and is a “weak” (nonspecific) abnormality. Classification: intermittent slow, lateralized left hemisphere.

Continuous lateralized slowing in the right hemisphere. Note the clear polymorphic delta (~3 Hz) activity over the right hemisphere. Such focal slow activity, when persistent (>80% of the record), is strongly associated with a structural lesion. Classification: continuous slow, lateralized right hemisphere.

Intermittent lateralized slowing in the left hemisphere. Note the brief (~2 second) burst of delta (~2 Hz) activity over the left hemisphere. This type of focal slowing, unlike that showed in Figure 130.42, is indicative of mild dysfunction and is a “weak” (nonspecific) abnormality. Classification: intermittent slow, lateralized left hemisphere.
Figure 130.45 Breach rhythm. These are two different examples in two patients who are status post craniotomy. **Left sample:** Note the increase in amplitude very focally at C3, of what is likely a mu rhythm. Classification: asymmetry, increased mu, left central. **Right sample:** Here the increase is in fast (beta) activity at C4. Classification: asymmetry, increased beta, right central. The increased amplitude should not be mistaken for sharp waves.

Figure 130.46 PLEDs—periodic lateralized epileptiform discharges. These discharges occur periodically, here with a periodicity of 1–1.5 seconds, and are lateralized (here in the right hemisphere). Note that the discharges look to be of low amplitude but in reality are ~100 μV (see scale). Classification: PLEDs, lateralized right hemisphere.
defect, which attenuates the high-frequency filter function of the intact skull. As a result, faster frequencies (alpha, spindles, beta) are of higher amplitude on the side of the defect. Since morphology is often sharply contoured, determining the epileptogenicity of these discharges can be extremely difficult, and in this situation one should probably err on the conservative side by not interpreting them as epileptiform. Finally, it should be kept in mind that amplitude asymmetries are best interpreted on referential montages, since amplitude is highly dependent on interelectrode distances.

**PLEDs** Periodic lateralized epileptiform discharges (PLEDs) (Figure 130.46) are a special type of focal abnormality. As implied by their name, they are periodic, lateralized, and often epileptiform in morphology. Periodicity is the most characteristic feature, and the one that sets PLEDs apart from other focal abnormalities. Periodicity refers to a relatively constant interval between two discharges and varies between 0.5 and 3 seconds, most often around 1 second. The epileptiform morphology of the discharges is not invariable, as PLEDs are often closer to slow waves than to sharp waves in morphology. PLEDs are caused by acute destructive focal lesions and are a transitory phenomenon: they tend to disappear in weeks, even if the causal lesion persists. With time, periodicity and duration increase, and the record takes on a less specific focal slow appearance that is more likely to persist. By far the most common etiology is an acute cerebrovascular event, followed by focal encephalitis such as herpes [26–28]. Although the periodic patterns of Creutzfeldt–Jakob disease are usually generalized and bisynchronous, occasionally, especially early in the course, they may be unilateral or markedly asymmetric and thus take on the appearance of PLEDs [29].

As implied by their name, PLEDs have a high association with clinical seizures, and on average about 80% of patients with PLEDs will have clinical seizures. In clinical practice, PLEDs are usually managed as interictal discharges (spikes or sharp waves). They indicate a high risk of focal seizures but are usually not treated with antiepileptic drugs unless there is clinical evidence for seizures, although there is some controversy about this [10].

**REFERENCES**


