

A comparison of continuous video-EEG monitoring and 30-minute EEG in an ICU

Omar I. Khan¹, Christina J. Azevedo², Alendia L. Hartshorn³, Justin T. Montanye⁴, Juan C. Gonzalez⁵, Mark A. Natola⁶, Stephen D. Surgenor⁷, Richard P. Morse⁷, Richard E. Nordgren⁷, Krzysztof A. Bujarski⁷, Gregory L. Holmes⁸, Barbara C. Jobst⁷, Rod C. Scott⁸, Vijay M. Thadani⁷

¹ St. Luke's University Health Center, Bethlehem, PA

² Yale School of Medicine, New Haven, CT

³ Mission Neurology, Asheville, NC

⁴ MidState Medical Center, Meriden, CT

⁵ Erlanger Health System, Chattanooga, TN

⁶ University of Texas Southwestern, Dallas, TX

⁷ Department of Neurology, Dartmouth-Hitchcock Medical Center, Lebanon, NH

⁸ University of Vermont, College of Medicine, Burlington VT, USA

Received July 1, 2014; Accepted October 8, 2014

ABSTRACT – *Aim.* To determine whether there is added benefit in detecting electrographic abnormalities from 16-24 hours of continuous video-EEG in adult medical/surgical ICU patients, compared to a 30-minute EEG.

Methods. This was a prospectively enrolled non-randomized study of 130 consecutive ICU patients for whom EEG was requested. For 117 patients, a 30-minute EEG was requested for altered mental state and/or suspected seizures; 83 patients continued with continuous video-EEG for 16-24 hours and 34 patients had only the 30-minute EEG. For 13 patients with prior seizures, continuous video-EEG was requested and was carried out for 16-24 hours. We gathered EEG data prospectively, and reviewed the medical records retrospectively to assess the impact of continuous video-EEG.

Results. A total of 83 continuous video-EEG recordings were performed for 16-24 hours beyond 30 minutes of routine EEG. All were slow, and 34% showed epileptiform findings in the first 30 minutes, including 2% with seizures. Over 16-24 hours, 14% developed new or additional epileptiform abnormalities, including 6% with seizures. In 8%, treatment was changed based on continuous video-EEG. Among the 34 EEGs limited to 30 minutes, almost all were slow and 18% showed epileptiform activity, including 3% with seizures. Among the 13 patients with known seizures, continuous video-EEG was slow in all and 69% had epileptiform abnormalities in the first 30 minutes, including 31% with seizures. An additional 8% developed epileptiform abnormalities over 16-24 hours. In 46%, treatment was changed based on continuous video-EEG.

Conclusion. This study indicates that if continuous video-EEG is not available, a 30-minute EEG in the ICU has a substantial diagnostic yield and will lead to the detection of the majority of epileptiform abnormalities. In a small percentage of patients, continuous video-EEG will lead

Correspondence:

Vijay M. Thadani
Department of Neurology,
Dartmouth-Hitchcock Medical Center,
One Medical Center Drive,
Lebanon, NH 03756, USA
<vijay.m.thadani@hitchcock.org>

to the detection of additional epileptiform abnormalities. In a sub-population, with a history of seizures prior to the initiation of EEG recording, the benefits of continuous video-EEG in monitoring seizure activity and influencing treatment may be greater.

Key words: continuous video-EEG, ICU, critically ill, non-convulsive status epilepticus

Continuous EEG is a non-invasive technique that is used to detect a variety of neurological abnormalities. As continuous video-EEG monitoring (cvEEG) has become more common, intensivists and neurologists alike recognize that patients in the intensive care unit (ICU) setting demonstrate a variety of clinical and electrographic epileptiform abnormalities. EEG abnormalities include generalized or focal slowing in the delta frequency range (RDA), sharp waves (SW), and periodic discharges (PDs), which may be subdivided into lateralized (LPDs), bilateral (BIPDs), or generalized periodic discharges (GPDs), of which some may be stimulus-induced (SI) (Kaplan, 2006; Oddo *et al.*, 2009; Hirsch *et al.*, 2013).

