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**Review Article**

# Continuous Electroencephalogram Monitoring in the Critically Ill

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## Abstract

The use of continuous electroencephalogram (cEEG) monitoring in the intensive care unit is becoming more widespread, with improvements in data storage capability and networking and the increasing awareness of nonconvulsive seizures. Current and potential uses for this technology include seizure detection, ischemia detection, and prognostication. Nonconvulsive seizures are common in the critically ill, particularly those with acute brain injury and those who are comatose. The implications of some of the electrographical patterns observed in critically ill patients are not yet clear. This article discusses findings with cEEG to date, pitfalls in performing and interpreting these studies, and where we should turn our attention with this underutilized brain monitoring technique.

**Key Words:** Critical illness; continuous EEG monitoring; periodic discharges; nonconvulsive seizures; nonconvulsive status epilepticus; ischemia detection; quantitative EEG; SIRPIDs; coma; prognosis.

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## Introduction

With digital electroencephalograms (EEGs) and recent improvements in storage capability, we can now easily record EEGs 24 hours of every day. This technology has led to the increased use of continuous EEG (cEEG) monitoring in the intensive care unit (ICU). cEEG monitoring plays a critical role in assessing the neurological status of many critically ill patients. EEGs remain the only way to directly monitor cerebral function and the only means to detect epileptiform activity. Current and potential uses for this new technology include ischemia detection, detection of nonconvulsive seizures, and prognostication.

Prior to cEEG monitoring, the clinical examination and neuro-imaging were the main methods available for following the neurological condition of critically ill patients. Use of the clinical examination is limited by the frequent use of sedation (and occasionally paralytics) in the ICU, its inability to provide continuous information, and the fact that many patients with severe brain injuries (such as those with poor-grade subarachnoid hemorrhage [SAH]) have few findings on examination that can be followed reliably over time. Neuro-imaging has its own limitations, because it usually involves transporting critically ill patients who are often ventilator-dependent, those who are on intravenous drips, and those who require constant physiological monitoring that may not be available during transportation or scanning.

EEG monitoring in the ICU presents unique challenges to the neurophysiologist. There are many sources of artifact commonly observed in the ICU that may not be commonly seen during routine EEGs. Analyzing the amount of data produced by 24-hour recordings can be time-consuming. Digital analysis and data compression techniques may be particularly useful for the analysis of these recordings. Finally, it has become clear that there are several unique EEG patterns seen in patients with acute brain injuries that are of uncertain significance. Currently, it is

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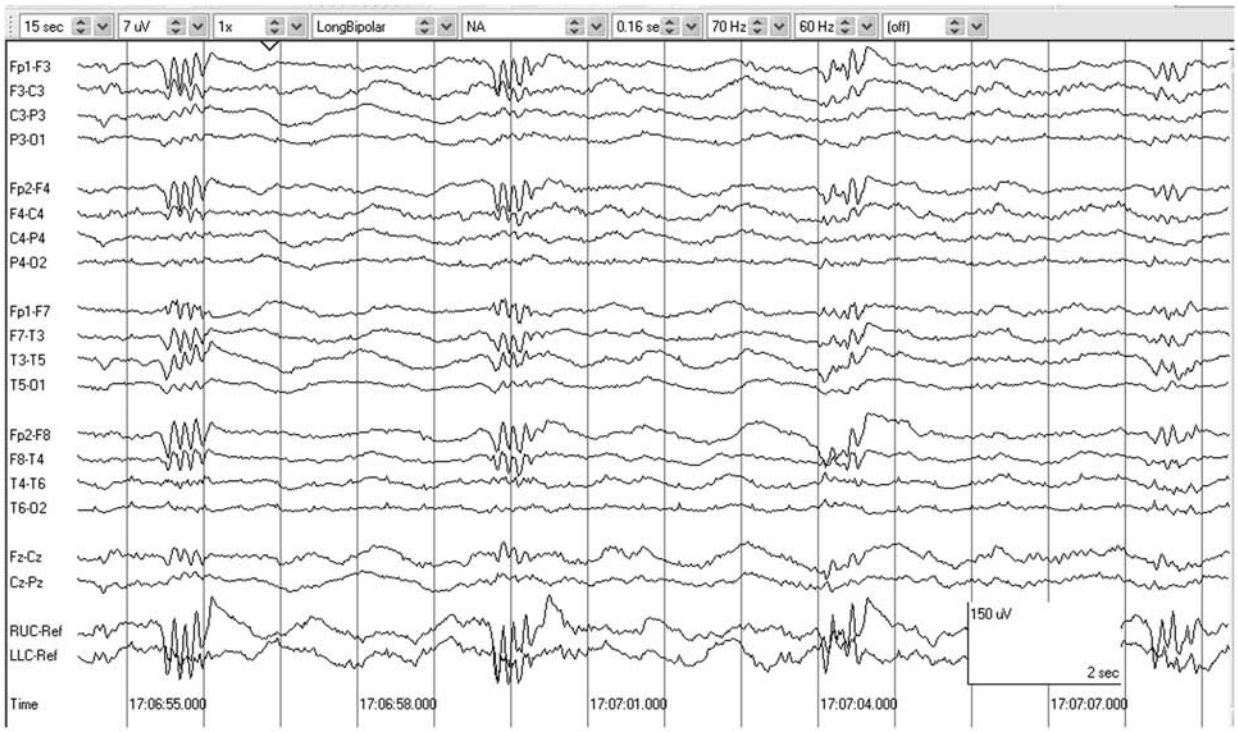


**Fig. 1.** Respirator artifact in a 22-year-old comatose patient with acquired immune deficiency syndrome. The frontally dominant rhythmic theta discharges coincided with triggering of the respirator.

the formidable challenge of the clinician to determine which of these patterns warrant treatment.

## Technical Considerations

cEEG monitoring requires meticulous technical oversight to obtain recordings of adequate quality. Electrodes should be secured with collodion when possible and checked at least twice per day; patients may be agitated and are often physically manipulated and transported for procedures and imaging studies. Many patients in the neurological ICU have altered cranial anatomy, including skull defects, drains, and intracranial catheters; these must be well-documented to allow accurate interpretation, and electrode placement may need to be customized. Scalp edema is particularly common following neurosurgical procedures and can attenuate the EEG—particularly faster frequencies. Intravenous fluid pumps, monitors, and ventilators are common sources of electrical artifact (Figure 1). Dripping intravenous fluids as well as moving water commonly found in ventilator tubing can also produce static charges *(1)*. Chest percussion and beds with automatic percussion capability can produce rhythmic, and even evolving, artifacts that can be confused with seizures (Figure 2). Oscillating beds can also mimic normal patterns, such as a posterior dominant rhythm.

## Data Analysis

The amount of data generated by 24-hour EEG recordings can be cumbersome to analyze. Furthermore, changes in a patient's clinical status can happen quickly, and the ability to quickly detect changes at a reversible stage is critical. We recommend review of the electrographical data at least twice a day and more frequently as clinically warranted. Improved networking capabilities have made frequent review more feasible by allowing for remote monitoring. As ischemia detection becomes more practical, real-time monitoring with built-in computer alarms will be needed to allow instant detection of pending infarction.

