Detection of electrographic seizures with continuous EEG monitoring in critically ill patients

J. Claassen, S. A. Mayer, R. G. Kowalski, et al.

Neurology 2004;62;1743-1748
DOI 10.1212/01.WNL.0000125184.88797.62

Detection of electrographic seizures with continuous EEG monitoring in critically ill patients

J. Claassen, MD; S.A. Mayer, MD; R.G. Kowalski, BS; R.G. Emerson, MD; and L.J. Hirsch, MD

Abstract—*Objective:* To identify patients most likely to have seizures documented on continuous EEG (cEEG) monitoring and patients who require more prolonged cEEG to record the first seizure. *Methods:* Five hundred seventy consecutive patients who underwent cEEG monitoring over a 6.5-year period were reviewed for the detection of subclinical seizures or evaluation of unexplained decrease in level of consciousness. Baseline demographic, clinical, and EEG findings were recorded and a multivariate logistic regression analysis performed to identify factors associated with 1) any EEG seizure activity and 2) first seizure detected after >24 hours of monitoring. *Results:* Seizures were detected in 19% (n = 110) of patients who underwent cEEG monitoring; the seizures were exclusively nonconvulsive in 92% (n = 101) of these patients. Among patients with seizures, 89% (n = 98) were in intensive care units at the time of monitoring. Electrographic seizures were associated with coma (odds ratio [OR] 7.7, 95% CI 4.2 to 14.2), age <18 years (OR 6.7, 95% CI 2.8 to 16.2), a history of epilepsy (OR 2.7, 95% CI 1.3 to 5.5), and convulsive seizures during the current illness prior to monitoring (OR 2.4, 95% CI 1.4 to 4.3). Seizures were detected within the first 24 hours of cEEG monitoring in 88% of all patients who would eventually have seizures detected by cEEG. In another 5% (n = 6), the first seizure was recorded on monitoring day 2, and in 7% (n = 8), the first seizure was detected after 48 hours of monitoring. Comatose patients were more likely to have their first seizure recorded after >24 hours of monitoring (20% vs 5% of noncomatose patients; OR 4.5, $p = 0.018$). *Conclusions:* CEEG monitoring detected seizure activity in 19% of patients, and the seizures were almost always nonconvulsive. Coma, age <18 years, a history of epilepsy, and convulsive seizures prior to monitoring were risk factors for electrographic seizures. Comatose patients frequently required >24 hours of monitoring to detect the first electrographic seizure.

NEUROLOGY 2004;62:1743–1748

Acute seizures and status epilepticus (SE) are common in all types of acute brain injury. In the Neurologic Intensive Care Unit (Neuro-ICU), up to 34% of patients undergoing EEG monitoring have nonconvulsive seizures (NCS), and 76% of these cases are nonconvulsive SE.[1] Even after excluding all patients with any clinical evidence or history of seizures, still 8% of comatose patients have NCS.[2] NCS have been described in 27% of patients with altered consciousness,[3] 48% of patients after the termination of generalized convulsive SE,[4] 22% with severe traumatic brain injury (TBI),[5] 6% with ischemic stroke,[6] and 28% with intracerebral hemorrhage (ICH).[6] It is important to diagnose these patients as early as possible as the excessive metabolic demand and increased blood flow associated with ictal activity may further compromise at-risk brain tissue following acute brain insults. NCS have also been associated with increased brain edema and midline shift after ICH.[6]

It is unclear how long continuous EEG (cEEG) monitoring should be continued before subclinical seizures may be excluded. In this large hospital-based series of patients referred for cEEG monitoring, we sought to identify risk factors for electrographic seizure activity and to identify patients that require >24 hours of monitoring to record the first seizure. We use the terms "nonconvulsive" and "subclinical" interchangeably in this setting because NCS in stuporous or comatose patients such as these can only be detected by EEG. By "subclinical," we are referring to the fact that they would otherwise be unnoticed; we are not necessarily implying that they are not contributing to a patient's impaired mental status.

