Published in final edited form as:

Curr Neurol Neurosci Rep. 2013 March ; 13(3): 330. doi:10.1007/s11910-012-0330-3.

Electroencephalographic Monitoring in the Pediatric Intensive Care Unit

Nicholas S. Abend, MD,

Division of Neurology, Children's Hospital of Philadelphia; Departments of Pediatrics and Neurology, Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA. CHOP Neurology, CTRB 10016, 3501 Civic Center Blvd, Philadelphia PA 19104

Kevin E. Chapman, MD,

Department of Pediatrics and Neurology, University of Colorado at Denver, Children's Hospital Colorado, Denver CO

William B. Gallentine, DO,

Division of Pediatric Neurology, Duke University Medical Center. Contact Info: T0913 Children's Health Center, DUMC Box 3936, Durham, NC 27710

Joshua Goldstein, MD,

Child Neurology, Feinberg School of Medicine, Northwestern University. Contact Info: Anne and Robert H. Lurie Children's Hospital, Box 51, 225 E. Chicago Ave, Chicago, IL 60611

Ann E. Hyslop, MD,

Pediatric Neurology, Miami Children's Hospital, Miami FL. Contact Info: Pediatric Neurology, Miami Children's Hospital, 3100 SW 62nd Avenue, Miami, Florida 33155

Tobias Loddenkemper, MD,

Division of Epilepsy and Clinical Neurophysiology, Department of Neurology, Boston Children's Hospital and Harvard Medical School, 300 Longwood Ave, Boston, MA

Kendall B Nash, MD,

Departments of Neurology and Pediatrics, University of California at San Francisco, San Francisco, CA. Address 350 Parnassus Avenue, Suite 609, San Francisco, CA 94143

James J. Riviello Jr, MD, and

Division of Pediatric Neurology and Comprehensive Epilepsy Center, Department of Neurology, NYU School of Medicine, New York, NY. Contact Info: NYU Comprehensive Epilepsy Center, 223 East 34th Street, New York, NY 10016

Cecil D. Hahn, MD, MPH

Division of Neurology, Department of Paediatrics, The Hospital for Sick Children and University of Toronto. Contact Info: 555 University Avenue, Toronto, Ontario M5G 1X8 Canada

On behalf of the Pediatric Critical Care EEG Group (PCCEG)

Nicholas S. Abend: abend@email.chop.edu; Kevin E. Chapman: Kevin.Chapman@childrenshospital.org; William B. Gallentine: william.gallentine@duke.edu; Ann E. Hyslop: ann.hyslop@mchdocs.com; Tobias Loddenkemper: tobias.loddenkemper@childrens.harvard.edu; Kendall B Nash: nashkh@neuropeds.ucsf.edu

Disclosure

N. S. Abend: none; K. E. Chapman: none; W. B. Gallentine: none; J. Goldstein: none; A. E. Hyslop: none; T. Loddenkemper: Laboratory Accreditation Board for Long Term (Epilepsy and Intensive Care Unit) Monitoring, Council of the American Clinical Neurophysiology Society, American Board of Clinical Neurophysiology, Associate Editor for Seizure, and performs video electroencephalogram long-term monitoring, electroencephalograms, and other electrophysiological studies at Boston Children's Hospital and bills for these procedures; K. B. Nash: none; J. J. Riviello, Jr.: spouse is a medical editor for Up To Date; C. D. Hahn: none.

Abstract

Continuous EEG monitoring is used with increasing frequency in critically ill children to provide insight into brain function and to identify electrographic seizures. EEG monitoring use often impacts clinical management, most often by identifying electrographic seizures and status epilepticus. Most electrographic seizures have no clinical correlate, and thus would not be identified without EEG monitoring. There is increasing data that electrographic seizures and electrographic status epilepticus are associated with worse outcome. Seizure identification efficiency may be improved by further development of quantitative EEG trends. This review describes the clinical impact of EEG data, the epidemiology of electrographic seizures and status epilepticus, the impact of electrographic seizures on outcome, the utility of quantitative EEG trends for seizure identification, and practical considerations regarding EEG monitoring.