Patients in the ICU may have clinical seizures including focal motor, myoclonic, complex partial, and convulsive seizures, and may also have subclinical electrographic seizures (eSZs), and non-convulsive status epilepticus (NCSE) (Mirski and Varelas, 2008; Sutter *et al.*, 2011). NCSE and eSZs are under-diagnosed clinical entities and cvEEG monitoring in the ICU has shown the prevalence of NCSE to be 8-48% in adults and children, depending on which population is studied (Towne *et al.*, 2000; Claassen *et al.*, 2004; Jette *et al.*, 2006; Friedman *et al.*, 2009; Abend *et al.*, 2011a; Shafi *et al.*, 2012; Abend *et al.*, 2013; Payne *et al.*, 2014). In spite of the obvious advantages of cvEEG, there are many unresolved issues, including the optimal duration of monitoring, whether treatment of certain EEG patterns without clinical correlation is warranted, and whether cvEEG is beneficial and cost-effective with regard to patient outcome (Holmes, 2014). When technician time, interpretation time, data storage, and equipment costs are taken into consideration, the costs and labour associated with cvEEG monitoring are likely to be significant. Such efforts and expense may be justified if, as some studies have suggested, the data from cvEEG lead to modification of treatment, and possibly improvements in outcome (Kilbride *et al.*, 2009; Abend *et al.*, 2011b; Payne *et al.*, 2014).

To address these questions, we conducted a 22-month prospective, non-randomized study to further evaluate cvEEG in the ICU setting. In this study, patients in a medical/surgical ICU, including some but not all neurology and neurosurgery patients for whom a routine 30-minute EEG was requested by the treating team, were evaluated with 16-24 hours of cvEEG monitoring. We attempted to determine whether cvEEG monitor-

ing in a mixed ICU population provides any additional benefit in detecting seizures or guiding treatment when compared to a standard 30-minute EEG.

Methods

Study design/inclusion criteria

Approval for this study was given by the Dartmouth-Hitchcock Medical Center Institutional Review Board. This was a prospective, non-randomized 22-month study of 130 consecutive adult (>18 years) patients in a general medical/surgical ICU, including cardiology, neurology and neurosurgery patients. For 117 patients, a 30-minute EEG was requested by medical or surgical/anaesthesia intensivists, neurologists or neurosurgeons. This was for alterations in mental state from various causes including suspected seizures (*table 1*). All 117 patients had a standard 30-minute EEG with video for which the patient was charged. For 83 patients, the recording was then continued for another 16-24 hours with no additional charge to the patient. For 34 patients, the recording was limited to 30 minutes for logistical reasons, including lack of equipment or procedures such as surgery or radiographic testing that required the recording to be discontinued. For 13 patients, cvEEG was requested at the outset because of epilepsy and/or known seizure activity. In these patients, video-EEG recording was initiated and continued for 16-24 hours.

Patients thus comprised three groups as follows:

– **Group 1:** 83 patients, for whom a 30-minute EEG was requested, received a 30-minute EEG with video, followed by 16-24 hours of cvEEG monitoring. A few were monitored for a longer time, but those data were not included in the study.

– **Group 2:** 34 patients, for whom a 30-minute EEG was requested, received only a 30-minute EEG with video.

– **Group 3:** 13 patients, all with epilepsy and/or ongoing seizures and all already taking one or more anti-epileptic medications, for whom cvEEG monitoring was requested, received a 30-minute EEG with video followed by 16-24 hours of cvEEG monitoring. A few were monitored for a longer time, but those data were not included in the study.

Table 1. Characteristics of patients undergoing EEG in the ICU.

	Group 1 83 patients 16-24 hours EEG No. patients (%)	Group 2 34 patients 30-minute EEG No. patients (%)	Group 3 13 patients 16-24 hours EEG No. patients (%)	p value
Hypoxic-ischaemic encephalopathy	29 (35)	10 (29)	0 (0)	0.038
Intra-cranial haemorrhage	22 (27)	10 (29)	1 (8)	0.287
Ischaemic stroke	7 (8)	1 (3)	0 (0)	0.332
Metabolic derangement or infection	16 (19)	9 (26)	3 (23)	0.744
Brain tumour	7 (8)	2 (6)	1 (8)	0.895
Traumatic brain injury	2 (2)	2 (6)	0 (0)	0.488
Epilepsy with breakthrough seizures	0 (0)	0 (0)	8 (62)	<0.001

Technical aspects

All EEGs were 18-channel recordings using the 10-20 system. EKG and video were also recorded. Grass Technologies Comet Portable EEG machines were used, with gold-plated disc electrodes and collodion adhesive. All electrode impedances were maintained at less than 10 kilo-ohms at the start of the recording. The EEG was acquired referentially at a 500-Hz sampling rate, with high-pass and low-pass filters set at 1 Hz and 35 Hz or 70 Hz, respectively. Sensitivity was set between 5 and 10 $\mu\text{V}/\text{mm}$. A 60-Hz notch filter was used in the majority of the recordings. All patients received photic stimulation but not hyperventilation during the first 30 minutes of the recording. ICU nurses were trained to press the alarm button and write notes for clinical seizures or other significant events.