Some have argued that critical care nurses can provide the first line of oversight by continuously monitoring EEG waveforms as they monitor other physiological waveforms, such as the electrocardiogram. Jordan *(2)* reported successfully training ICU nurses in bedside EEG interpretation. In a series by Leira et al. *(3)*, various bedside caregivers without expertise in EEG interpretation (including critical care nurses, neurology residents, and EEG technicians) were given a pretest requiring them to identify epileptiform patterns. This was followed by a computer-based lecture and a posttest. Overall, 61% of epileptiform discharges were correctly identified prior to the educational intervention, and 67% were identified after intervention. Following the intervention, EEG technicians correctly identified 94% of epileptiform discharges, whereas critical care nurses identified only 46%. These results suggest that although EEG technicians probably could be quickly trained to continuously monitor electrographical data, oversight by critical care nurses may require more extensive training.

Digital analysis and data reduction techniques offer the possibility of more efficient review as well as the ability to

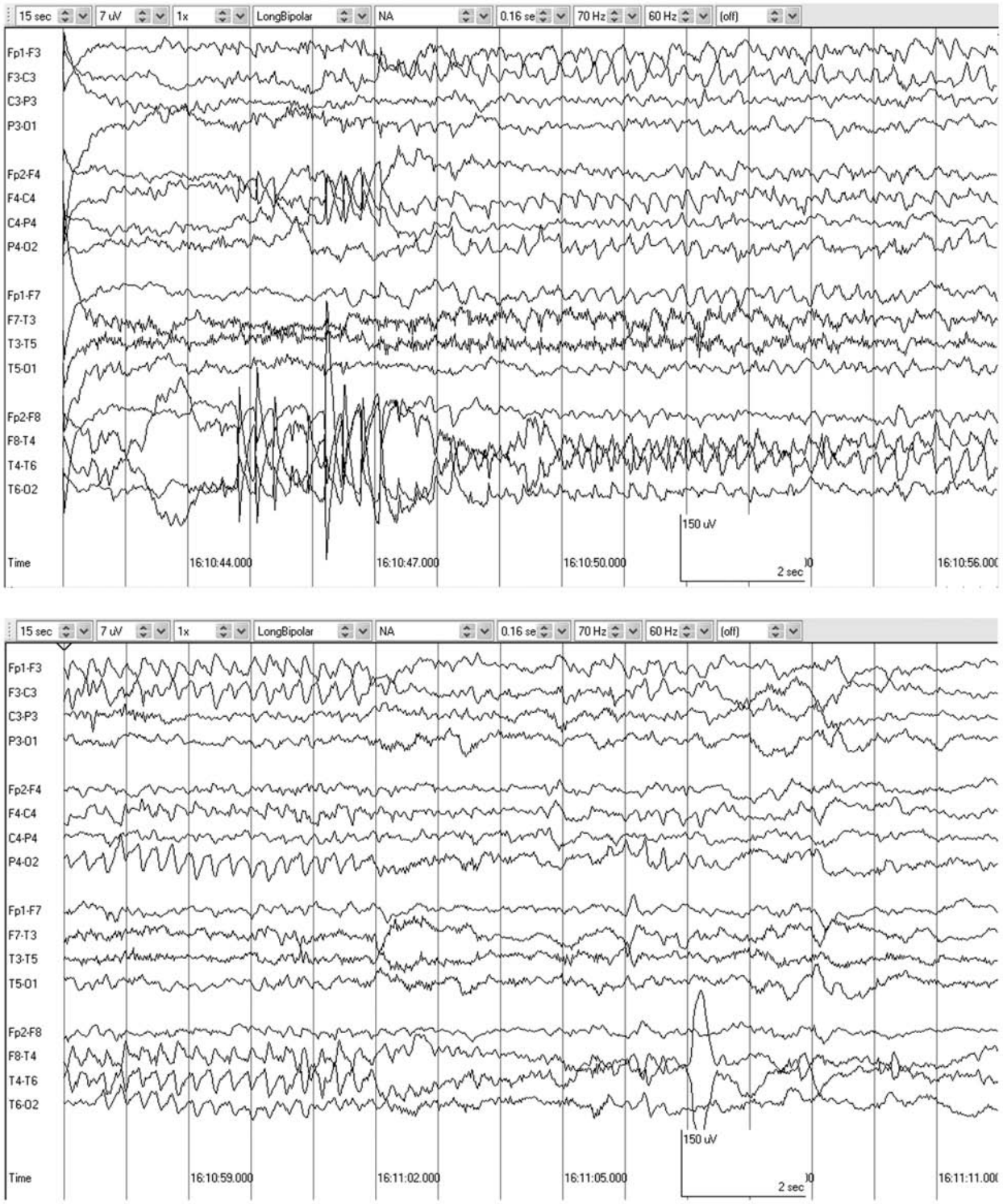


**Fig. 2.** Patting artifact. Two consecutive pages of continuous EEG in a 1-month-old infant with apneic spells. The burst of rhythmic delta seen above represents patting artifact, but could be confused with an ictal discharge. The artifactual origin of the discharge could be determined by its spatial distribution; however, the addition of video allows this determination to be made much more efficiently and confidently.

examine large time intervals. Numerous automatic seizure and spike detectors are available; however, they were developed primarily to detect the types of seizures typically observed in ambulatory patients with epilepsy. As we discuss later, seizures in critically ill patients can appear quite different from those in ambulatory patients, limiting the usefulness of most seizure detection algorithms. In our experience, compressed spectral array (CSA) using Fast Fourier Transformation has been quite

