The EEG in meningitis shows various degrees of slow-wave abnormalities, depending on the type of meningitis and the degree of involvement of the central nervous system.

Moderate to severe diffuse slow-wave abnormalities are often present in acute purulent meningitis, and paroxysmal epileptiform activity may be present in those patients who have seizures.

The electroencephalographic findings in tuberculous meningitis vary according to the location of the inflammatory process. In basal meningitis, the EEG may be normal and show only mild nonspecific slowing. When the inflammatory process involves the cortical meninges, moderate to severe slowing occurs, depending on the degree of cortical involvement, the rate of progression of the disease process, the level of consciousness, the presence of metabolic or systemic factors, the pulmonary state of the patient, and the effects of medication. As with purulent meningitis, more severe slow-wave abnormalities are present in children, with the slowing often being maximal over the posterior head regions.

In aseptic meningitis, the EEG may be normal or show only mild slowing; the electroencephalographic findings may not necessarily correlate with the clinical severity of the inflammatory process or the development or degree of post-infectious sequelae.

Patients in whom meningoencephalitis develops in association with infectious mononucleosis may have mild to moderate diffuse or focal slow-wave abnormalities that may or may not coincide with the area of maximal neurologic dysfunction. On occasion, focal epileptiform abnormalities have been observed in patients who experience seizures.

The rate and degree of the improvement in the electroencephalographic abnormalities after treatment have some diagnostic and prognostic value. One of the characteristic features of meningococcal meningitis is the rapid improvement in the electroencephalographic findings, with the findings often returning to normal within 1 or 2 wk after treatment. In other types of purulent meningitis and tuberculous meningitis, the electroencephalographic abnormalities often require several weeks to resolve.

The EEG usually returns to normal in patients with uncomplicated meningitis; however, persistent electroencephalographic abnormalities or evidence of deterioration in the EEG suggests an unfavorable course, the development of a complication such as an abscess or hydrocephalus, or the presence of residual brain damage.

Although the electroencephalographic findings are not essential for making the specific diagnosis of meningitis, the EEG and particularly serial recordings are helpful in following the course of the disease, detecting the development of complications or relapse, and indicating the presence of sequelae or residual brain damage.

The electroencephalographic findings in encephalitis are similar to those in meningitis, although the abnormalities are often more severe; this may be a helpful point in the differential diagnosis.

The EEG is almost always abnormal during the acute phase of encephalitis, with the most frequent finding being the presence of diffuse high-voltage, arrhythmic and/or rhythmic delta slowing. Diffuse polymorphic arrhythmic delta activity is more likely to occur when the white matter is involved, whereas paroxysmal, bisynchronous slow-wave activity is more likely to be present when the disease process involves the subcortical gray matter. The degree of slowing depends on the severity of the infection, the amount of cerebral involvement, the level of consciousness, and other associated systemic or metabolic factors. In general, the leukoencephalitides, which primarily involve the white matter and which are caused by the group B non-neutrotropic viruses (measles, rubella, variola) and the post-vaccinal states, are associated with more severe electroencephalographic abnormalities than are those caused by the group A neutrotropic viruses (mumps, St. Louis and equine encephalitis). Children often show more severe electroencephalographic abnormalities than do adults. Epileptiform abnormalities also may be present, particularly if the patient is having seizures.

Slow-wave abnormalities have also been observed during the acute stages of uncomplicated childhood infections, such as measles, mumps, rubella, chickenpox, and scarlet fever, in which there is no overt evidence of nervous system involvement. The electroencephalographic
Brain abscesses may occur as a result of meningitis, septicemia, or septic emboli or as an extension of an infectious process involving the ears, mastoids, and sinuses.

In the early stages of an acute supratentorial abscess, the EEG may show diffuse slowing with a poorly defined focus. This pattern is more likely to occur with meningo-encephalitis, if the patient is obtunded, and when the more focal abnormalities are obscured by more generalized slow-wave abnormalities. Focal slowing becomes more apparent as the suppurative process becomes localized; marked focal polymorphic delta slowing can develop overlying the site of the abscess, particularly if the lesion is located close to the surface of the brain. If there are multiple abscesses, multiple electroencephalographic foci may be present. More generalized, intermittent, or shifting bursts of rhythmic slow waves (that is, a projected rhythm) also may be present; these bursts may be seen with a disturbance of the frontal lobe or as a secondary effect of the mass lesion on midline structures. On infrequent occasions, focal or lateralized periodic sharp- or slow-wave complexes (periodic lateralized epileptiform discharges) may be present over the involved area of the brain.

