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How useful is EEG and EEG monitoring in the acutely ill and how to interpret it?

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Emergency electroencephalography (EEG) is most often requested in patients with acute alterations in mental status. The EEG may indicate that a diffuse central nervous system (CNS) disorder, a focal process, or ongoing seizure activity, often without motor manifestations, such as nonconvulsive status epilepticus (NCSE), is responsible for the clinical presentation. EEG findings in encephalopathies include diffuse slowing of background rhythms (the most common finding), frontal intermittent rhythmic delta activity (FIRDA), and triphasic waves (TWs), a pattern present in a variety of metabolic encephalopathies, most commonly hepatic failure. Other patterns include an alpha–theta coma pattern, a spindle coma pattern, periodic patterns, such as periodic epileptiform discharges (PEDs) that can be lateralized or generalized, and a burst-suppression pattern. Findings, such as focal polymorphic delta activity or localized attenuation of faster frequencies suggest a focal lesion. EEG can be helpful in making a diagnosis, contribute to an established diagnosis, as well as rule out a diagnosis. It can aid in prognostication, particularly if the etiology is known, and in the treatment of epileptic disorders (Varelas et al., 2003; Firosh Khan et al., 2005; Praline et al., 2007).

The possibility of status epilepticus (SE), particularly nonconvulsive status epilepticus (NCSE), is the primary indication for an emergent or ''stat'' EEG (Quigg et al., 2001; Benbadis, 2008). These patients are often obtunded or comatose, and frequently there is no clear etiology of coma or the mental status changes. Because of the spectrum of EEG ictal patterns and the diversity of clinical presentations, ''Is it nonconvulsive status epilepticus (NCSE)?'' is often a difficult question for an electroencephalographer to answer, particularly in an intensive care unit (ICU) setting (Brenner, 2002). Although EEG is required for diagnosis, criteria for NCSE are controversial in obtunded/comatose patients. In addition, the diagnosis is often delayed due to the absence of motor findings, or if findings are present, they often consist of subtle facial, limb, or nystagmoid movements (Drislane et al., 2008). The situation is clearer in ambulatory, confused patients with NCSE (''walking wounded''), as in absence SE or complex partial status, than it is in the ''ictally comatose'' (Walker et al., 2005).

In critically ill patients with marked mental status changes, and frequent nonconvulsive seizures (NCS), defined as electrographic discharges lasting >10 s and evolving with changes in frequency, amplitude, and distribution, the diagnosis of NCSE is relatively straightforward. In contrast to EEG discharges that show a clear evolution, there are a number of periodic EEG patterns—such as periodic lateralized epileptiform discharges (PLEDs), bilateral independent PLEDs (BIPLEDs), generalized periodic epileptiform discharges (GPEDs), as well as TWs, and stimulus-induced rhythmic periodic or ictal discharges (SIRPIDs)—reported as being associated with NCSE (Kaplan, 1999; Brenner, 2004; Hirsch et al., 2004; Kaplan, 2007) that are controversial, particularly as to whether or not they are ictal. Although often interictal, at times they can represent an ictal pattern. Furthermore, these periodic discharges are not specific, and like NCSE can occur in toxic–metabolic encephalopathies, degenerative disorders, CNS infections, following anoxia, or after convulsive seizures (Treiman et al., 1990; Brenner, 2005). Where these periodic patterns fit in an ictal–interictal continuum, and which patterns warrant aggressive treatment, is uncertain (Chong & Hirsch, 2005). This has led to a concerted effort by a number of North American neurophysiology centers to help standardize nomenclature, create a central database, and perform multicenter studies to help answer these questions (Hirsch et al., 2005; Gerber et al., 2008).

Faster frequency generalized discharges (>2.5–3 Hz) are more consistent with an ictal pattern than are slower ones (Young et al., 1996; Chong & Hirsch, 2005; Kaplan, 2007). However, the EEG alone often cannot answer whether the patient is in status, and needs to be interpreted in the clinical context (Korabathina & Benbadis, 2007). A test dose of a benzodiazepine can sometimes be useful to indicate if the patient is in NCSE, particularly if there is a good clinical response or marked improvement of the

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EEG. Unfortunately, some EEG patterns, including TWs due to hepatic or renal encephalopathy, may improve as well (Fountain & Waldman, 2001). Furthermore, although treatment may improve the EEG, with resolution of the epileptiform activity, the patient often does not improve clinically, perhaps because of the underlying brain disorder or the sedative effects of the drug. Failure to improve following treatment does not mean that the patient is not in SE; rather, no conclusion can be reached regarding the presence or absence of NCSE (Jirsch & Hirsch, 2007).