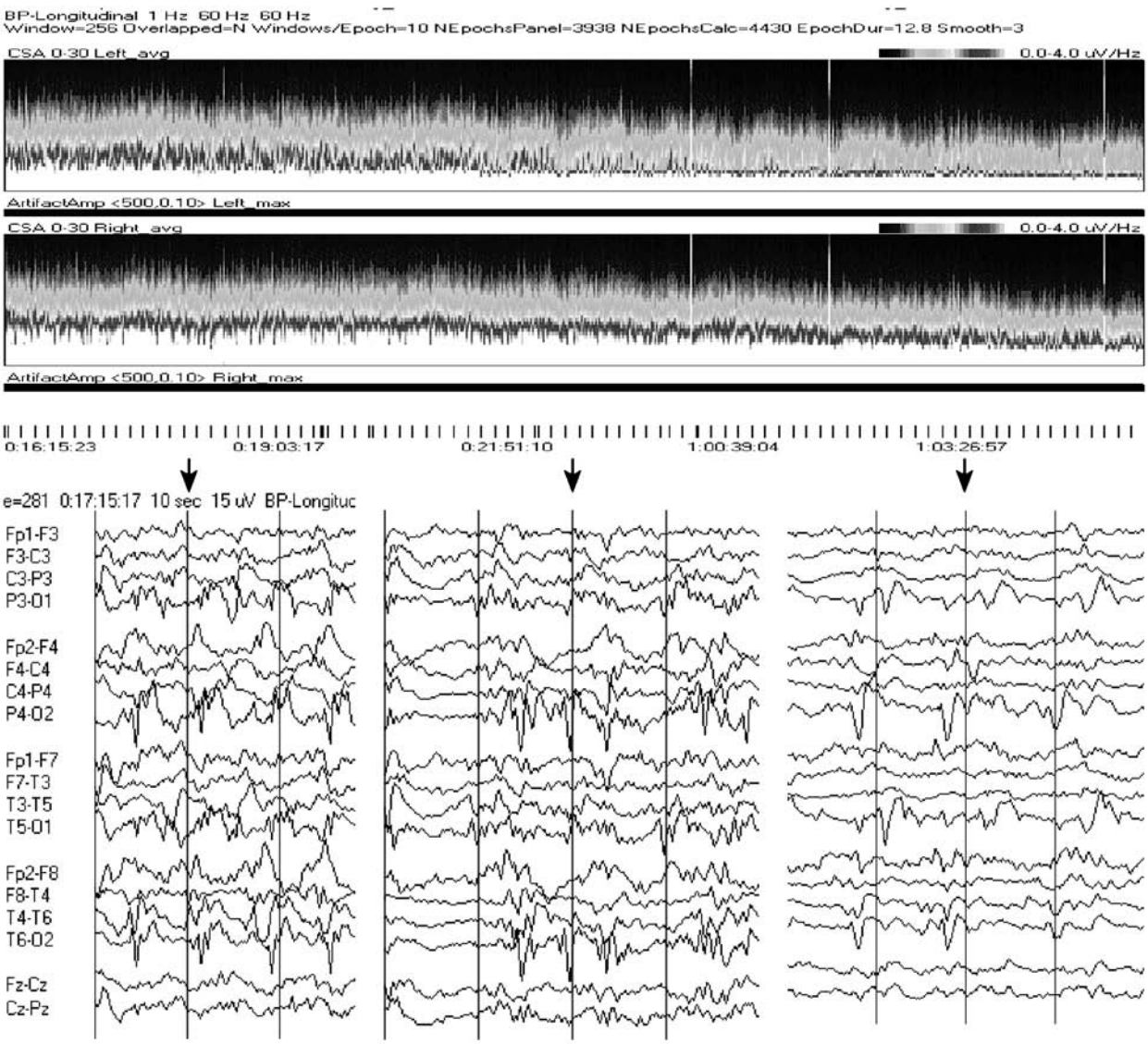


**Fig. 3.** Resolving NCSE–CSA. CSA showing gradual resolution of NCSE over 11 hours. CSA is particularly useful for recognition of such long-term trends. Arrows indicate the approximate time periods in the CSA from which the EEG samples were taken.

useful, including for seizure detection. It has been shown that high-amplitude seizure activity or repetitive epileptiform discharges produces an increase in the amplitude of total power or in the alpha/delta ratio *(4)*. However, artifacts can produce similar findings, and CSA does not detect individual epileptiform discharges. Therefore, correlation with the raw EEG data and interpretation by an experienced electroencephalographer is essential *(5)*.

Additionally, CSA provides easy recognition of state changes and hemispheric asymmetries as well as the ability to detect trends occurring over longer periods of time that may not be easily noticed while reviewing raw EEG (Figure 3). For example, we have found several patients who have had seizures that tend to occur in regular cycles lasting hours. A typical patient might have a seizure, followed by suppression, followed by a gradual increase in the power of faster frequencies until, eventually, another seizure appears many minutes after the first (Figure 4). CSA also transforms electrographical data into an easily interpreted picture, leading some to suggest it could be useful as a means for non-expert caregivers to screen cerebral function at the bedside. However, this application of CSA has not been formally tested. Finally, quantitative EEG has a role in ischemia detection and in determining prognosis, both of which are discussed later.

## Duration of Monitoring

The duration of monitoring greatly depends on the clinical situation. Patients with nonconvulsive seizures are monitored until seizures are controlled. A patient with SAH might be monitored throughout the vasospasm period to detect ischemia. The more difficult issue involves how long to monitor those patients with impaired mental status who are not having known seizures. Claassen et al. *(6)* retrospectively reviewed 110

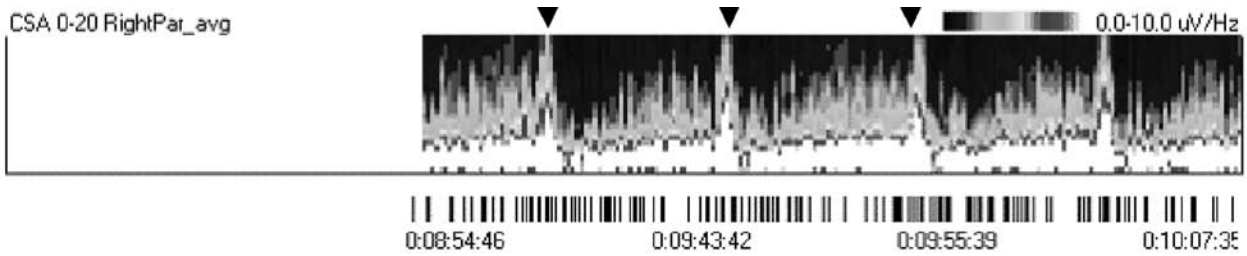


**Fig. 4.** Cyclic seizures detected by compressed spectral array. Note the gradual increase in the power of faster frequencies preceding each seizure. The above sample is derived from approximately 75 minutes of EEG data (black arrows indicate seizures).

patients who were found to have electrographical seizures during monitoring with cEEG. Seizures were detected in half of these patients within the first hour and in 87% during the first 24 hours. In 7% of patients, however, the first seizure was not detected for more than 48 hours. In this study, mental status appeared to be the most important factor in determining the necessary duration of monitoring. Among comatose patients, 20% had their first seizure after more than 24 hours of monitoring, and 13% required more than 48 hours of monitoring. However, only 5% of the noncomatose patients had their first seizure after more than 24 hours. The authors concluded that 24 hours usually was sufficient time to look for nonconvulsive seizures in patients who were not comatose but concluded that 48 hours or more of monitoring may be needed to detect nonconvulsive seizures in comatose patients.