EEG interpretation and clinical treatment

All EEG data were reviewed the day following the recording by board-certified neurologists who were experienced electroencephalographers. There were six EEG readers who had been reading EEGs in the same department for between 10 and 20 years. There was no formal attempt to standardize EEGs readings or demonstrate internal validity, but we knew from experience that disagreements were few. Abnormalities were noted and categorized, and formal reports were generated as part of the patient's electronic medical record. Specific abnormalities noted were generalized or focal slowing, epileptiform discharges, and electrographic or clinical seizures.

EEG slowing was characterized for purposes of formal EEG reports, as generalized or focal and as continuous or intermittent, but these were combined (not treated as separate categories) for the purposes of this report. Slowing was defined as absence or minimal presence of alpha rhythm and preponderance of theta and delta frequencies. If beta and theta frequencies predomi-

nated, the recording could not, strictly speaking, be described as slow, but was treated as abnormal if there was little or no reactivity.

Clinical seizures were readily identified but the distinction between PDs and subclinical electrographic seizures was sometimes an arbitrary one. Broadly speaking, events were characterized as clinical seizures if there was motor activity accompanied by periodic EEG discharges. Subclinical seizures were distinguished from PDs depending on whether they were discrete events, and whether the EEG showed a characteristic faster-to-slower frequency or lower-to-higher-amplitude rhythmic build-up before returning to baseline. Epileptiform discharges were described as focal or generalized spikes and slow waves, and periodic epileptiform discharges (PEDs, or PDs according to new terminology). PEDs were subdivided into lateralized (PLEDs, or LPDs according to new terminology), bilateral independent (BIPLDs, or BIPDs according to new terminology), generalized (GPEDs, or GPDs according to new terminology), and burst suppression patterns. Periodic patterns, as well as clinical and subclinical events, were characterized as spontaneous or stimulus-induced (SI) (Hirsch and Kull, 2004; Kaplan, 2006; Oddo *et al.*, 2009; Hirsch *et al.*, 2013).

In Groups 1 and 3, the patients who received prolonged monitoring, the first 30 minutes of the EEG was compared with the subsequent 16-24-hour recording to see if any additional information was obtained. Information was communicated to the treating team the following day, within 24 hours of the initiation of the recording.

Enrolment in the study was prospective, with waiver of informed consent from the Institutional Review Board (IRB), allowing long-term monitoring to be initiated in the ICU at any time. Some clinical and EEG data, including presence or absence of seizures and PDs, were collected prospectively at the time of recording. We also subsequently compared the clinical and electrographic findings in all three groups, and reviewed

medical records to see whether the additional data obtained from cvEEG impacted treatment or outcome. Specifically, we reviewed records retrospectively to determine whether additional EEG abnormalities or seizures were detected by cvEEG recording beyond 30 minutes, whether the detections influenced changes in antiepileptic drug therapy (new drugs or higher doses of previously used drugs), and whether there was any survival advantage with cvEEG.

In general, the ICU policy was to treat all clinical and sub-clinical seizures with antiepileptic drugs with the goal of full seizure control, and to treat all PDs with antiepileptic drugs but not to the point of suppressing all discharges. Changes in the antiepileptic drug regimen were made for uncontrolled or worsening seizures, and for worsening periodic patterns, unless they were thought to be agonal.

In the decision-making process, drugs used for ICU sedation, such as propofol and midazolam, were counted among antiepileptic drugs. Patients undergoing cvEEG for monitoring of deliberately induced barbiturate coma were excluded from the study.

Statistical analysis

After data were collected, findings were analyzed with chi square tests. The statistical software package SPSS (version 21; Chicago, Illinois) was used.

Results

The underlying diagnoses and reasons for EEG, which included suspected seizures, are listed in *table 1*. There were no significant differences in diagnoses between Groups 1 and 2 (*table 1*; chi square data). As expected from the enrolment criteria, patients in Group 3 had epilepsy and seizures suspected to be ongoing as the primary reason for obtaining EEG. Correspondingly, epilepsy with breakthrough seizures was significantly more frequent as a diagnosis in Group 3 than in Groups 1 and 2 ($p < 0.001$). EEG findings and clinical outcomes in the three groups are summarized in *table 2*.

Group 1

Eighty-three patients receiving cvEEG all had abnormal EEGs with slowing (focal or generalized) and abnormal reactivity during the first 30 minutes of recording. Epileptiform findings in the first 30 minutes, including GPEDs (GPDs) (*figure 1*), burst-suppression (*figure 2*), and PLEDs (LPDs) (*figure 3*), were seen in 28/83 patients (34%). Focal sharp waves and triphasic waves were also observed. This included 2/83 patients (2%) who had clinical seizures. Five of these 28 patients (18%) developed additional epileptic abnormalities in the next

16-24 hours, including two more with clinical seizures. In four patients, treatment was changed as a result of the prolonged EEG.