Patients and methods. *Study population.* We identified all patients who underwent cEEG monitoring at the Columbia University campus of New York–Presbyterian Hospital between June 1996 and December 2002. Because of the retrospective nature of this study, the need for written informed consent was waived by the hospital institutional review board. Patients were identified using1) the Department of Neurology cEEG log, 2) the Epilepsy Division log containing all cEEG reports for that time period, and 3) a computerized search of the hospital clinical information system for patients who received cEEG monitoring. A complete list of all patients undergoing cEEG monitoring was compiled by cross-referencing the three sources.

The indication for cEEG monitoring was categorized as 1) detection of subclinical seizures or evaluation of unexplained de-

From the Division of Critical Care Neurology (Drs. Claassen and Mayer, R.G. Kowalski) and Comprehensive Epilepsy Center (Drs. Claassen, Emerson, and Hirsch), Department of Neurology, College of Physicians and Surgeons, Columbia University, New York.

The authors thank the attendings, fellows, and technicians of the Columbia Comprehensive Epilepsy Center for assistance in performing and interpreting the EEG and the Neuroscience ICU staff for their support of this project.

Received September 25, 2003. Accepted in final form January 26, 2004.

Address correspondence and reprint requests to Dr. J. Claassen, Division of Critical Care Neurology, Neurologic Institute, 710 W. 168 St., Unit 1, New York, NY 10032; e-mail: jc1439@columbia.edu

Table 1 *Definitions of EEG patterns*

Name	Acronym	Definition
Electrographic seizures		Rhythmic discharge or spike and wave pattern with definite evolution in frequency, location, or morphology lasting at least 10 s; evolution in amplitude alone did not qualify
Periodic epileptiform discharges	PED	Repetitive sharp waves, spikes, or sharply contoured waves at regular or nearly regular intervals and without clear evolution in frequency or location (includes PLED, GPED, BiPLED, triphasic waves)
Periodic lateralized epileptiform discharges	PLED	Consistently lateralized PED
Generalized PED	GPED	Bilateral and synchronous PED with no consistent lateralization
Bilateral PLED	BiPLED	PLED occurring bilaterally, but independently and asynchronously
Triphasic waves		Generalized periodic sharp waves or sharply contoured delta waves with triphasic morphology (typically negative–positive–negative polarity, each phase longer than the prior), at 1–3 Hz, with/without anterior–posterior or posterior–anterior lag
Frontal intermittent rhythmic delta activity	FIRDA	Moderate- to high-voltage monorhythmic and sinusoidal 1- to 3-Hz activity seen bilaterally, maximal in anterior leads, no evolution

crease in level of consciousness, 2) titration of continuous IV (cIV) antiepileptic drug (AED) therapy in patients with refractory SE, and 3) titration of cIV pentobarbital in patients with increased intracranial pressure. Patients in whom monitoring was initiated for titration of cIV-AED for refractory SE or titration of cIV pentobarbital for increased intracranial pressure were excluded from the analysis.

Data collection. All clinical data were gathered from chart review, EEG reports, discharge summaries, and resident sign-out notes. Baseline demographic data (age, gender), past medical history (epilepsy, stroke, brain tumor, and neurosurgical procedures), and the location of the patient at the time of cEEG monitoring (non-ICU hospital floor ["ward"], neuro-ICU, pediatric or neonatal ICU, or medical, cardiac, or surgical ICU) were recorded. Based on chart information, one of the study neurologists retrospectively determined the neurologic status of patients at the time monitoring was initiated (awake, lethargic or stuporous, and comatose), the presence of any convulsive seizures during the current illness prior to cEEG monitoring, and global outcome at hospital discharge (Glasgow Outcome Score [GOS]). The primary admission diagnosis was classified as unexplained decrease in level of consciousness, epilepsy-related seizures, ischemic stroke, subarachnoid hemorrhage (SAH), ICH, TBI, brain tumor, toxic–metabolic encephalopathy, CNS infection, hypoxic–ischemic encephalopathy (HIE), and post neurosurgery (not otherwise specified). For the statistical analysis, we also categorized the above admission diagnoses into epilepsy related, structural (i.e., stroke, brain tumor), or nonstructural (i.e., metabolic encephalopathy or HIE).