## Detecting Nonconvulsive Seizures

cEEG monitoring has led to the discovery that nonconvulsive seizures are more common than previously believed. Because EEG remains the only test available for detecting epileptiform activity, estimates of the prevalence of nonconvulsive seizures greatly depend on how often patients are monitored with EEG. One study of 124 critically ill neurological patients found that 35% of those patients had nonconvulsive seizures; more than 75% of the patients with seizures were in nonconvulsive status epilepticus (NCSE; ref. *7*). Similarly, another study found that nonconvulsive seizures were present in 37% of patients with impaired consciousness undergoing urgent EEG *(8)*. Towne et al. *(9)* studied 236 comatose patients who had EEGs but who were not suspected of having seizures (those with clinical seizures and subtle movements were excluded) and found that 8% of those patients had NCSE. These patients had no overt clinical signs of seizures and, therefore, could only have been diagnosed by EEG. We reviewed 570 consecutive patients *(6)* who underwent cEEG and found nonconvulsive seizures and NCSE in 19 and 10% of patients, respectively. Two-thirds of these patients were in the neurological ICU. In this series, diagnoses associated with nonconvulsive seizures were epilepsy, central nervous system infection, brain tumor, and "status post neurosurgical procedure." Multivariate analysis was performed to identify risk factors for nonconvulsive seizures. The most significant risk factor was coma: 56% of 96 comatose patients undergoing EEG monitoring had nonconvulsive seizures. Other risk factors for EEG-detected seizures were age less than 18 years (36% had seizures), a history of epilepsy (41% had seizures), and convulsive seizures prior to monitoring (43% had seizures). If two of these four risk factors were present, 40% had seizures; if three of four risk factors were present, 65% had seizures; and if all four risk factors were present, 88% had seizures. Electrographically, periodic epileptiform discharges (PEDs; especially lateralized, but also generalized), not considered seizures in this and most other studies, were associated with seizures.

The lack of a clear definition for nonconvulsive seizures further complicates estimates of the prevalence of nonconvulsive seizures. Nonconvulsive seizures and NCSE seem to have different electrographic manifestations in critically ill patients compared to healthy patients with epilepsy. Granner and Lee *(10)* reviewed electrographic data from 85 episodes of NCSE showing a clinical response to anti-epileptic medications. They found typical and atypical spike-and-wave discharges, polyspike-and-wave discharges, and rhythmic delta with intermixed spikes. Discharge frequencies ranged from 1 to 3.5 Hz (mean: 2.2 Hz). Only 4% of the discharges were 3 Hz or faster. The average frequencies were similar both for those with generalized discharges and those with focal discharges. On the other hand, focal seizures in healthy patients tend to produce faster discharges, particularly in the theta and alpha frequency ranges. This study confirmed the observation that NCSE tends to be slower in frequency than the seizures typically seen in healthy patients with epilepsy.

The nature of seizures in the critically ill has led to one of the most commonly encountered questions in interpreting cEEG: Where do we draw the line between ictal and interictal patterns? For example, the distinction between triphasic waves, NCSE, and generalized PEDs in Creutzfeldt–Jacob disease or postanoxic coma cannot be made based on electrographical findings alone *(11–13)*. The diagnosis of NCSE in these patients can be proven only by a therapeutic intervention (i.e., administration of a benzodiazepine) resulting in resolution of the electrographic pattern as well as clinical improvement. Resolution of the electrographic pattern alone is not sufficient to make a definitive diagnosis of NCSE, because even metabolic patterns can be abolished by benzodiazepines *(12)*. In practice, the clinician and electroencephalographer are often left with an equivocal pattern that improves or resolves with benzodiazepines, but the patient does not improve; this does not help the decision of whether or not the pattern represented NCSE.

Both PEDs and nonconvulsive seizures appear to carry a poor prognosis. One study of 50 patients with status epilepticus (SE)

found that 44% of patients with PEDs had poor outcomes compared to 19% of those without PEDs *(14)*. Etiologies did not differ significantly between the two groups, nor were structural abnormalities significantly associated with PEDs. Jaitly et al. *(15)* found that periodic lateralized epileptiform discharges (PLEDs) and "after SE ictal discharges" were both associated with an approximate 40% mortality rate following SE. It is not known whether treating PEDs could have affected the outcomes, although the same issue could be argued for nonconvulsive seizures.

The clinical significance of nonconvulsive seizures remains uncertain. It is not known whether this electrical activity produces neuronal damage or if it is simply a sign of an injured brain. Animal models of seizures are limited by the fact that they often include provocation of seizures using electrical stimulation or chemical agents that may induce neuronal damage independent of epileptiform activity *(16)*. One animal study used bicuculline to produce convulsive SE in baboons *(17)*. This produced prominent metabolic disturbances, fever, and arrhythmias. Pathology revealed neuronal damage in the neocortex, the cerebellar Purkinje and basket cells, and the hippocampus. In a second study, baboons were paralyzed and artificially ventilated while seizures were induced. Although pathological damage in the cerebellum of these animals was almost completely prevented, neuronal damage in the neocortex and the hippocampus persisted, albeit to a lesser extent compared to the initial study *(18)*. This suggests that some neuronal damage occurs independently of systemic factors. However, it should be noted that NCSE is not simply convulsive SE without the convulsions. Because the electrical activity observed with nonconvulsive seizures tends to be slower and less well-organized than typical convulsive seizures, its ability to produce neuronal damage may be lower than with convulsive seizures *(16)*.

In humans, DeGiorgio et al. *(19)* measured levels of neuron -specific enolase (a marker of acute neuronal injury) in 31 patients with SE. Patients were divided into four groups: those with absence SE, those with complex partial SE, those with generalized convulsive SE (GCSE), and those with subclinical GCSE. Neuron-specific enolase was elevated in all four groups, suggesting that seizure activity without convulsions can produce neuronal injury. They also noted that the highest levels were in those with subtle or subclinical SE and that seizure activity alone (without any acute brain injury) could produce elevations.

We recently described a common and interesting phenomenon termed "stimulus-induced rhythmic, periodic, or ictal-appearing discharges" (SIRPIDs). These are rhythmic or periodic patterns induced by alerting stimuli, including some patterns that have ictal-appearing evolution (Figure 5). In a retrospective study by Hirsch et al. *(20)*, SIRPIDs were found in 33 of 150 patients monitored with continuous EEG. Of those 33 patients, 18 had patterns that qualified as electrographical seizures but that consistently occurred in response to altering stimuli such as suctioning, examination, chest percussion, or loud noise. Other stimulus-induced patterns included PLEDs, generalized periodic epileptiform discharges (GPEDs), triphasic waves, and frontal rhythmic delta activity. Two-thirds of the patients with SIRPIDs had acute brain injuries, and half had seizures in addition to SIRPIDs. There was no difference in the prevalence of clinical seizures between those with and without SIRPIDs. It is not known if these patterns carry the same significance as seizures or PEDs that are not stimulus-related. However, a distinction between the two probably should be made and is only possible by recording simultaneous video with the EEG or routinely interacting with the patient at the bedside while monitoring the EEG.

## Status Epilepticus

cEEG monitoring plays an essential role in the management of patients with SE who do not quickly regain consciousness following cessation of convulsions (Figure 6). A study of 164 patients *(21)* with GCSE found that 48% of patients continued to have electrographical seizures after all convulsions stopped; 14% of patients had NCSE. This could only have been determined with EEG, although a few patients did have subtle twitching. Mortality was significantly higher in those with NCSE (51%) or ictal discharges (32%) compared to those with no ictal discharges (13%). These findings were independent of age and etiology. Similar results were found in the VA Cooperative Study *(22)*, in which 20% of those with GCSE who received "adequate" treatment clinically were found to have nonconvulsive seizures or NCSE on EEG. Those patients who persisted in NCSE had twice the mortality of those whose electrographic seizure activity was controlled. The most important factor determining the prognosis of patients with NCSE is the underlying cause of the seizures. Those patients with NCSE caused by acute brain injuries tend to have a poorer prognosis than those with remote brain injuries, with one study finding an odds ratio of 6.0 for mortality *(23–25)*. Given these data, we believe that EEG is required when a patient does not quickly regain consciousness following treatment for SE.