No epileptiform findings occurred in the first 30 minutes of cvEEG recording in 55/83 patients (66%). However, 7/55 patients (13%) developed epileptiform findings in the next 16-24 hours, including two with electrographic and one with clinical seizures. In three patients, treatment was changed as a result of the prolonged EEG.

Overall, in the group of 83 patients who received an additional 16-24 hours of EEG monitoring, 5/28 patients (18%) who had epileptiform EEG abnormalities in the first 30 minutes developed additional and different epileptiform abnormalities later, and 7/55 (13%) who did not have epileptiform abnormalities initially developed them subsequently. Thus, 12/83 patients (14%) developed new or additional epileptiform abnormalities overnight, and in 7/83 (8%), treatment was changed based on prolonged, as opposed to routine, EEG.

Another way to look at these data is that 28 patients showed epileptiform EEG abnormalities in the first 30 minutes and another seven in the next 16-24 hours. Thus, 80% of patients with epileptiform EEG abnormalities were identified in the first 30 minutes of EEG recording. These results are summarized graphically in *figure 4*.

Group 2

Thirty-four patients received a routine 30-minute EEG. One had a normal EEG, and 33 patients had abnormal EEGs with slowing (focal or generalized) and abnormal reactivity.

Six of 34 patients (18%) had EEGs that, in addition to slowing and abnormal reactivity, showed epileptiform activity, including 1/34 (3%) who had electrographic seizures. Of the 34 patients, 27 (79%) had EEGs that were slow and/or poorly reactive in a manner consistent with encephalopathy, but did not show epileptiform abnormalities. These results are summarized graphically in *figure 5*.

Group 3

Thirteen patients, who were known to have seizures, were monitored with cvEEG for a minimum of 16-24 hours.

During the initial 30 minutes of the EEG study, 9/13 (69%) had epileptiform abnormalities, including three who were in NCSE and one who had focal motor seizures and associated focal EEG abnormalities. One patient initially had a burst-suppression pattern on EEG that overnight evolved into focal epileptiform discharges.

Table 2. Summary of results from continuous video-EEG monitoring.

	Group 1 83 patients 16-24 hours EEG No. patients (%)	Group 2 34 patients 30-minute EEG No. patients (%)	Group 3 13 patients 16-24 hours EEG No. patients (%)	p value
Normal EEG	0 (0)	1 (3)	0 (0)	0.241
Generalized slowing/poor reactivity	55 (66)	27 (79)	4 (31)	0.007
Epileptiform EEG activity (PEDs, burst-suppression, triphasic waves)	28 (34)	6 (18)	9 (69)	0.002
Seizures recorded in first 30 minutes of EEG				
-Electrographic	0 (0)	1 (3)	3 (23)	<0.001
-Clinical	2 (2)	0 (0)	1 (8)	0.290
Additional epileptiform EEG activity recorded overnight (PEDs, burst suppression, triphasic waves, seizures)	12 (14)	Not applicable	1 (8)	0.310
Additional seizures recorded overnight				
-Electrographic	2 (2)	Not applicable	0 (0)	0.570
-Clinical	3 (4)	Not applicable	0 (0)	0.486
Treatment change based on 16-24 hours of EEG	7 (8)	Not applicable	6 (46)	<0.001
Discharged from hospital	42 (51)	16 (47)	10 (77)	0.21



Figure 1. EEG showing generalized periodic epileptiform discharges (GPEDs, or GPDs according to new terminology).



Figure 2. EEG showing burst-suppression.



Figure 3. EEG showing periodic lateralized epileptiform discharges (PLEDs, or LPDs according to new terminology).

In the first 30 minutes, 4/13 patients (31%) demonstrated slowing without epileptiform activity. Overnight, one of these patients (8%) went on to develop epileptiform activity on EEG without clinical seizures.

In 6/13 patients (46%), treatment was influenced by prolonged EEG monitoring.