In general, the degree of the electroencephalographic abnormalities reflects the severity of the inflammatory process. The electroencephalographic abnormalities occur most frequently with measles infection, in which moderate to severe slow-wave abnormalities may be present as early as 1 to 4 days before the rash appears, reaching a maximum on the first day of the rash and then subsiding during the next 8 to 10 days. Transient slow-wave abnormalities also have been observed over the posterior head regions after measles vaccination.

The electroencephalographic abnormalities usually diminish in association with clinical improvement, but on occasion the electroencephalographic changes lag behind the clinical findings. However, persistent or increased abnormalities, particularly if they are focal, are associated with an increased likelihood of brain damage or post-encephalitic epilepsy. A return to a normal electroencephalographic pattern does not preclude residual brain damage.

Diffuse slow-wave abnormalities with, at times, more focal features and epileptiform activity may be seen in California encephalitis and there is a fairly good correlation between the EEG and clinical findings, both during the acute stages of the disease and on follow-up examinations.

The entero-encephalitides caused by Coxsackie and ECHO viruses, which predominantly affect infants and young children, are accompanied by varying degrees of diffuse slow-wave abnormalities in the EEG. Western equine, Eastern equine, St. Louis, and Japanese encephalitis are also associated with variable slow-wave abnormalities in the EEG, which may or may not show a correlation with the clinical picture.

In tick-borne viral encephalitis (spring-summer encephalitis), slow-wave abnormalities may be present prior to the onset of symptoms. The abnormalities do not necessarily correspond with the clinical symptoms and severity of the infection; slow-wave abnormalities, however, may continue to be present in those patients with post-encephalitic symptoms. EEG recordings have only rarely been done in rabies. They have been described as showing a depression or "extreme desynchronization" in one case, and nonspecific findings in two other cases (Gastaut and Miletto, 1955; Radermecker, 1977). Diffuse slow-wave abnormalities similar to those seen in other post-vaccinal states may be present following rabies vaccination.

The EEG recordings in the rickettsial infections (Eurasian typhus or spotted fever, Rocky Mountain spotted fever, tsutsugamushi fever) range from normal to those showing diffuse or focal slow-wave abnormalities, with epileptiform activity being present in those patients who develop seizures. The degree of EEG abnormality usually reflects the degree of encephalitic involvement.

Encephalitis or meningitis caused by fungal diseases (histoplasmosis, blastomycosis, and coccidioidomycosis) are associated with diffuse slow-wave abnormalities in the EEG. These changes are similar to those produced by bacterial and viral agents. More focal EEG abnormalities may be present if there is focal cerebral involvement by mycotic abscesses. As fungal infections tend to recur, the EEG may be helpful in following the clinical course of the patient and alerting one to a recurrence of the infection or the development of complications. As a rule, most of the different types of encephalitis do not give rise to specific types of EEG patterns. Instead, the EEG abnormalities are most often expressed as diffuse or focal slow-wave abnormalities, with the degree and extent of the slowing reflecting the intensity of parenchymal involvement.

**EEG IN BRAIN ABSCESS**

Brain abscesses may occur as a result of meningitis, septicemia, or septic emboli or as an extension of an infectious process involving the ears, mastoids, and sinuses.

In the early stages of an acute supratentorial abscess, the EEG may show diffuse slowing with a poorly defined focus. This pattern is more likely to occur with meningo-encephalitis, if the patient is obtunded, and when the more focal abnormalities are obscured by more generalized slow-wave abnormalities. Focal slowing becomes more apparent as the suppurative process becomes localized; marked focal polymorphic delta slowing can develop overlying the site of the abscess, particularly if the lesion is located close to the surface of the brain. If there are multiple abscesses, multiple electroencephalographic foci may be present. More generalized, intermittent, or shifting bursts of rhythmic slow waves (that is, a projected rhythm) also may be present; these bursts may be seen with a disturbance of the frontal lobe or as a secondary effect of the mass lesion on midline structures. On infrequent occasions, focal or lateralized periodic sharp- or slow-wave complexes (periodic lateralized epileptiform discharges) may be present over the involved area of the brain.