Prompt diagnosis and treatment of NCSE is crucial. Delayed treatment has been associated with poor outcome *(24)*. Mortality increased from 36% in those in whom NCSE was diagnosed within 30 minutes of onset to 75% in those in whom the diagnosis of NCSE was delayed for more than 24 hours *(24)*. The ability to treat NCSE greatly depends on the duration of the SE. Animal experiments have shown that the efficacy of diazepam and phenytoin diminishes with increasing seizure duration *(26)*. In humans, the efficacy of first-line therapy for SE has been shown to decrease from 80% when initiated within 30 minutes of onset to 40% after 2 hours from onset *(27,28)*.

cEEG monitoring is an essential part of the management of SE in patients treated with continuous intravenous infusions of anticonvulsants. Pentobarbital, commonly used to treat refractory SE, is traditionally titrated to a dose that produces a burst suppression pattern on the EEG. Seizures can still occur in a patient in suppression-burst, and even occasionally in a patient whose EEG is otherwise completely suppressed from barbiturates; therefore, intermittent EEGs are inadequate for monitoring these patients. cEEG monitoring is crucial not only while a patient is in iatrogenic coma but also while medication is being withdrawn to look for breakthrough nonconvulsive seizures. However, one should be aware that emergence from pentobarbital coma may produce epileptiform discharges and PEDs that can falsely suggest recrudescence of NCSE (Figure 7). Although these patterns are not well-described in the literature, some believe they can be recognized by their morphology and distribution, which is usually different than that of the patient's prior ictal activity *(1)*.

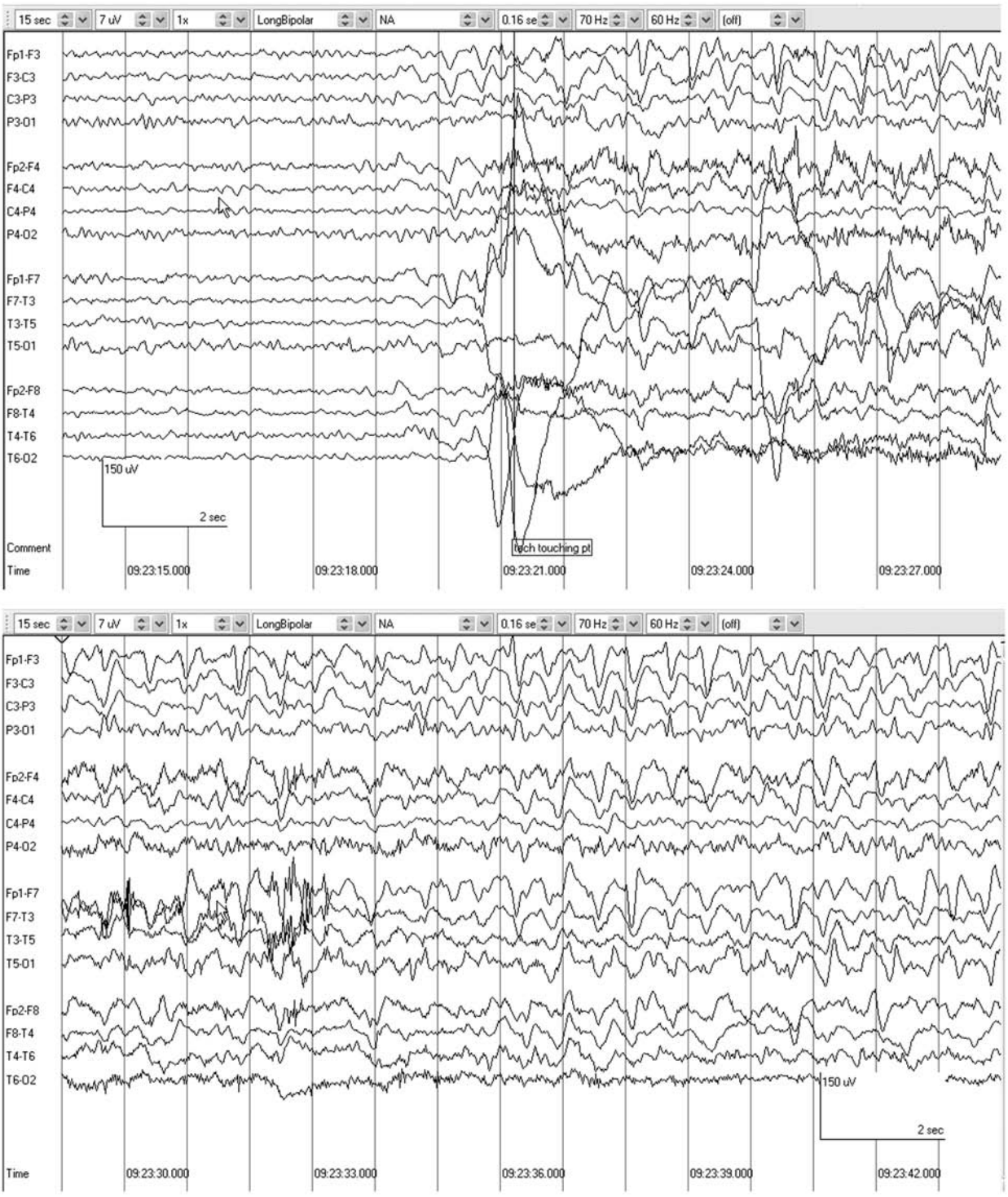


**Fig. 5.** SIRPIDs. Continuous EEG of a 75-year-old patient with a Hunt and Hess grade IV SAH. The background shows moderate diffuse slowing. With an alerting stimulus, generalized periodic or rhythmic sharply contoured slow waves appear, more prominent on the left than the right, with equivocal evolution. These discharges were consistently provoked with stimulation.