Another way to look at these data, maintaining the comparison with Group 1, is that nine patients

showed epileptiform EEG abnormalities within the first 30 minutes and another patient showed epileptiform EEG abnormalities within the next 16-24 hours. Thus, 90% of patients with epileptiform EEG abnormalities were identified in the first 30 minutes of EEG recording. These results are summarized graphically in figure 6. A summary of all the results is provided in table 2. Group 1 and group 2 were very similar in terms of neurological disease. The only difference was that a

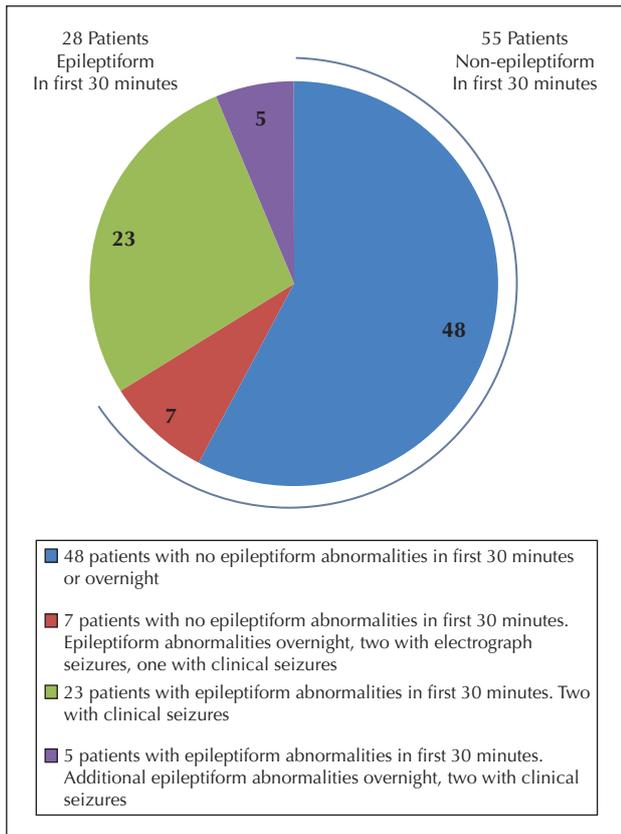


Figure 4. Graphical summary of EEG data from Group 1.

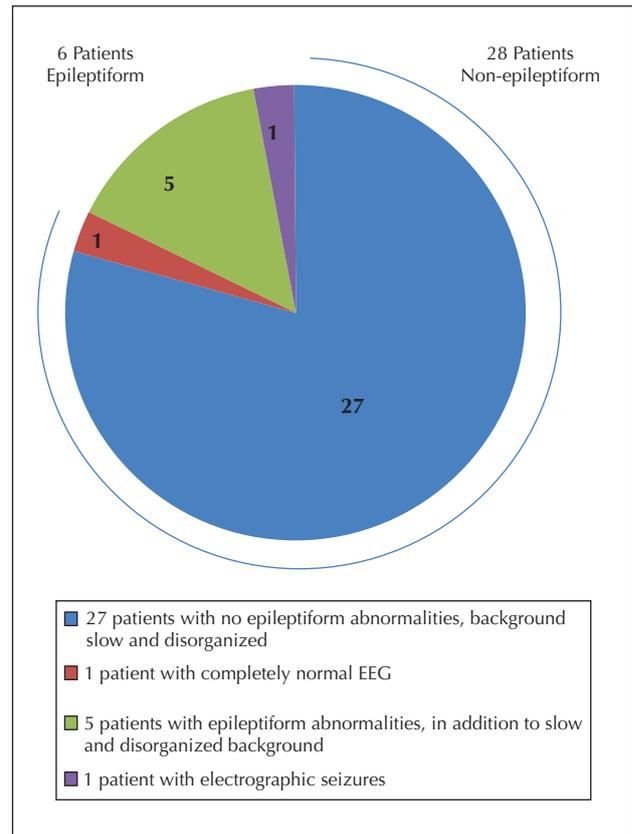


Figure 5. Graphical summary of EEG data from Group 2.

higher percentage (34% vs. 18%; $p=0.002$) of Group 1, the cvEEG group, showed epileptiform activity in the first 30 minutes of recording. We do not have an explanation for this, and do not know whether a higher percentage of Group 2 would have developed epileptiform EEG abnormalities, had we continued their EEG recording beyond 30 minutes. There may have been an unidentified selection bias owing to lack of randomization, but the only apparent reasons why some patients underwent a 30-minute EEG study and others a continuous overnight EEG were logistical and should not cause the population receiving 30-minute EEGs to have fewer seizures in the first 30 minutes compared to the population receiving cvEEG. Survival outcomes in Group 1 with overnight cvEEG and Group 2 with 30-minute EEG were similar (51% vs. 47%).