CEEG was recorded digitally using 21 electrodes placed according to the International 10–20 System. Recordings were not viewed continuously but were reviewed at least twice daily by a board-certified electroencephalographer. To determine clinical correlates for episodes of electrographic seizures, digital video was screened whenever available and the referring clinical team was contacted every day at the time of creating EEG reports. The presence of convulsive seizures and NCS (for seizure definition, see table 1) as documented by the cEEG report was recorded (chart review, discharge summaries, and resident sign-out notes provided additional information). Seizures were considered convulsive if any of the following was described: "generalized tonic-clonic seizures," "grand mal seizures," "convulsions," "rhythmic jerking," "rhythmic twitching," or similar descriptions. If none of these was present and cEEG confirmed seizures, the seizures were considered nonconvulsive, whether or not subtle movements (e.g., subtle facial twitching, eye deviation, nystagmus) were observed.[7] We recorded the number of continuous hours of cEEG monitoring and categorized the time of cEEG monitoring that was needed to document the first seizure as follows: present at the start of cEEG, within 1 hour, between hours 1 and 6, 6 and 12, 12 and 24, during day 2, between days 2 and 7, and after 7 days of monitoring. We also recorded the presence of periodic epileptiform discharges (PED), including periodic lateralized epileptiform discharges (PLED), generalized PED (GPED), bilateral independent PLED (BiPLED), triphasic waves, frontal intermittent rhythmic delta activity, and suppression–burst activity (see table 1).

Statistical analysis. Data were analyzed using commercially available statistical software (SPSS 9.0; Chicago, IL). A univariate analysis was conducted to identify significant associations with 1) recording seizures on cEEG monitoring among the entire cohort and 2) the need for prolonged cEEG monitoring (>24 hours) to document the first seizure, using χ^2 analysis for dichotomized and categorical variables, the Student t-test for normally distributed continuous variables, and the Mann–Whitney U test for nonnormally distributed continuous variables. Significant variables ($p < 0.05$) were then included in multivariate logistic regression models (forward stepwise; data are reported as odds ratios [OR] and 95% CI) to identify independent predictors of cEEG seizures and the need for prolonged cEEG monitoring. To confirm the validity of our findings in adults, we repeated the above analysis after first excluding infants and children younger than 2 years and then in a third analysis excluding all patients younger than 18 years.

Results. *Study cohort.* Among 603 patients who underwent cEEG monitoring between June 22, 1996, and December 31, 2002, detection of subclinical seizures or unexplained decrease in level of consciousness was the indication for monitoring in 570 patients (95%). Thirty-three patients (5%) were excluded from the analysis because monitoring was initiated to titrate cIV-AED therapy for patients with refractory SE (n = 28) or to titrate cIV pentobarbital therapy for patients with increased intracranial pressure (n = 5).

Mean age of the 570 included patients was 52 ± 25 years, 52% (n = 294) were female, 13% (n = 75) were younger than 18 years, and 7% (n = 41) were age 2 or younger. The most common admission diagnoses for patients who underwent cEEG monitoring were SAH and unexplained decrease in level of consciousness (table 2).