Similarly, intravenous midazolam has been used to treat refractory SE. cEEG monitoring is used to titrate the dosage, usually to cessation of clinical and electrographical seizures. Midazolam is then tapered after a period of seizure freedom, a determination that can only be made with cEEG. In a series of 33 patients with refractory SE who were treated with intravenous midazolam, 18% had seizures within 6 hours of treatment, 56% had seizures more than 6 hours after initiation of

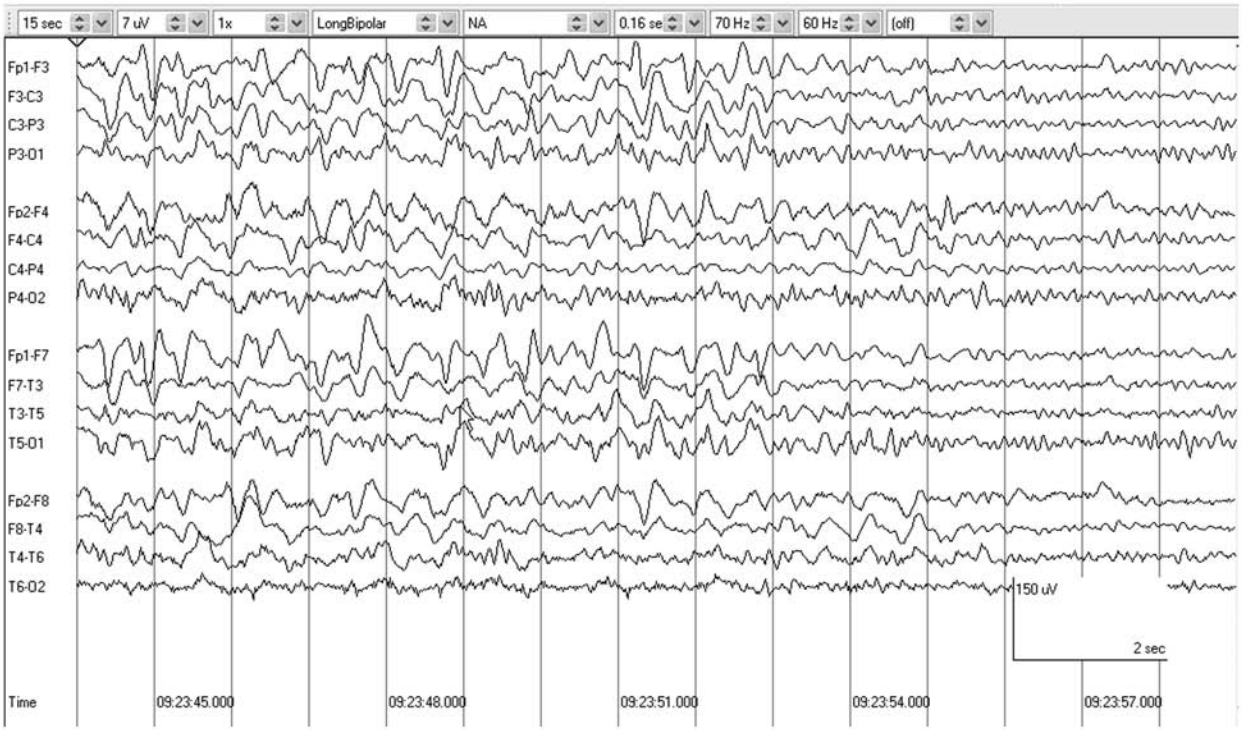


**Fig. 5.** Continued.

therapy, and 68% had seizures after treatment was completed *(29)*. Breakthrough seizures were clinically subtle or purely electrographical in 89% of patients and were associated with an increased risk of seizures posttreatment. Without cEEG, these seizures would not have been noticed.

## Coma and Metabolic Encephalopathy

Continuous EEG monitoring has advantages over routine EEGs in the evaluation of coma and metabolic encephalopathy. Although a typical EEG provides an approximately 30-minute snapshot of a patient's background, cEEG provides one with a greater opportunity to observe state changes and sleep–wake cycles. Quantitative EEG is particularly useful for demonstrating state changes and has a role in determining prognosis in comatose patients (*see* Prognosis). cEEG monitoring is crucial for detecting nonconvulsive seizures, which, as discussed earlier, are commonly found in comatose patients and are not necessarily detectable during a routine EEG.

Aside from detecting seizures, EEG (routine or continuous) can also provide some insight into the etiology of coma. For example, spindle coma or alpha coma is often seen with overdose of tricyclic antidepressants, barbiturates, or benzodiazepines; slow improvement over many hours, which is best detected with quantitative EEG analysis (mentioned earlier), would be consistent with an improving toxic exposure. A fluctuating theta-delta pattern—particularly when triphasic waves are observed—suggests a metabolic encephalopathy. Patients who are "locked-in" demonstrate patterns associated with normal alertness and responsiveness. Finally, coma caused by a structural lesion with mass effect may produce ipsilateral background attenuation with bifrontal and asymmetric delta activity *(1)*.

## Intracerebral Hemorrhage

In a series of 63 patients with intracerebral hemorrhage by Vespa et al. *(30)*, 18 had seizures, almost all of which were nonconvulsive. NCSE occurred in half of those with nonconvulsive seizures. Seizures were observed in 34% of patients with lobar hemorrhage. Surprisingly, 21% of patients with deep subcortic hemorrhages also had seizures. In this study, electrographical seizures were associated with an increased amount of midline shift, independent of ICH size and location, with a trend toward worse outcome. Therefore, subclinical seizures alone may cause increased edema and shift, suggesting that preventing subclinical seizures may be helpful.

## Stroke

cEEG monitoring has two primary roles in acute cerebral infarction: ischemia detection and seizure detection. The incidence of early seizures in patients with acute ischemic stroke in one study was 4.8% *(31)*. The incidence is likely higher when one takes into acount nonconvulsive seizures which are only detected with EEG. Vespa et al. *(30)* found nonconvulsive or convulsive seizures in 6% of 46 patients with ischemic stroke in the ICU. Another study found that the mortality of acute stroke with SE was three times higher than that of acute stroke alone *(23)*. In this study, the presence of SE after stroke remained a significantly poor prognostic factor after accounting for stroke size and location. The use of cEEG for ischemia detection is discussed in the next section.