Patients in Group 3 had a higher likelihood of epileptiform EEG abnormalities in the first 30 minutes of recording (69% vs. 34% and 18%; $p=0.002$) than patients in Groups 1 and 2. They were also more likely than patients in Groups 1 and 2 to have seizures in the first 30 minutes (23% vs. 2% and 3%, respectively; $p<0.001$). Patients in Group 3 also had a higher likelihood of treatment changes based on cvEEG than patients in Group 1,

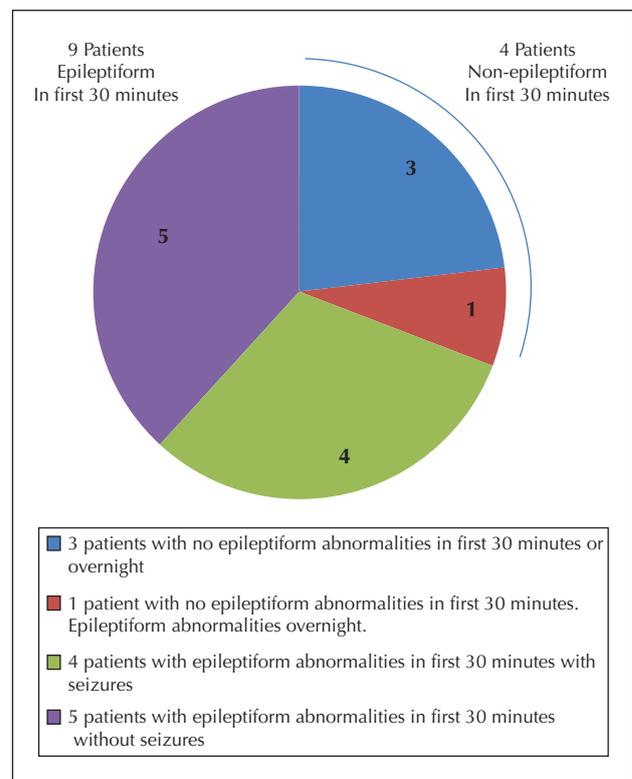


Figure 6. Graphical summary of EEG data from Group 3.

46% vs. 8% ($p < 0.001$). They also had a higher rate of survival and discharge from hospital (77%) than patients in Group 1 (51%) and Group 2 (47%), but this was not statistically significant.

Discussion

Our study is among the first in the adult population to compare, prospectively, a standard 30-minute EEG with cvEEG. Our results suggest that in a general medical/surgical ICU population, epileptiform abnormalities are detected in a 30-minute EEG for a majority of patients, but extending the recording will increase the yield of epileptiform activity by about 20%. Our study is supported by a recent retrospective study in which 74% of patients with seizures had relevant abnormalities detected in the first 30 minutes of EEG (Shafi et al., 2012). Another recent study showed significantly more epileptiform abnormalities detected by long-term EEG monitoring than by a routine EEG, but was performed in a specifically neurological population (Rai et al., 2013).

We believe that our study is widely applicable in the adult ICU population. Many studies of cvEEG have been performed in neurological/neurosurgical ICUs where the likelihood of evolving EEG abnormalities, and the benefit derived from treating them, is probably higher than in the general medical/surgical ICU population (Pandian et al., 2004; Kramer et al., 2012; Rai et al., 2013). We used a medical/surgical ICU with a mixed population of patients, studied consecutively and with a variety of diagnoses (table 1). We did not separate or analyze separately patients with primarily neurological or non-neurological diagnoses because we knew that the neurological patients included were not representative of the entire neurological population. Many patients with neurological diagnoses in our institution were not included in the study because, as noted in the Methods section, they were in other specialized units. Electrographic findings varied considerably among patients (table 2). It is still controversial whether the findings of electrographic seizures, NCSE or PDs are a marker of underlying disease severity, or whether these abnormalities independently contribute to poor patient outcomes (Kurtz et al., 2014; Payne et al., 2014). Payne et al. showed that in a large prospective study of 259 children admitted to paediatric and cardiac intensive care units, who underwent cvEEG and had seizures (behavioural, electrographic or both), the seizures had a major negative impact on outcome. In this group of critically ill children, neurological decline was observed in 67% of the children. Using multivariable analysis, which adjusts for diagnosis and illness severity, the investigators showed that seizures,

independent of illness severity, resulted in increased morbidity, but not mortality (Payne et al., 2014).

PDs have generally been conceptualized as acute injury patterns, but are highly associated with seizures. It is not clear whether in and of themselves they result in injury (Chong and Hirsch, 2005; Kennedy and Gerard, 2012; Foreman et al., 2012; Rai et al., 2013). Our study did not address the issue of which electrographic patterns should be treated more aggressively than others.