Seizures on cEEG monitoring. Seizures were recorded in 110 (19%) of the 570 patients. The median duration of cEEG monitoring was longer in patients with electrographic seizures than in those without seizures (4.5 vs 2.0 days; $p < 0.001$, Mann–Whitney U test). Seizures were most frequently detected with cEEG monitoring in patients with epilepsy-related seizures (33%), CNS infection (29%), brain tumor (23%), and after neurosurgical inter-

Table 2 *Primary admission diagnoses and frequency of seizures*

Admission diagnoses	n	CEEG findings		
		Any seizure	NCS	NCSE
Epilepsy-related seizures	51	17 (33)	16 (31)	10 (20)
CNS infection	35	10 (29)	9 (26)	6 (17)
Brain tumor	43	10 (23)	10 (23)	5 (12)
Post neurosurgery	13	3 (23)	3 (23)	1 (8)
Hypoxic–ischemic encephalopathy	25	5 (20)	4 (16)	3 (12)
Subarachnoid hemorrhage	108	20 (19)	19 (18)	14 (13)
Traumatic brain injury	51	9 (18)	9 (18)	4 (8)
Toxic–metabolic encephalopathy	38	7 (18)	8 (21)	3 (8)
Unexplained decrease in LOC*	105	17 (17)	16 (15)	5 (5)
Intracerebral hemorrhage	45	6 (13)	6 (13)	4 (9)
Ischemic stroke	56	6 (11)	5 (9)	4 (7)
Overall	570	110 (19)	105 (18)	59 (10)

Data are given as n (% of patients with this admission diagnosis).

* Although cEEG monitoring was initiated for the detection of subclinical seizures or unexplained decrease in level of consciousness in all 570 patients, unexplained decrease in level of consciousness was the primary admission diagnosis in these 105 patients.

cEEG = continuous EEG; NCS = nonconvulsive seizure; NCSE = nonconvulsive status epilepticus; LOC = level of consciousness.

ventions (23%) (see table 2). Patients with seizures were younger, were more likely to have a history of epilepsy or to have undergone a neurosurgical procedure, more often had epilepsy-related seizures as the admission diagnosis, were more likely to have had convulsive seizures prior to the start of cEEG monitoring, and were more often comatose at the time cEEG monitoring was started (table 3). Logistic regression analysis identified the following independent predictors of cEEG-documented seizures: coma on neurologic exam (OR 7.7, 95% CI 4.2 to 14.2; among 97 comatose patients, 56% had seizures on cEEG vs 12% of 473 noncomatose patients), age <18 years (OR 6.7, 95% CI 2.8 to 16.2; among 75 patients <18 years old, 36% had seizures vs 17% of 495 patients older than 18 years), a past medical history of epilepsy (OR 2.7, 95% CI 1.3 to 5.5; among 68 patients with epilepsy, 41% had seizures vs 16% of 502 patients without epilepsy), and convulsive seizures prior to monitoring (OR 2.4, 95% CI 1.4 to 4.3; among 134 patients with convulsive seizures, 43% had seizures on cEEG vs 12% of 436 patients without convulsive seizures). Among patients with no or only one of these four risk factors, 18% had seizures, with two risk factors 40%, with three 65%, and with all four risk factors present, 88% had seizures. All predictors remained significant after excluding infants (≤2 years). After limiting the analysis to adults only (>18 years), all of these predictors remained significant except age (data not shown).

Most patients had exclusively NCS (n = 101); six patients had only convulsive seizures, and three patients had both convulsive seizures and NCS. The majority of patients with cEEG-documented seizures were treated in the Neuro-ICU (61%; n = 67). Among the remaining patients, 20% (n = 22) were located in the pediatric or neonatal ICU, 11% (n = 12) were not in an ICU, and 8% (n = 9) were in medical, cardiac, or surgical ICUs.

Time to record seizures on cEEG. Seizures were detected during the first 24 hours of cEEG monitoring in 88% of all patients who would eventually have seizures detected by cEEG (figure 1). In another 5% (n = 6), the first seizure was recorded on monitoring day 2, and in 7% (n = 8), the first seizure was detected after 48 hours of monitoring.