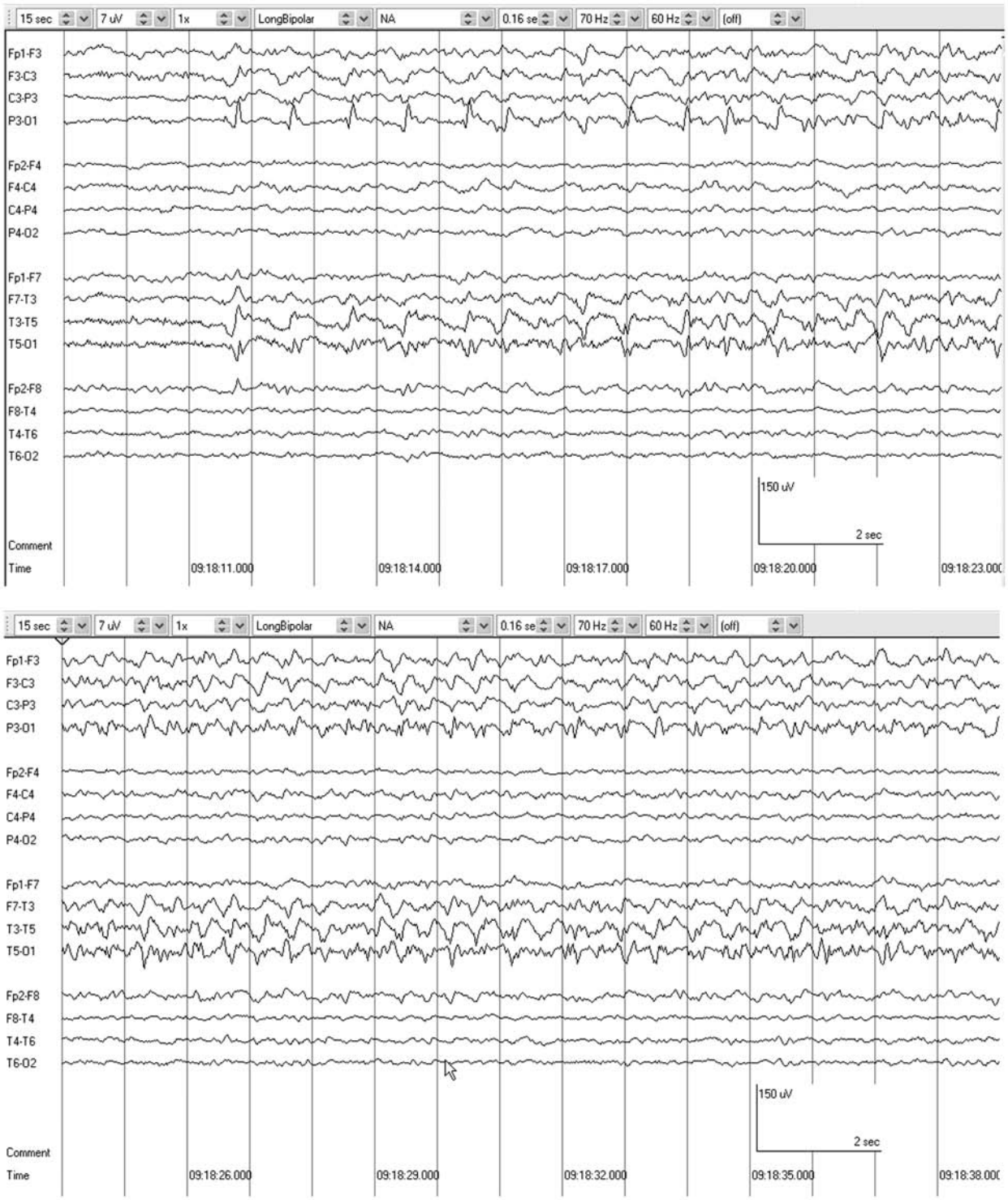


**Fig. 6.** NCSE. Three consecutive pages of EEG from a 68-year-old man with a history of a right fronto-parietal stroke. Prior to this study, the patient developed convulsive SE that clinically resolved after treatment with lorazepam. Nearly continuous electrographical seizures were seen on EEG arising from the left hemisphere. There was no clinical correlate other than impaired mental status.

## Ischemia Detection

The ability of EEG to detect cerebral ischemia at an early stage makes it an ideal tool for monitoring patients at risk for ischemic insults. EEG abnormalities occur when cerebral blood flow (CBF) drops to 25 to 30 mL/100 g/minute, whereas cell death does not occur until blood flow drops to 10 to 12 mL/100 g/minute *(32)*. This allows EEG to detect abnormalities

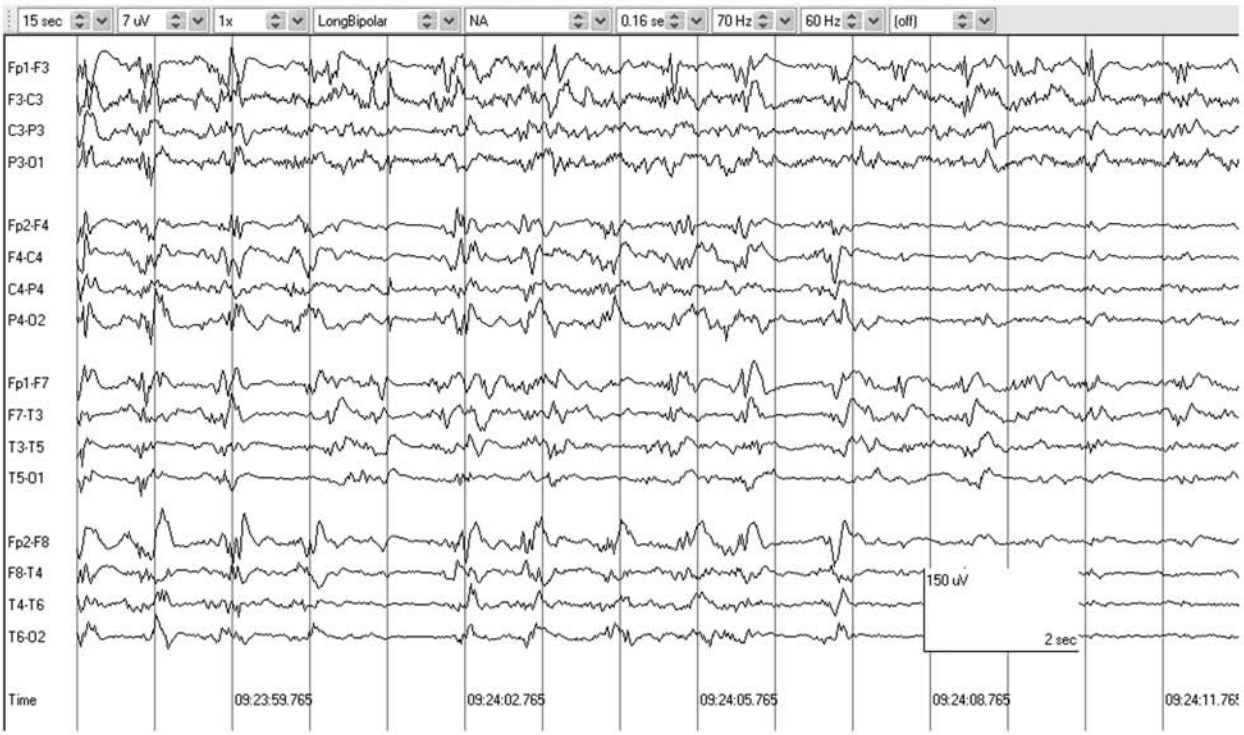


**Fig. 7.** Emergence from pentobarbital coma. Continuous EEG from an 18-year-old patient with acute myelocytic leukemia treated with pentobarbital for raised intracranial pressure caused by a large left intracerebral hemorrhage. Bilateral independent periodic epileptiform discharges were seen only during emergence from pentobarbital. This patient never had a seizure.

at a potentially reversible stage. Signs of ischemia on EEG are a loss of fast (beta) activity, followed by slowing into the theta and then delta ranges, and, finally, flattening of the EEG with burst suppression or continuous suppression *(33)*. The pattern of regional attenuation without delta has been found to be predictive of massive infarction with malignant edema *(34)*. Jordan and Stringer *(35)* found that hemispheric slowing correlated with moderate-to-severe reductions in CBF (as determined by stable Xenon CT CBF measurements). Using hypervolemic therapy, the EEG improved and the ischemia resolved. Quantitative EEG may play a role in ischemia detection as well and is discussed in the next section.