Patients in Group 3 had a significantly higher likelihood of epileptiform EEG abnormalities in the first 30 minutes of recording than patients in Groups 1 and 2 (69% vs. 34% and 18%, respectively), but this is not surprising as they were known in advance to have seizures and were selected for cvEEG on that basis. They were also significantly more likely than patients in Groups 1 and 2 to have seizures in the first 30 minutes (23% vs. 2% and 3%, respectively). Patients in Group 3 also had a higher likelihood of treatment changes based on cvEEG than patients in Group 1.

There are several limitations to our study. First, patients were not randomized to routine 30-minute EEG vs. cvEEG, although the two groups in this study, Group 1 and Group 2, appear, for purposes of comparison, to be similar. However, this study reflects the "real life" clinical practice in many institutions without the resources to perform cvEEG for every candidate patient, or where patients fail to undergo cvEEG because of procedures such as surgery or radiographic testing that require the recording to be discontinued.

Second, for most patients, monitoring was limited to 16-24 hours. If we could have monitored longer, additional abnormalities might have appeared, and changes in treatment based on those abnormalities might have improved outcomes and provided additional justification for the monitoring procedure. While different studies are not strictly comparable, in the adult population, it appears that a majority of abnormalities are detected within 24 hours, but data from paediatric studies suggest that longer EEG monitoring may be necessary for epileptiform abnormalities to be detected and such studies may benefit from further monitoring (Claassen et al., 2004; Jette et al., 2006; Kilbride et al., 2009; Abend et al., 2011a; Abend et al., 2011b; Abend et al., 2013; Shafi et al., 2012).

Third, we do not know whether there is a minimum period of recording, beyond the standard 30-minute EEG but shorter than cvEEG, that could capture a larger majority of the new abnormal EEG findings and influence medical management. We also do not know whether reporting EEG results to the treating team in real time, as opposed to 24 hours later, would influence treatment and outcome.

Fourth, the study was not designed to quantify the benefits of cvEEG and we cannot comment on whether

the changes in treatment based on cvEEG really helped the patients. Nor, in a non-randomized study, can we determine a survival or other advantage for cvEEG; although assuming Groups 1 and 2 to be comparable, 16-24 hours of cvEEG did not appear to contribute to survival.

The resources required to perform cvEEG are not trivial when considering the costs of equipment, technicians, and interpretation. The Neurointensive Care Section of the European Society of Intensive Care Medicine (Claassen *et al.*, 2013) recently recommended EEG for generalized convulsive status epilepticus and to rule out non-convulsive seizures in brain-injured patients and in comatose ICU patients without primary brain injury who have unexplained and persistent altered consciousness. Continuous over intermittent EEG was recommended for refractory status epilepticus patients, for patients with status epilepticus and suspected ongoing seizures, and for comatose patients with unexplained and persistent altered consciousness. Yields from cvEEG may be higher in some other subgroups as well (Carrera *et al.*, 2006, Carrera *et al.*, 2008). Whether cvEEG provides a significant clinical benefit in these population groups remains to be determined.

In conclusion, we found that a 30-minute EEG recorded in a medical/surgical ICU will detect the majority of epileptiform abnormalities. However, in a small percentage of patients, cvEEG will detect new epileptiform abnormalities. In a sub-population with a history of seizures prior to the initiation of EEG recording, the benefits of cvEEG in monitoring seizure activity and influencing treatment may be greater. While prolonged cvEEG provides additional information, this study indicates that if cvEEG is not available, a 30-minute EEG in the ICU has a substantial diagnostic yield. □

Acknowledgements and disclosures.

The study was supported by departmental funds.

Costs of the study were covered by the Department of Neurology, Geisel School of Medicine at Dartmouth.

An earlier version of this study was presented as an abstract and poster:

Khan O, Montanye J, Azevedo C, Gonzalez J, Arshad S, Natola M, Surgenor S, Morse, R, Nordgren R, Bujarski K, Holmes G, Jobst B, Thadani V. Value of overnight EEG monitoring in the ICU. American Epilepsy Society (2009). Annual Meeting web site, Boston, Massachusetts, USA.

None of the authors have any conflict of interest to disclose.

References

Abend NS, Gutierrez-Colina AM, Topjian AA, *et al.* Non-convulsive seizures are common in critically ill children. *Neurology* 2011a;76: 1071-7.

Abend NS, Topjian AA, Gutierrez-Colina AM, Donnelly M, Clancy RR, Dlugos DJ. Impact of continuous EEG monitoring on clinical management in critically ill children. *Neurocrit Care* 2011b; 15: 70-5.

Abend NS, Arndt DH, Carpenter JL, *et al.* Electrographic seizures in pediatric ICU patients. *Neurology* 2013; 81: 383-91.