First seizure after >24 hours of cEEG monitoring. Patients who were comatose at the time cEEG monitoring was started were more likely to require monitoring for >24 hours to detect the first seizure (OR 4.5, $p = 0.018$) (see table 3). No other variables were associated with delayed (>24 hours) detection of seizure activity. Among comatose patients with seizures on cEEG monitoring, 20% (11/54) needed >24 hours to record the first seizure (figure 2) compared with only 10% (3/29) of lethargic or stuporous patients and none of those who were alert (n = 21). Coma remained a significant predictor of the need for prolonged monitoring after excluding infants (2 years or younger) and also after excluding patients younger than 18 years (data not shown). All infants with seizures on cEEG monitoring (n = 17) had their first seizure within the first 24 hours of monitoring (NS).

EEG patterns associated with seizure activity. Among patients with seizures on cEEG monitoring, the following EEG patterns were seen more often than in those without seizures (table 4): PLED (40 vs 11%; $p < 0.001$), GPED (17 vs 6%; $p < 0.001$), and suppression burst (32 vs 3%; $p < 0.001$). In a separate analysis, we identified other cEEG findings that were more frequently observed in patients who required prolonged cEEG monitoring. Only PLED (OR 3.1, $p = 0.047$) were seen more frequently in patients with the first seizure detected after >24 hours of monitoring (see table 4). Among 44 patients with seizures and PLED on cEEG, 21% (n = 9) required >24 hours of monitoring to

Table 3 *Recording of any seizures on cEEG monitoring and delayed recording of first seizure*

Parameters	Seizures on cEEG monitoring			Time of cEEG monitoring to first seizure >24 h		
	Yes, n = 110	No, n = 460	*p*	Yes, n = 14	No, n = 96	*p*
Demographics						
Age, y	44 ± 28	54 ± 24	0.001	52 ± 25	43 ± 28	NS
Children, ≤2 y	16 (15)	25 (5)	0.001	0 (0)	16 (17)	NS
Adults, ≥18 y	83 (76)	412 (90)	<0.001*	11 (79)	72 (75)	NS
Past medical history†						
Epilepsy	28 (26)	40 (15)	0.016*	5 (36)	23 (25)	NS
Stroke	22 (21)	58 (27)	NS	2 (14)	20 (22)	NS
Neurosurgical procedure	13 (12)	12 (6)	0.047	2 (14)	11 (12)	NS
Brain tumor	9 (9)	20 (9)	NS	2 (14)	7 (8)	NS
Admission diagnosis						
Acute structural brain lesion	64 (58)	287 (62)	NS	9 (64)	55 (57)	NS
Acute nonstructural brain lesion	29 (26)	139 (30)	NS	1 (7)	28 (29)	NS
Epilepsy related	17 (16)	34 (7)	0.008	4 (29)	13 (14)	NS
Convulsive seizures prior to cEEG‡	57 (52)	77 (25)	<0.001*	7 (50)	50 (53)	NS
Neurologic status at time of cEEG start						
Coma	54 (49)	43 (9)	<0.001*	11 (79)	43 (45)	0.018

Data are given as n (% of total for column) or mean ± SD.

* Independent predictors of outcome in a forward stepwise logistic regression analysis. Significance is taken at $p < 0.05$.
† Data available in 360 patients (in 107/110 patients with seizures on continuous EEG [cEEG]).
‡ Data available in 423 patients (in 109/110 patients with seizures on cEEG).

record the first seizure compared with 8% (n = 5) of 66 patients with seizures but without PLED. When analyzed together in a multivariate model with coma at the time cEEG monitoring was started, only coma remained in the model.

Outcome. Of the 110 patients with seizures on cEEG, 62 were alive at discharge, 40 were dead, and in 8, the discharge status was unknown. Among 62 alive patients, functional status at discharge (GOS) was available in 42: Three were normal or minimally disabled (GOS 1), 12 were independent and moderately disabled (GOS 2), 24 were dependent and severely disabled (GOS 3), and 3 were in a vegetative state (GOS 4). Patients with the first seizure after >24 hours of monitoring were no more likely to be dead or severely disabled than those whose seizures were detected within the first 24 hours of monitoring.