In general, the degree of the electroencephalographic abnormalities reflects the severity of the inflammatory process. The electroencephalographic
The EEG often shows a characteristic pattern and temporal evolution which can be of great value in making the diagnosis of herpes simplex encephalitis, especially when serial recordings are obtained. During the earlier stages of the disease process, the background activity is disorganized and polymorphic delta activity develops in a focal or lateralized fashion, with a predominance over the involved temporal region. Soon after this, focal or lateralized sharp or slow-wave complexes appear, usually having a maximal expression over the involved temporal region. These complexes rapidly evolve into a periodic pattern, with the sharp waves having a stereotyped appearance and recurring every 1 to 3 seconds. The periodic pattern is usually seen between 2 and 5 days after the onset of the illness but, on occasion, has been observed up to 24 and 30 days after the onset of the illness. If there is bilateral involvement of the brain, bilateral periodic complexes may be present, occurring synchronously or independently over the two hemispheres but often having a time locked relationship with one another. Focal or lateralized electrographic seizure discharges, consisting of repetitive sharp or slow waves or spike or polyspike bursts, may be present over the involved area or hemisphere. During this time, there is a transient obliteration of the periodic discharges on the side of the seizure discharges. In the later stages of a fatal herpes simplex infection, the EEG assumes an almost isoelectric appearance.

In nonfatal herpes simplex encephalitis, the periodic complexes disappear as the disease process resolves and are replaced by focal or lateralized slow-wave abnormalities or attenuation of activity over the involved area. The resolution of the electroencephalographic abnormalities often lags behind changes seen with an acute focal supratentorial abscess. Infratentorial abscesses produce less severe slow-wave abnormalities, and at times there may be little or no change on the EEG. When present, the slow-wave abnormalities usually consist of bilaterally synchronous or shifting groups of intermittent rhythmic slow waves.

Chronic abscesses develop more slowly and insidiously and often without overt clinical signs of the infectious process. These are usually well-encapsulated abscesses that develop after the initial infection has been cured. A chronic abscess behaves like a progressive mass lesion and shows the same type of electroencephalographic findings as a tumor (that is, focal slow-wave abnormalities, asymmetry, or attenuation of the background activity) and, if there is increased intraventricular pressure, a projected rhythm. If the abscess develops very slowly, only minor or subtle electroencephalographic changes may be present.

After treatment, the slow-wave abnormalities improve; however, the EEG rarely returns to normal. If surgical intervention is employed, the postoperative electroencephalograms show a rapid decrease in the degree of slow-wave abnormalities within the first few days after surgery; however, some slowing and asymmetry of activity often continue to be present over the surgical area. Epileptiform abnormalities are not very common in the acute stages of the abscess, however, about 75% of patients with cerebral abscesses subsequently suffer seizures, and those patients in whom the amount of epileptiform activity increases within the first 1 to 5 years have a greater tendency of developing subsequent seizures.

### EEG IN HERPES SIMPLEX ENCEPHALITIS

The EEG often shows a characteristic pattern and temporal evolution which can be of great value in making the diagnosis of herpes simplex encephalitis, especially when serial recordings are obtained. During the earlier stages of the disease process, the background activity is disorganized and polymorphic delta activity develops in a focal or lateralized fashion, with a predominance over the involved temporal region. Soon after this, focal or lateralized sharp or slow-wave complexes appear, usually having a maximal expression over the involved temporal region. These complexes rapidly evolve into a periodic pattern, with the sharp waves having a stereotyped appearance and recurring every 1 to 3 seconds. The periodic pattern is usually seen between 2 and 5 days after the onset of the illness but, on occasion, has been observed up to 24 and 30 days after the onset of the illness. If there is bilateral involvement of the brain, bilateral periodic complexes may be present, occurring synchronously or independently over the two hemispheres but often having a time locked relationship with one another. Focal or lateralized electrographic seizure discharges, consisting of repetitive sharp or slow waves or spike or polyspike bursts, may be present over the involved area or hemisphere.

Figure 2. EEG showing focal delta slowing over the right frontal region in a 9-yr-old boy with a right frontal abscess.

Figure 3. EEG of 68-yr-old man with herpes simplex encephalitis. Periodic sharp waves over the left hemisphere.

During this time, there is a transient obliteration of the periodic discharges on the side of the seizure discharges. In the later stages of a fatal herpes simplex infection, the electrographic seizure discharges may occur in association with the periodic discharges without altering them. Additionally, during the later stages of the disease process, the periodic complexes often have a more broad slow-wave appearance and a longer interburst interval. During the final stages of a fatal infection, the EEG assumes an almost isoelectric appearance.

In nonfatal herpes simplex encephalitis, the periodic complexes disappear as the disease process resolves and are replaced by focal or lateralized slow-wave abnormalities or attenuation of activity over the involved area. The resolution of the electroencephalographic abnormalities often lags behind changes seen with an acute focal supratentorial abscess.
complexes in association with a febrile illness and a rapid evolution of neurologic signs is strongly suggestive of herpes simplex encephalitis. Although the findings in herpes simplex encephalitis are not pathognomonic for the disease, the presence of unilateral or bilateral periodic complexes in association with a febrile illness and a rapid evolution of neurologic signs is strongly suggestive of herpes simplex encephalitis.