With advances in computer technology, storage capacity, networking, and telecommunications, the use of EEG in evaluating acutely ill patients in the ICU has increased considerably. The goal of continuous EEG (CEEG)

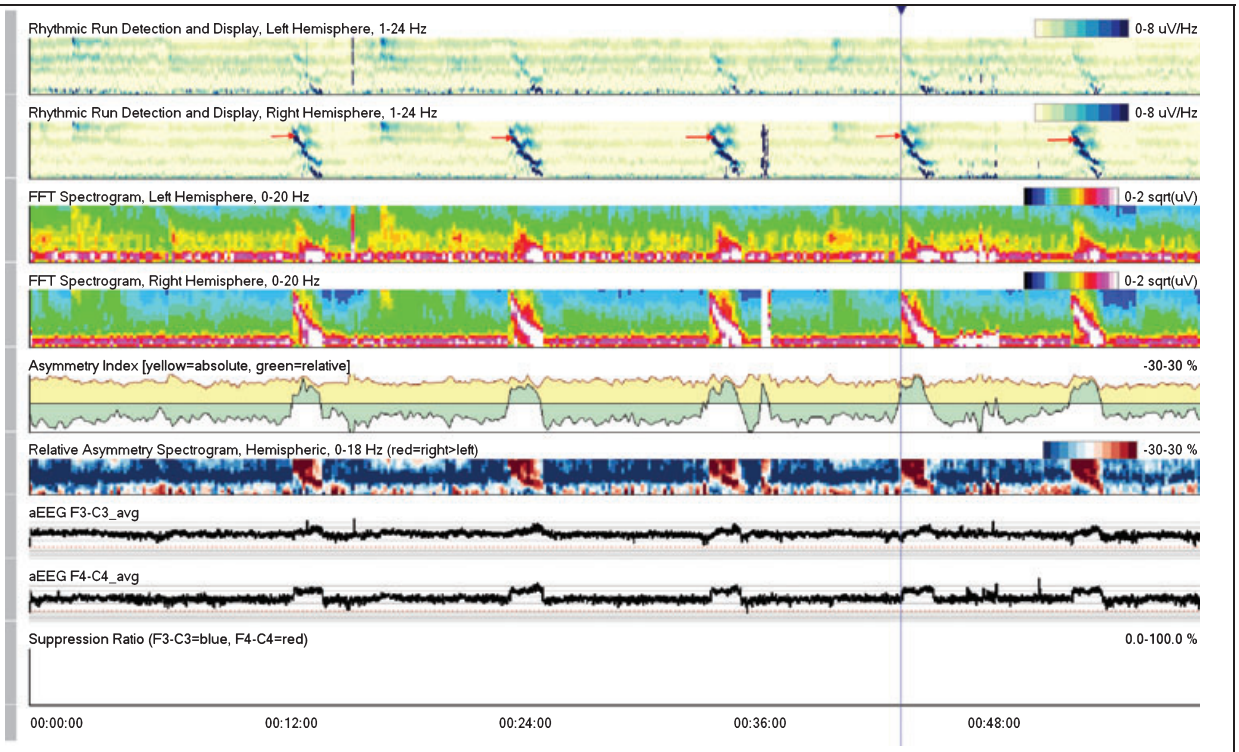

Figure 1.
A 1 h quantitative electroencephalography (QEEG) display in a 71-year-old man with seizures. The top two panels (left hemisphere and right hemisphere electrodes, respectively) are rhythmic run detectors that display rhythmicity from 1–24 Hz that becomes darker when there is rhythmic or periodic activity, and will show a dark band at the frequency of the rhythmic pattern. This allows detection of evolution at times as well (via a diagonal line as the dominant rhythmic frequency gradually changes). Five right-sided seizures are shown (arrows). The most striking finding in this graphic display of EEG activity is that there are marked periodic changes evident across multiple panels simultaneously. The third and fourth panels also represent the left and right hemispheres. Time is shown along the x-axis, frequency on the y-axis (0–20 Hz), and power on the z-axis, with power shown on a color scale where the highest power is white, followed by pink and red (see color scales in the upper right of each panel. There are bursts of power on the right during the seizures (fourth panel). The fifth panel measures symmetry. The asymmetry index shows total absolute asymmetry in yellow. This measure goes up with asymmetry in any frequency or direction. The green relative asymmetry tracing shows laterality: Down-going indicates more power on the left, and upgoing on the right. Note in this case the green tracing is upgoing during the seizures, indicating more power on the right. The sixth panel in red and blue is an asymmetry spectrogram. It shows asymmetry at each frequency from 1–18 Hz averaged over the entire hemisphere. Red means more power on the right in that frequency, whereas blue indicates more power on the left. During the seizures, the right hemisphere becomes dominant. The seventh and eighth panels represent the amplitude-integrated EEG (aEEG), for a single left (F3-C3) and right channel (F4-C4). There is an increase in amplitude on the right during the seizures. The bottom panel does not show anything in this case. It is often helpful in those cases in which suppression is occurring.

monitoring, which consists of prolonged EEG monitoring, often in conjunction with digital video recording, is to help detect and protect patients with an acute brain injury from secondary injuries, such as seizures or cerebral ischemia. Indications include identification of NCS (the most common type of seizure in the ICU) or NCSE, characterization of clinical spells, detection of ischemia, management of a burst-suppression pattern, monitoring treatment and prognosis (Claassen et al., 2004). The most frequent use of CEEG involves monitoring of seizures, as well as status

