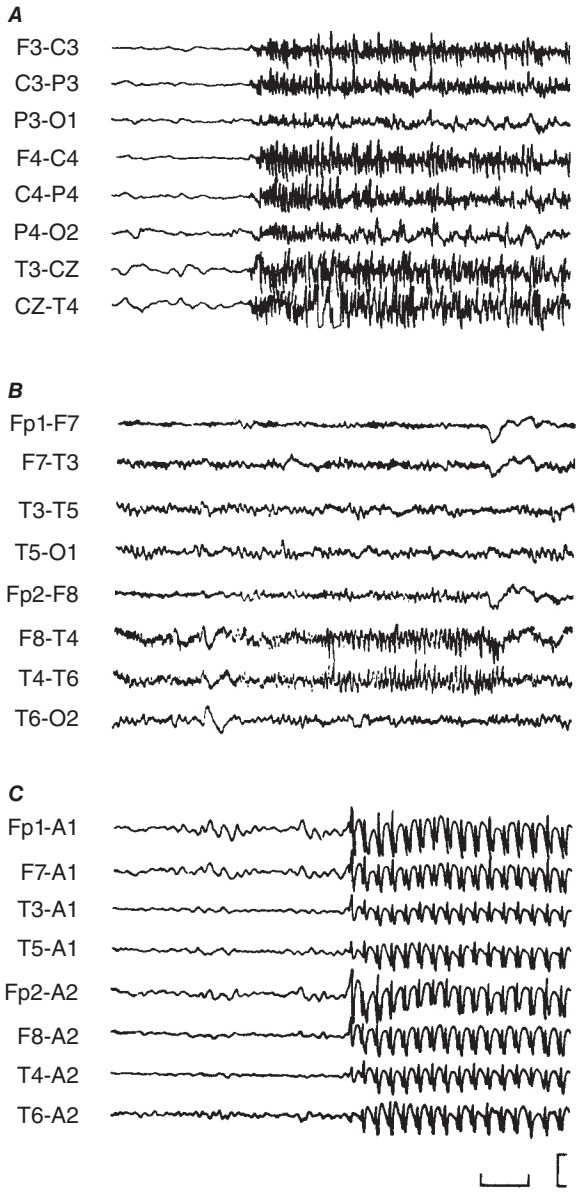

FIGURE 442e-2 **Electrographic seizures.** ***A.*** Onset of a tonic seizure showing generalized repetitive sharp activity with synchronous onset over both hemispheres. ***B.*** Burst of repetitive spikes occurring with sudden onset in the right temporal region during a clinical spell characterized by transient impairment of external awareness. ***C.*** Generalized 3-Hz spike-wave activity occurring synchronously over both hemispheres during an absence (petit mal) attack. Horizontal calibration: 1 s; vertical calibration: 400 μV in *A,* 200 μV in *B,* and 750 μV in *C. (From MJ Aminoff [ed]: Aminoff's Electrodiagnosis in Clinical Neurology, 6th ed. Oxford, Elsevier Saunders, 2012.)*

commonly encountered regardless of whether seizures occur. The EEG findings are of limited utility in determining whether anticonvulsant medication can be discontinued after several seizure-free years. Further seizures may occur after withdrawal of anticonvulsant medication despite a normal EEG or, conversely, may not occur despite a continuing EEG abnormality. The decision to discontinue anticonvulsant medication is made on clinical grounds, and the EEG is helpful only for providing guidance when there is clinical ambiguity or the patient requires reassurance about a particular course of action.

The EEG has no role in the management of tonic-clonic status epilepticus except when there is clinical uncertainty about whether seizures are continuing in a comatose patient. In patients treated by drug-induced coma for refractory status epilepticus, the EEG findings indicate the level of anesthesia and whether seizures are occurring. During status epilepticus, the EEG shows repeated electrographic seizures or continuous spike-wave discharges. In nonconvulsive status epilepticus, a disorder that may not be recognized unless an EEG is performed, the EEG may also show continuous spike-wave activity ("spike-wave stupor") or, less commonly, repetitive electrographic seizures (focal status epilepticus).

THE EEG AND COMA

The EEG tends to become slower as consciousness is depressed, regardless of the underlying cause (Fig. 442e-1). Other findings may suggest diagnostic possibilities, as when electrographic seizures are found or a focal abnormality indicates a structural lesion. The EEG generally slows in metabolic encephalopathies, and triphasic waves may be present. The findings do not permit differentiation of the underlying metabolic disturbance but help to exclude other encephalopathic processes by indicating the diffuse extent of cerebral dysfunction. An EEG responsive to external stimulation is helpful prognostically because electrocerebral responsiveness implies a lighter level of coma than a nonreactive EEG, and thus a better prognosis. Serial records provide a better guide to prognosis than a single record and supplement the clinical examination in following the course of events. As the depth of coma increases, the EEG becomes nonreactive and may show a burst-suppression pattern, with bursts of mixed-frequency activity separated by intervals of relative cerebral inactivity. In other instances there is a reduction in amplitude of the EEG until eventually activity cannot be detected. Such electrocerebral silence does not necessarily reflect irreversible brain damage, because it may occur reversibly in hypothermic patients or with drug overdose. The prognosis of electrocerebral silence, when recorded using an adequate technique, therefore depends on the clinical context in which it is found. In patients with severe cerebral anoxia, for example, electrocerebral silence in a technically satisfactory record implies that useful cognitive recovery will not occur.

In patients with clinically suspected brain death, an EEG recorded using appropriate technical standards may be confirmatory by showing electrocerebral silence, but disorders that may produce a similar but reversible EEG appearance must be excluded. The presence of residual EEG activity in suspected brain death fails to confirm the diagnosis but does not exclude it. The EEG is usually normal in patients with locked-in syndrome (Chap. 446), and helps in distinguishing this disorder from the comatose state with which it is sometimes confused clinically.

THE EEG IN OTHER NEUROLOGIC DISORDERS

In developed countries, computed tomography (CT) scanning and magnetic resonance imaging (MRI) are used as a noninvasive means of screening for focal structural abnormalities of the brain, such as tumors, infarcts, or hematomas (Fig. 442e-1). The EEG is still used for this purpose in many parts of the world, however, although infratentorial or slowly expanding lesions may not be recognized. Focal slow-wave disturbances, a localized loss of electrocerebral activity, or more generalized electrocerebral disturbances are common findings but do not indicate the nature of the underlying pathology.

In patients with an acute encephalopathy, focal or lateralized periodic slow-wave complexes, sometimes with a sharpened outline, suggest a diagnosis of herpes simplex encephalitis, and periodic lateralizing epileptiform discharges (PLEDs) are commonly found with acute hemispheric pathology such as a hematoma, abscess, or rapidly expanding tumor. The EEG findings in dementia are usually nonspecific and do not distinguish reliably between different underlying causes except in rare instances when the presence of complexes occurring with a regular repetition rate ("periodic complexes") supports a diagnosis of Creutzfeldt-Jakob disease (Fig. 442e-1) or subacute sclerosing panencephalitis. In most patients with dementia, the EEG is normal or diffusely slowed, and the findings alone cannot indicate whether a patient is demented or distinguish between dementia and pseudodementia.

CONTINUOUS EEG MONITORING

The brief EEG obtained routinely in the laboratory often fails to reveal abnormalities that are transient and infrequent. Continuous monitoring over 12 or 24 hours or longer may detect abnormalities or capture