Carrera E, Michel P, Despland PA, *et al.* Continuous assessment of electrical epileptic activity in acute stroke. *Neurology* 2006; 67: 99-104.

Carrera E, Claassen J, Oddo M, Emerson RG, Mayer SA, Hirsch LJ. Continuous electroencephalographic monitoring in critically ill patients with central nervous system infections. *Arch Neurol* 2008; 65(12): 1612-8.

Chong D, Hirsch L. Which EEG patterns warrant treatment in the critically ill? Reviewing the evidence for treatment of periodic epileptiform discharges and related patterns. *J Clin Neurophysiol* 2005; 22(2): 79-91.

Claassen J, Mayer SA, Kowalski RG, Emerson RG, Hirsch JL. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. *Neurology* 2004; 62: 1743-8.

Claassen J, Taccone FS, Horn P, Holtkamp M, Stocchetti N, Oddo M. Recommendations on the use of EEG monitoring in critically ill patients: Consensus statement from the neurointensive care section of the ESICM. *Int Care Med* 2013; 39: 1337-51.

Foreman B, Claassen J, Khaled KA, *et al.* Generalized periodic discharges in the critically ill. *Neurology* 2012; 79: 1951-60.

Friedman D, Claassen J, Hirsch L. Continuous EEG monitoring in the ICU. *Anesth Analg* 2009; 109: 506-23.

Hirsch LJ, Kull LL. Continuous EEG monitoring in the intensive care unit: An overview. *Am J END Technol* 2004; 44: 137-58.

Hirsch LJ, LaRoche SM, Gaspard N, *et al.* American Clinical Neurophysiology Society's Standardized Critical Care EEG Terminology: 2012 Version. *J Clin Neurophysiol* 2013; 39(1): 1-27.

Holmes GL. To know or not to know: Does EEG monitoring in the paediatric intensive care unit add anything besides cost? *Brain* 2014; 137: 1276-7.

Jette N, Claassen J, Emerson RG, Hirsch LJ. Frequency and predictors of nonconvulsive seizures during continuous electroencephalographic monitoring in critically ill children. *Arch Neurol* 2006; 63: 1750-5.

Kaplan PW. EEG monitoring in the intensive care unit. *Am J END Technol* 2006; 46: 81-97.

Kennedy JD, Gerard EE. Continuous EEG monitoring in the intensive care unit. *Curr Neurol Neurosci Rep* 2012; 12: 419-28.

Kilbride RD, Costello DJ, Chiappa KH. How seizure detection by continuous electroencephalographic monitoring affects the prescribing of antiepileptic medications. *Arch Neurol* 2009; 66(6): 723-8.

Kramer AH, Jette N, Pillay N, Federico P, Zygun DA. Epileptiform activity in neurocritical care patients. *Can J Neurol Sci* 2012; 39: 328-37.

Kurtz P, Gaspard N, Wahl AS, *et al.* Continuous electroencephalography in a surgical intensive care unit. *Int Care Med* 2014; 40: 228-34.

Mirski MA, Varelas PN. Seizures and status epilepticus in the critically ill. *Crit Care Clin* 2008; 24: 115-47.

Oddo M, Carrera E, Claassen J, Mayer S, Hirsch L. Continuous electroencephalography in the medical intensive care unit. *Crit Care Med* 2009; 37: 2051-6.

Pandian JD, Cascino GD, So EL, Manno E, Fulgham JR. Digital video-electroencephalographic monitoring in the neurological-neurosurgical intensive care unit: Clinical features and outcome. *Arch Neurol* 2004; 61(7): 1090-4.

Payne ET, Zhao XY, Frndova H, et al. Seizure burden is independently associated with short term outcome in critically ill children. *Brain* 2014; 137: 1429-38.

Rai V, Jetli S, Rai N, Padma MV, Tripathi M. Continuous predictors of outcome in patients with altered sensorium. *Seizure* 2013; 22: 656-61.

Shafi MM, Westover MB, Cole AJ, Kilbride RD, Hoch DB, Cash SS. Absence of early epileptiform abnormalities predicts lack of seizures on continuous EEG. *Neurology* 2012; 79: 1796-801.

Sutter R, Fuhr P, Grize L, Marsch S, Ruegg S. Continuous video-EEG monitoring increases detection rate of nonconvulsive status epilepticus in the ICU. *Epilepsia* 2011; 52(3): 453-7.

Towne AR, Waterhouse EJ, Boggs JG, et al. Prevalence of NSCE in comatose patients. *Neurology* 2000; 54: 864-74.