Discussion. In this retrospective study of 570 hospitalized patients who underwent cEEG monitoring, seizures were recorded in 19%. The vast majority of these seizures (92%) were nonconvulsive. Seizures were most frequent in younger patients (36% of children under age 18 had seizures), those with a past medical history of epilepsy (41%), patients who had

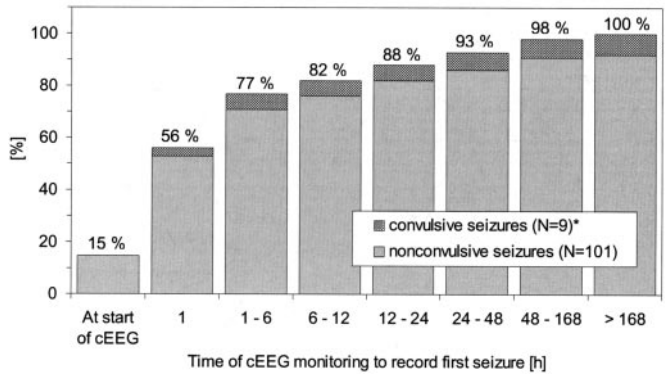

*Figure 1. Time elapsed between start of continuous EEG (cEEG) monitoring and detection of the first seizure (n = 110). *Three of these nine patients had nonconvulsive seizures as well.*

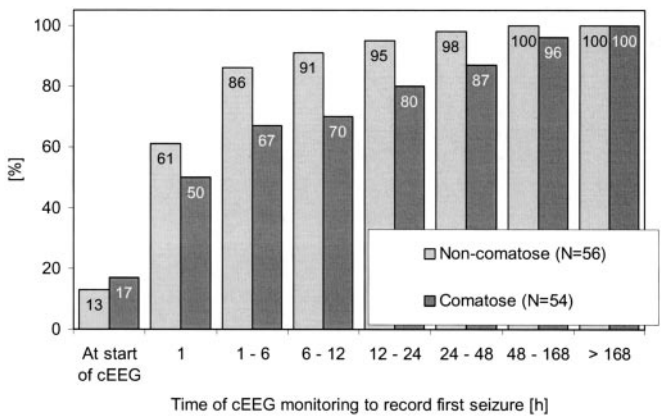

Figure 2. Time to record the first seizure, comparing noncomatose and comatose patients. cEEG = continuous EEG.

Table 4 *Other cEEG findings in patients with seizures on cEEG, n = 110*

Findings	Seizures on cEEG monitoring			Time of cEEG monitoring to first seizure >24 h		
	Yes, n = 110	No, n = 460	*p*	Yes, n = 14	No, n = 96	*p*
Periodic epileptiform findings						
Any	49 (45)	82 (20)	<0.001	9 (64)	40 (42)	NS
PLED	44 (40)	46 (11)	<0.001	9 (64)	35 (37)	0.047
GPED	19 (17)	24 (6)	<0.001	2 (14)	17 (18)	NS
BiPLED	7 (6)	13 (3)	NS	0 (0)	7 (7)	NS
Triphasic waves	4 (4)	25 (6)	NS	0 (0)	4 (4)	NS
Frontal intermittent rhythmic delta activity	11 (10)	35 (9)	NS	2 (14)	9 (10)	NS
Suppression burst	35 (32)	13 (3)	<0.001	4 (29)	31 (32)	NS

Data are given as n (%). Some patients had multiple EEG patterns documented on continuous EEG (cEEG). The observed EEG findings do not have a constant temporal relationship, and seizures may precede other EEG findings in individual patients or vice versa.

PLED = periodic lateralized epileptiform discharges; GPED = generalized PED; BiPLED = bilateral PLED.

convulsive seizures prior to the start of cEEG monitoring (43%), and particularly those who were comatose at the time monitoring was started (56%). Overall, 88% of patients had the first seizure detected within 24 hours of cEEG. However, this was dependent on the patient's neurologic status. The first seizure was detected in under 24 hours of recording in 95% of noncomatose patients but in only 80% of comatose patients ($p = 0.018$). In fact, after 48 hours, 13% of the comatose patients with seizures on cEEG monitoring still had not yet had their first seizure.