EEG IN SUBACUTE SCLEROSING PANENCEPHALITIS (SSPE)

SSPE is an inflammatory disease occurring in children and adolescents, believed to be caused by the measles virus and which is characterized by abnormal movements, a progressive intellectual deterioration, and a diagnostic electroencephalographic pattern. The characteristic electroencephalographic pattern consists of high-voltage (300 to 1500 mv) repetitive polyphasic and sharp- and slow-wave complexes ranging from 0.5 to 2 sec in duration, usually recurring every 4 to 15 sec. On rare occasions, these complexes may occur at intervals ranging up to 1 to 5 min. The periodic complexes may be present at any stage of the disease, but they usually are seen during the intermediate stages. Although the form and appearance of the periodic complexes is fairly constant and stereotyped in a single recording, the shape of the complexes varies in different patients and can change from time to time in the same patient at different stages of the disease process. The complexes are usually generalized and asynchronous, but at times they may be asymmetric, have a time lag from side to side or front to back, or occur in a more lateralized or focal fashion, particularly in the earlier stages of the disease.

Initially, the complexes may occur at irregular intervals, but, once established, the complexes recur at regular intervals, although the repetition rate may vary during the course of the disease. Afferent stimuli do not usually affect the periodic complexes; however, on rare occasions, the complexes can be evoked by external stimuli. This occurs when the complexes are present in an inconstant manner, either when they first make their appearance or toward the end of the period of remission. Once the regular pattern of the complexes has been established, however, the complexes are no longer influenced by external stimuli. Drugs usually have little effect on the periodic complexes, although one report described the occurrence of periodic pattern after an intravenous injection of diazepam.

A prominent feature of SSPE is the stereotyped motor jerks or spasms occurring with the periodic complexes. The movements are often described as myoclonic jerks; however, they do not have the momentary lightning-quick nature of true myoclonus; instead, the movements consist of an initial "shock-like abruptness," followed by a momentary arrest of the movement, and then a gradually melting away to the position of rest. On less frequent occasions, the periodic complexes may be associated with an inhibitory phenomenon such as an arrest of movement, loss of tone, or drop attacks. The abnormal movements usually become evident about the same time that the periodic complexes appear on the EEG, however, on occasion, and particularly in the early stages of the disease, the periodic complexes may be present without the associated motor movements. On the other hand, the presence of the MOTOR jerks in the absence of the periodic complexes is uncommon. The motor movements often disappear during sleep, despite a persistence of the periodic complexes. Certain drugs, such as diazepam, may reduce or abolish the motor movements without altering the electroencephalographic complexes.

The resting EEG may be relatively normal when the complexes first appear. As the disease evolves, however, the EEG shows various changes, consisting of slowing and disorganization of the background, an asymmetry of the background activity, or both. These changes are followed by an increase in the slow-wave abnormalities, usually occurring in a diffuse manner but at times having a focal or lateralized emphasis and coinciding with the area of maximal neurologic involvement. In the later stages of the disease, polymorphic delta activity or intermittent frontal dominant monorhythmic slow-wave activity may be present. On occasion, there may be a transient flattening or attenuation of activity after the periodic complexes. Various types of epileptiform discharges, spikes, sharp waves, or spike-and-wave complexes occurring in a focal or generalized fashion also may be present. Patients who have a remission or an improvement in the clinical state show a corresponding improvement on the EEG.

The typical stages of sleep become less recognizable as the disease progresses, and identifiable sleep stages become limited to two main types. These are a low-voltage fast pattern with or without spindle activity and a high-voltage slow-wave pattern. In the later stages of the disease, sleep spindles, V waves, and K complexes disappear and the electroencephalographic differentiation of the various stages of sleep is no longer possible. The periodic complexes often persist during sleep, although their shape and frequency may be modified. On rare occasions, periodic complexes may be activated or occur predominantly during the sleep recording.

In the final stages of the disease, there is often a reduction in amplitude and abundance of the electroencephalographic activity and the recording may become almost isoelectric. In some instances, however, alpha activity may still be present shortly before death.

Although other entities may be associated with a periodic pattern, the stereotyped electroencephalographic complexes occurring in a regular and periodic fashion and having a constant relationship to motor movements make this pattern one of the most characteristic and specific of all electroencephalographic patterns. Close attention to the EEG and clinical features aid in the diagnosis of SSPE and distinguish it from other types of encephalopathies or disease entities.
References