## Subarachnoid Hemorrhage

As in the case of acute stroke, cEEG may be used for seizure and ischemia detection in patients with SAH. In a series of 100 consecutive stuporous or comatose patients with SAH, Dennis et al. *(36)* found NCSE in 8 patients. However, only 26 of those 100 patients had cEEG; therefore, it is quite likely that some electrographical seizures were not diagnosed. Risk factors for NCSE in this study were older age, worse Hunt and Hess grade, and the presence of a ventricular drain. Notably, all eight patients with NCSE died.

Patients who are 4 to 10 days post-SAH are at high risk for vasospasm and subsequent infarction. Clinical changes may not occur early enough to prevent infarction, and patients with grade IV or grade V SAH have few findings on clinical examination that can be followed reliably. Quantitative EEG has been used to reliably detect early ischemia in these patients. Using trend analysis of total power (1–30 Hz), Labar et al. *(37)* detected delayed cerebral ischemia prior to clinical changes in 4 of 11 patients with vasospasm. Vespa et al. *(38)* used quantitative EEG to study 32 patients with SAH, 19 of whom developed angiographic vasospasm. In this series, a loss of relative alpha variability was 100% sensitive for the detection of vasospasm. In 10 of 14 patients, this loss of relative alpha variability preceded angiographic changes or changes in transcranial Doppler by at least 2 days. These two studies were performed primarily with good-grade patients. Clinical changes in higher grade patients are even less reliable because of the paucity of exam findings that can be followed over time. Preliminary results by Claassen et al. *(39)* from 42 grade IV or V patients monitored with cEEG for 7 days showed that the alpha/delta ratio was the most useful of many quantitative EEG parameters for the detection of delayed cerebral ischemia. In this study, six consecutive recordings with a more than 10% decrease in the alpha/delta ratio from baseline were 100% sensitive and 76% specific for delayed cerebral ischemia (DCI). Any single measurement with a more than 50% decrease in the alpha/delta ratio from baseline was 89% sensitive and 84% specific for DCI.

## Traumatic Brain Injury

The incidence of early clinical seizures after head trauma has been reported to be between 4 and 10% *(40–43)*. However, the incidence of nonconvulsive seizures following traumatic brain injury is not established. In a study by Vespa et al. *(33)*, 22% of patients with severe traumatic brain injuries had

seizures on cEEG, with the majority being purely nonconvulsive. Patients with traumatic brain injury also frequently require aggressive intracranial pressure management, sometimes requiring pentobarbital-induced coma. Similarly to those patients with refractory SE, pentobarbital dosing is usually titrated to burst suppression and requires EEG monitoring. In fact, animal studies suggest that barbiturate doses higher than those that produce burst suppression do not further diminish cerebral metabolic rate but do lead to increased cardiovascular depression *(44)*. Systemic hypotension—particularly as a result of pentobarbital therapy—has been shown to negate or reverse the potential beneficial effects of pentobarbital in these patients *(45)*. Finally, quantitative EEG appears to have prognostic value in these patients. Vespa et al. studied 89 patients with moderate-to-severe traumatic brain injury. Among those with a Glasgow Coma Score of to 8 or less, a percent alpha variability of 0.1 or less highly predicted poor outcome or death (positive predictive value: 86%) and independently improved prognostic accuracy *(46)*.

## Prognosis

EEG provides prognostic information in comatose patients; reactivity is particularly important. In a study of comatose patients with traumatic or anoxic brain injuries, Synek et al. *(47)* found that invariant, unreactive alpha activity, burst suppression, and periodic bursts of epileptiform discharges all implied a poor prognosis. Using compressed spectral array, Bricolo et al. *(48)* found that monotonous delta activity was associated with poor outcomes in 95% of comatose patients, compared to 30% in patients with variable or sleep–wake patterns. Quantitative EEG has also been prognostically useful in patients with traumatic brain injury, as discussed earlier *(46)*. Finally, nonconvulsive seizures and PEDs appear to be associated with worse prognosis, although the extent to which this occurs independently of etiology is not clear (*see* earlier section on detecting nonconvulsive seizures). Prospective studies are required to make this determination.

## Cost–Benefit

The variability in how cEEG is used from center to center makes it nearly impossible to make a general statement regarding the cost–benefit analysis of cEEG. One study of 100 patients *(4)* with acute severe head trauma found that hospital costs per patient declined from $88,690 to $49,578 during the 4-year period during which cEEG was used on a regular basis. Length of stay declined from 24.3 to 13.6 days, and the mean Glasgow Outcome Score improved from 2 to 3. It is not known whether such a change can be attributed solely to EEG monitoring and whether benefit exists for other types of patients at other centers.

The effect of cEEG on management is fairly obvious in patients with SE treated with continuous intravenous antiepileptic medications, as discussed previously. In at least one study, however, cEEG was shown to impact decision making in a broader population of patients. In a 1995 series of 124 patients by Jordan et al. *(7)*, cEEG monitoring was found to have an essential impact on clinical decision making in 51% of patients and to have a significant contribution to the decision-making process in 31% of patients. Decisions made based on EEG results included changing or starting anti-epileptic medications, obtaining neuro-imaging, and adjusting cerebral perfusion pressure or mean arterial blood pressure. In this series, cEEG detected potentially treatable pathology in 82% of patients. A smaller study of 15 patients by Claassen et al. *(49)* found that cEEG influenced therapeutic management on almost 50% of monitoring days.

## Conclusion

As a result of recent technological improvements, the continuous recording of EEG and video 24 hours a day is now feasible. Potential uses for cEEG include the detection of nonconvulsive seizures, monitoring continuous intravenous antiepileptic medications, ischemia detection, and prognostication. With the growing use of cEEG, it has become clear that nonconvulsive seizures are quite common in patients with impaired consciousness. The longer nonconvulsive seizures continue untreated, the worse the prognosis and the less successful treatment is likely to be. Therefore, it is critical that patients who do not awaken quickly following SE and those with unexplained stupor or coma receive an EEG as soon as possible.

Further studies are needed to determine whether the types of nonconvulsive seizures typically observed in critically ill patients produce independent neuronal damage and which patterns warrant treatment. Furthermore, the significance of SIRPIDs and patterns that are usually not considered ictal, such as PLEDs, also requires study. Serial measurements of neuronal injury markers such as neuron-specific enolase in critically ill patients with nonconvulsive seizures or SIRPIDs may provide some assessment of ongoing neuronal injury. Measurements of brain lactate using magnetic resonance spectroscopy or cerebrospinal fluid measurements might be similarly helpful. Ideally, a controlled clinical trial would be performed to determine the appropriate treatment for these patterns. A detailed cost–benefit analysis of cEEG would also be useful for decision-making regarding the allocation of healthcare resources. Finally, prospective outcome studies are needed to help determine which electrographical patterns are predictive of outcome.

As cEEG continues to be used more widely, these questions will be answered, although more will certainly arise. Eventually, real-time ischemia detection will be used to detect ischemia at its onset, allowing prevention of stroke in high-risk patients. Similarly, EEG patterns proved to be associated with ongoing neuronal injury will be detected and promptly treated. Both of these very attainable goals have the potential to lead to preserved neuronal function and improved quality of life for these critically ill patients.

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