Keywords

EEG; EEG monitoring; seizure; status epilepticus; intensive care unit; critical care

Introduction

Critically ill children now undergo continuous electroencephalographic (CEEG) monitoring with increasing frequency. CEEG monitoring provides real-time insight into brain function, highlights interval changes in brain function over time, and permits identification of electrographic seizures. Most electrographic seizures in critically ill children have no associated clinical signs and thus cannot be identified without cEEG monitoring. Electrographic seizures, particularly when frequent or prolonged, have been associated with worse outcome. This review summarizes current evidence regarding the utility of cEEG monitoring in critically ill children with a focus on the epidemiology of electrographic seizures and their impact on outcomes.

EEG Monitoring – Indications and Impact

Common indications for CEEG monitoring in the PICU are summarized in Table 1. A recent survey of CEEG use in the pediatric intensive care units (PICUs) of 61 large pediatric hospitals in the United States and Canada reported that the median number of patients who underwent CEEG per month increased about 30% from 2010 to 2011.[1] All centers reported using CEEG to help determine whether events of unclear etiology were seizures. About 90% of centers reported using CEEG in patients considered at risk for electrographic seizures, such as patients with altered mental status following a convulsion, altered mental status in a patient with a known acute brain injury, and altered mental status of unknown etiology. About 50% of centers reported using CEEG routinely in patients with specific diagnoses, such as following resuscitation from cardiac arrest or with traumatic brain injury. [1] Similar data regarding indications were reported by a larger survey of 330 physicians addressing adult and pediatric ICU CEEG.[2]

A recent study reported that CEEG data led to changes in clinical management in 59% of 100 consecutive critically ill children for the indication of acute encephalopathy. These changes included initiating or escalating anticonvulsants due to seizure identification in 43 patients, determining that a specific event (movement or vital sign change) was not a seizure in 21 patients thereby limiting inappropriate treatment, and obtaining urgent neuroimaging in 3 patients.[3] While CEEG data often impacts management, further study is needed to determine whether these changes improve neurodevelopmental outcome.

Additional data regarding the impact of CEEG are available in adults. Studies of emergent EEGs in critically ill adults have reported that they are often rated as clinically useful,[4] contribute to establishing a diagnosis,[5, 6] identify NCS and NCSE,[5, 7] identify seizure mimics,[8] often guide clinical decisions,[9] and often impact clinical management through initiation, escalation, or discontinuation of anticonvulsant medications.[5, 10] Although limited cost effectiveness data are available, one study of 300 critically ill adults reported that CEEG costs were less than 1% of the total hospitalization costs and that CEEG implementation, likely in addition to other advancements in neurocritical care, was associated with a reduction in length of stay and hospital costs.[9]

EEG Background and Prognosis

Assessment of the EEG background provides useful prognostic information regarding the extent of brain injury in some critically ill children, and may identify interval changes in the degree of encephalopathy that could guide clinical management.[11] Although no individual neurodiagnostic modality has perfect prognostic value,[12] the use of EEG is appealing since it can be acquired at the bedside, either continuously or repeatedly over time, and despite inter-rater agreement limitations, provides objective data. Information regarding the patient's clinical status and underlying diagnosis is essential to ensure that EEG background abnormalities are not attributable to sedating medications, scalp edema or intracranial fluid collections.