We included all patients who underwent cEEG monitoring in our analysis, as opposed to a more specific population, to evaluate the comprehensive experience of our medical center with this diagnostic tool. Etiology, frequency, and significance of seizures may differ substantially in infants, children, and adults. To evaluate possible differences in these patient populations, we stratified our analysis by age and found that all predictors of seizures (with the exception of age) were the same after excluding children <2 years old or in a second set of analysis under age 18. Interestingly, all infants with seizures during cEEG monitoring had their first seizure within the first 24 hours of monitoring, though this did not reach statistical significance. We limited our analysis to patients monitored for the detection of subclinical seizures or evaluation of decreased level of consciousness. Patients in whom monitoring was initiated for titration of cIV-AED for refractory SE or titration of cIV pentobarbital for increased intracranial pressure were excluded because timing of seizures may be related to changes in medications rather than the disease process itself.

Seizure frequency in the current study was comparable with those in prior reports. Overall, we found electrographic seizures in 19% of our cohort compared with 8 to 34% in other studies that evaluated more specific patient populations.[1-6] Compared with prior studies, we found similar frequencies of electrographic seizures in patients with TBI (18% in our study vs 22 to 28% in the literature),[5,8] ischemic stroke (11 vs 6 to 26%),[5,8] and CNS infection (29 vs 33%).[8] We found lower frequencies of electrographic seizures in patients with ICH (13 vs 22 to 28%),[5,8] brain tumor (23 vs 54%),[8] and toxic–metabolic encephalopathy (18 vs 60%).[8] These differences may be related to variability of the study populations, the availability and ordering patterns for cEEG monitoring in each medical center, and differences in sample size and criteria used to define electrographic seizures.

Our findings may not accurately reflect the true frequency of seizures for any particular diagnosis or condition. CEEG monitoring was not initiated as part of a prospective protocol but was initiated for suspicious cases based on clinical judgment. This may have resulted in an overestimation of the seizure frequency by selecting seizure-prone cases. On the other hand, some patients with subclinical seizures may not have been monitored as we did not monitor all patients admitted to our institution with a particular admission diagnosis. Among the monitored patients, those with seizures were studied longer than those in whom no seizures were recorded (4.5 versus 2.0 days). Prolonged monitoring of patients in whom no seizures had been recorded may have revealed additional patients with delayed subclinical seizures. However, the median of 2 days of cEEG monitoring in these patients and the fact that 93% of seizures were detected by monitoring day 2 suggest that the majority of patients without seizures received adequate cEEG monitoring.

Our study does not provide information on how long to monitor individual patients with cEEG to detect seizures, but it can guide decisions about when to discontinue cEEG monitoring in the absence of ictal activity. The duration of cEEG monitoring should always be adjusted according to diagnostic

utility in each individual patient. In 9 to 31% of patients admitted with SE, seizures will be refractory to initial therapy.[9,10] Among patients treated with midazolam infusions, more than half will have electrographic breakthrough seizures during cIV therapy and more than half will have seizures after cIV midazolam has been stopped, and the majority of these seizures (89%) will be subclinical.[11] For all these patients, prolonged monitoring may be needed clinically even in the absence of any ictal findings. However, in the noncomatose patient with 24 hours of cEEG monitoring without evidence of ictal activity, the yield of further monitoring for the detection of seizures would seem to be low, unless clinical changes warrant further monitoring.