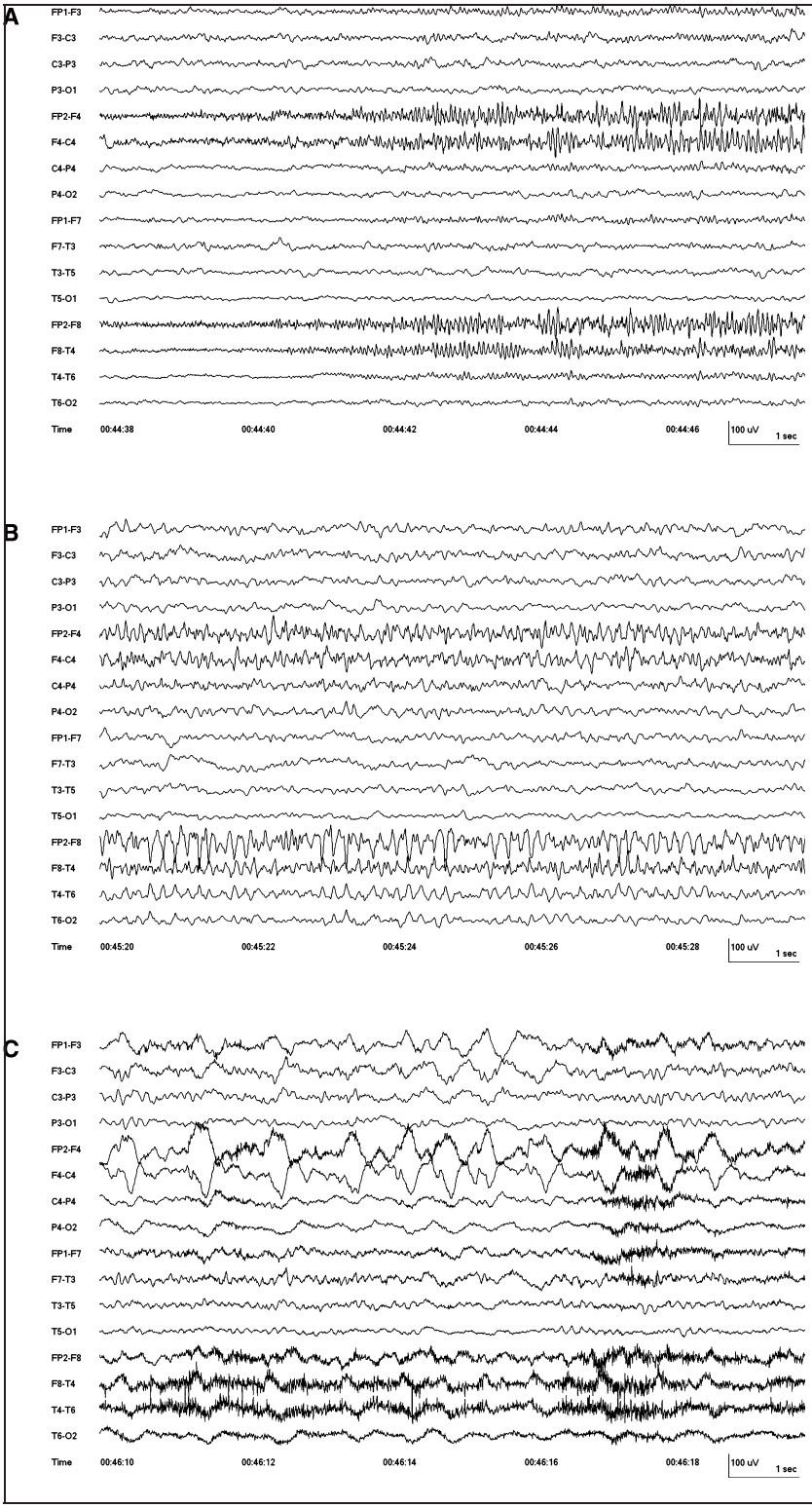

Figure 2.
Electroencephalography (EEG) of the fourth seizure (cursor line in Fig. 1) begins with right-sided beta activity (**A**), slows to the alpha frequency (**B**) and ends with delta activity (**C**).
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epilepticus. Monitoring for 24 h is probably adequate to detect seizures in noncomatose patients without PEDs; longer periods (48 h) may be required for comatose patients (Claassen et al., 2004). Following successful treatment of SE, monitoring should be continued for 24 h, as some patients will have seizures or go back into status (DeLorenzo et al., 1998).

Because monitoring for several days generates gigabytes of data, quantitative EEG tools are helpful in reducing the raw EEG data to review. There are a number of programs utilizing quantitative EEG (QEEG) to help identify seizures and trends (Scheuer & Wilson, 2004). Figure 1 is a 1 h QEEG displaying five seizures in a 71-year-old man, and Fig. 2A–C shows the raw EEG data during the fourth seizure.

What does the future hold for CEEG? Real time monitoring will improve, as will remote monitoring via cell phones and technology yet to be realized. There will be increased automation, including detection and notification alerts. However, at present CEEG is technically difficult, time-consuming, and expensive (Fountain, 2007). Identifying those conditions in which CEEG is most cost- effective and results in better outcomes needs to be determined.

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