The majority of data regarding EEG background and prognosis focuses on children with acute hypoxic-ischemic brain injury although some studies have focused on cohorts with more heterogeneous etiologies.[13–15] Patients with more severely abnormal EEG backgrounds tend to have poorer short-term outcome than patients with mild/moderate background abnormalities. Certain EEG background features are known to have prognostic significance [13, 16–22]. Pediatric studies have reported that burst suppression [15, 16], excessive discontinuity [20], severe attenuation [15, 17], lack of reactivity [19, 20], and periodic or multifocal epileptiform discharges [15] are associated with unfavorable prognosis. Conversely, rapid EEG improvement over hours [21], reactivity [18] and normal sleep patterns [18, 22] are associated with favorable prognosis. A study of children treated with therapeutic hypothermia after cardiac arrest resuscitation found that EEG backgrounds categorized as unreactive, discontinuous, burst-suppression, or lacking cerebral activity during the hypothermic or normothermic time periods were associated with unfavorable outcome, although not with perfect predictive value.[14] Thus, while some EEG background features appear to retain their predictive value during hypothermia, they still cannot be used in isolation for prognostication. Recently, a prospective study of 61 adults treated with therapeutic hypothermia following cardiac arrest reported that an unreactive or discontinuous EEG background pattern was associated with elevations in serum neuron specific enolase, suggesting that these early EEG abnormalities may reflect acute neuronal injury.[23]

Electrographic Seizures: Definitions

Electrographic seizures are commonly defined as abnormal, paroxysmal electroencephalographic events that differ from the background activity, last longer than ten seconds (unless associated with clinical signs), have a plausible electrographic field, and evolve in frequency, morphology and spatial distribution.[24, 25] Electrographic seizures may be either *convulsive* or *nonconvulsive*. *Convulsive* (also termed electroclinical) seizures are electrographic seizures that are coupled with clinical manifestations. *Non-convulsive* (also termed subclinical) seizures are electrographic seizures that occur without clinical manifestations. *Subtle* seizures are electrographic seizures accompanied by clinical changes

that are so mild that identification by careful observation in the absence of video-EEG monitoring would be very difficult. While subtle seizures are technically convulsive seizures, since identification generally required EEG monitoring, studies often group them with non-convulsive seizures.

Electrographic status epilepticus is commonly defined as uninterrupted electrographic seizures lasting 30 minutes or longer, or repeated electrographic seizures totaling more than 30 minutes in any one-hour period. Electrographic status epilepticus may be either convulsive or nonconvulsive. While this definition allows classification, it is not based on any scientific evidence that those with a greater than 50% seizure burden in an hour fare worse than those with less than a 50% seizure burden in an hour. More broadly, *non-convulsive status epilepticus (NCSE)* has been broadly defined as an enduring epileptic condition with reduced or altered consciousness, behavioral and vegetative abnormalities, or merely subjective symptoms like auras, but without major convulsive movements.[26]

Agreement among readers in identifying seizures is imperfect,[27] especially when differentiating seizures from rhythmic or periodic patterns. The border between seizures and rhythmic or periodic patterns is often difficult to distinguish with certainty, and has been termed the “ictal-interictal continuum” [24]. Prolonged seizures may be particularly difficult to identify when their onset or offset are unclear, or when they blend with periodic or rhythmic discharges. This is a particular problem in children with pre-existing epileptic encephalopathies, whose often highly abnormal EEGs with abundant inter-ictal epileptiform discharges may resemble NCSE.[28]

Epidemiology of Electrographic Seizures and Status Epilepticus

ES have been reported in 10–40% of children who underwent long-term EEG monitoring in PICUs or emergency departments.[29–39] The majority of ES are not accompanied by any clinical signs,[31, 35, 37–42] even in non-paralyzed patients, [40, 42] although some patients may exhibit subtle positive signs (ie. automatisms, minor facial twitching or blinking) or negative signs (ie. behavioral and cognitive impairment).[43] Certain risk factors for ES have been identified. Children with altered mental status and a known acute neurologic disorder appear to be at greater risk for ES than children who are comatose without an acute neurologic disorder.[29, 35] Children may be at increased risk for ES compared to adults.[44] Clinical risk factors for ES in children include younger age,[29, 37] prior convulsive status epilepticus[37] or acute seizures,[38, 41] structural brain injury[38, 41] including traumatic brain injury,[37] and cardiac arrest.[36] EEG abnormalities associated with ES in children include epileptiform discharges,[37, 41] periodic epileptiform discharges,[31] and lack of background reactivity.[31] ES have been also reported in children with specific conditions: acute ischemic stroke,[45] intracerebral hemorrhage,[46] and those undergoing extracorporeal membrane oxygenation [47, 48], but these studies did not perform CEEG in consecutive patients.