We found that comatose patients frequently require prolonged cEEG monitoring to detect seizure activity. Overall, 56% of comatose patients had seizures detected, confirming that subclinical seizures are extremely common in these patients.[2] Twenty percent of comatose patients did not have their first seizure until after the first 24 hours of monitoring, and 13% did not have it until >48 hours of monitoring had been completed. PLED, GPED, and suppression burst were frequently seen in patients with seizures on cEEG monitoring. We did not analyze the temporal relationship of these EEG findings and seizures and are therefore unable to determine the predictive information of these EEG findings for later seizures from this analysis. PLED were also associated with the late detection of seizures. PLED are seen frequently in the aftermath of SE[12,13] and have been associated with poor outcome.[14,15] In our study, 21% of patients with PLED had their first seizure after the first 24 hours of cEEG compared with 8% in those without PLED. Although not specifically studied, we suspect that in some patients, the decision to continue cEEG monitoring beyond the first 24 to 48 hours was due to the presence of periodic discharges.

Our study has several limitations. Given its retrospective design, ordering and treatment biases may have influenced the frequency of EEG findings. Clinical descriptions may have underestimated the frequency of convulsive activity, and outcome data were incomplete. Our study cannot answer some of the most crucial questions currently facing this field: Are seizures in these patients just epiphenomena? Are they a major factor contributing to impaired mental status? Are they causing neuronal injury and worsened outcome? Future prospective studies are needed to address these issues.

Acknowledgment

The authors thank Lewis Kull, REEG-EP-T, and Geraldine Allen.

References

1. Jordan KG. Nonconvulsive seizures (NCS) and nonconvulsive status epilepticus (NCSE) detected by continuous EEG monitoring in the neuro ICU. Neurology 1992;42:180. Abstract.
2. Towne AR, Waterhouse EJ, Boggs JG, et al. Prevalence of nonconvulsive status epilepticus in comatose patients. Neurology 2000;54:340–345.
3. Privitera M, Hoffman M, Moore JL, Jester D. EEG detection of nontonic-clonic status epilepticus in patients with altered consciousness. Epilepsy Res 1994;18:155–166.
4. DeLorenzo RJ, Waterhouse EJ, Towne AR, et al. Persistent nonconvulsive status epilepticus after the control of convulsive status epilepticus. Epilepsia 1998;39:833–840.
5. Vespa PM, Nuwer MR, Nenov V, et al. Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring. J Neurosurg 1999;91:750–756.
6. Vespa PM, O'Phelan K, Shah M, et al. Acute seizures after intracerebral hemorrhage: a factor in progressive midline shift and outcome. Neurology 2003;60:1441–1446.
7. Treiman DM, Meyers PD, Walton NY, et al. A comparison of four treatments for generalized convulsive status epilepticus. Veterans Affairs Status Epilepticus Cooperative Study Group. N Engl J Med 1998;339:792–798.
8. Jordan KG. Continuous EEG and evoked potential monitoring in the neuroscience intensive care unit. J Clin Neurophysiol 1993;10:445. Abstract.
9. Mayer SA, Claassen J, Lokin J, Fitzsimmons BF, Mendehlson F. Predictors of refractory status epilepticus. Arch Neurol 2002;59:205–210.
10. Bleck TP. Advances in the management of refractory status epilepticus. Crit Care Med 1993;21:955–957.
11. Claassen J, Hirsch LJ, Emerson RG, Bates JE, Thompson TB, Mayer SA. Continuous EEG monitoring and midazolam infusion for refractory nonconvulsive status epilepticus. Neurology 2001;57:1036–1042.
12. Treiman DM. Electroclinical features of status epilepticus. J Clin Neurophysiol 1995;12:343–362.
13. Garzon E, Fernandes RM, Sakamoto AC. Serial EEG during human status epilepticus: evidence for PLED as an ictal pattern. Neurology 2001;57:1175–1183.
14. Jaitly R, Sgro JA, Towne AR, Ko D, DeLorenzo RJ. Prognostic value of EEG monitoring after status epilepticus: a prospective adult study. J Clin Neurophysiol 1997;14:326–334.
15. Nei M, Lee J-M, Shanker VL, Sperling MR. The EEG and prognosis in status epilepticus. Epilepsia 1999;40:157–163.