Children undergoing surgery for congenital heart disease are at risk for post-operative seizures. Clinical seizures have been reported in 6% of 171 infants after D-transposition of the great arteries [49] and 18% of 164 infants who survived congenital heart disease surgery requiring deep hypothermic circulatory arrest.[50] Non-convulsive seizures (NCS) are even more common than clinical seizures.[49, 51–55]. In the D-transposition study, ES occurred in 20% of 136 infants undergoing 48 hours of CEEG monitoring, most of which had no clinical correlate.[49] In a second study, NCS occurred in 12% of 183 children who underwent 48 hours of CEEG after cardiac surgery.[52] A retrospective study of infants with congenital heart disease reported ES in 6% of 93 patients.[53] A study of children who underwent cardiac surgery with cardiopulmonary bypass and underwent EEG from

intubation until 22–96 hours after bypass reported ES in 8% of 36.[54] Finally, a study of 39 infants undergoing Norwood-type operations and continuous amplitude-integrated EEG identified intraoperative seizures in 23% and postoperative seizures in 18%.[55] Risk factors for seizures in children with congenital heart disease include co-existing genetic defects, aortic arch obstruction, the presence of a ventricular septal defect, treatment with deep hypothermic circulatory arrest rather than continuous cardiopulmonary bypass and prolonged deep hypothermic circulatory arrest.[49–51]

Current management strategies for electrographic seizures occurring among critically ill children are varied. When surveyed, neurologists reported that the most commonly used medications used to manage NCS and NCSE were lorazepam, phenytoin/fosphenytoin, and levetiracetam. The second and third line medications were these same three in varying combinations, and there was substantial variability in practice. Most physicians reported escalating treatment for NCS to include pharmacologic coma induction with intubation if seizures persisted after 2–3 standard anticonvulsants, for which the most commonly used medications were midazolam and pentobarbital.[2] This finding suggests that physicians are willing to take some medical risk in an attempt to terminate NCS and NCSE. Studies are needed to identify the optimal management regimen and then determine whether seizure identification and management improves outcome.

Duration of Continuous EEG Monitoring

A survey of neurologists regarding their current practice of CEEG monitoring in adults and children (excluding neonates) indicated that the majority perform CEEG monitoring for 1–2 days if no seizures occur, although there was substantial variability in practice.[2] These monitoring duration decisions are likely based on studies of critically ill children undergoing clinically indicated CEEG which report that half of patients with seizures are identified in the first hour of monitoring, and about 90% of patients with seizures are identified within the first 24 hours of monitoring [29, 31, 32, 35, 37, 38, 41, 42, 56]. However, none of these studies performed CEEG for an extended duration in all patients. Had CEEG monitoring duration continued for longer, seizures beginning later may have been identified in some patients.

Understanding the optimal duration of CEEG monitoring is important since seemingly small changes in monitoring duration have a substantial impact on resource needs [57]. Studies of children with specific types of acute brain injury and management may provide better focus. In a prospective study of 19 children undergoing therapeutic hypothermia after cardiac arrest, CEEG was initiated urgently and continued throughout the entire clinical protocol including 24 hours of normothermia. No children had ES in the initial six hours of monitoring (early hypothermia), one child had seizures in the next six hours (early hypothermia), four children had seizures during the next 12 hours (late hypothermia), and four children had seizures in the next 24 hours (re-warming) [36]. Similarly, in neonates and infants who underwent repair of congenital heart disease, studies have reported electrographic seizure at a mean of 21 hours [51, 52] and that most seizures occurred 13–36 hours after surgery.[49] These data suggest that with further study, CEEG duration may be tailored based on a patient's age, clinical status, and cause of acute encephalopathy.

Electrographic Seizures and Outcome

Two questions central to assessing the utility of CEEG are (1) does the occurrence of ES worsen outcome, and (2) does identification and management of ES improve outcome. The latter question has not yet been explored, but several recent studies have demonstrated an association between ES and worse neurodevelopmental outcome in critically ill children. In a study of 75 children, NCS were associated with higher mortality (15% versus 8%) and

neurologic morbidity (31% versus 4%).[38] In a second study of 204 critically ill comatose children and neonates, clinically-evident seizures, ES, a higher number and longer duration of ES, and a worse EEG background score were associated with worse outcome. In a multi-variable analysis, ES were associated with worse outcome (OR 15.4; 95% CI 4.7, 49.7). Furthermore, no children had favorable outcome if they had more than 139 seizures, more than 759 minutes of total seizures, or any individual seizure lasting longer than 360 minutes. [39] A third study evaluated short-term outcome in 200 prospectively enrolled critically ill children who underwent CEEG for altered mental status and an acute neurologic problem. Eighty-four children (42%) had seizures, which were categorized as ES in 41 (21%) and electrographic status epilepticus in 43 (22%). In multivariable analysis, electrographic status epilepticus was associated with an increased risk of mortality (OR 5.1; 95%CI 1.4, 18, $p=0.01$) and a decline in Pediatric Cerebral Performance Category (PCPC) (OR 17.3; 95%CI 3.7, 80, $p<0.001$), whereas ES were not associated with an increased risk of mortality (OR 1.3; 95%CI 0.3, 5.1; $p=0.74$) or decline in PCPC (OR 1.2; 95%CI 0.4, 3.9; $p=0.77$). This suggests that higher seizure burden is independently associated with worse outcome.[58] In a study of 154 children with status epilepticus, the presence of NCSE on initial EEG was associated with increased risk of refractory status epilepticus and increased risk of poor long-term outcome. Although children with NCSE were not evaluated independently, more aggressive management was associated with a better treatment response and outcome.[59]

Additional data on outcomes following seizures are available in the congenital heart disease population. A study of 164 infants with congenital heart disease with follow-up at one year identified abnormal neurological examination results in 11 of 15 (73%) patients with postoperative seizures versus 41 of 99 (41%) patients without seizures. Seizures were not associated with significantly lower scores on Bayley Scales of Infant Development, except for frontal-onset seizures which were associated with significantly lower Mental Development Index scores compared with non-frontal-onset seizures [60]. In a study of amplitude integrated EEG in infants undergoing Norwood-type procedures, both intraoperative and post-operative seizures were associated with higher mortality, but not associated with neurodevelopmental impairment [55]. An interesting set of studies have focused on children with D-transposition of the great arteries who underwent an arterial switch operation with successive neurodevelopmental assessments. In the initial study of 155 of 171 infants, early post-operative electrographic seizures were associated with an increased risk of MRI abnormalities and with an 11.2 point reduction in the psychomotor development index of the Bayley Scales of Infant Development at one year follow-up.[61] In a follow-up study that extended to 2.5 years, children with seizures were also more likely to have lower psychomotor development scores.[62] By four years, children with seizures had significantly lower mean intelligence quotient scores and increased risk of neurological abnormalities [63]. Among 139 children evaluated at adolescence, postoperative seizures were the medical variable most consistently associated with worse outcome.[64]

Further data are available from several adult studies. NCSE of longer duration has been associated with worse outcome.[25] NCS have been associated with worse discharge outcome in adults with central nervous system infections[65], intracerebral hemorrhage expansion[66] and death or severe neurologic disability in adults in the medical ICU.[67] In adults with traumatic brain injury, NCS have been associated with increases in intracranial pressure and metabolic dysfunction [68], as well as the development of ipsilateral hippocampal atrophy.[69]

In summary, although the evidence for an association between seizures and poor outcome is growing, further study is needed to more precisely define the causal link between ES and neurodevelopmental outcome, and establish whether identifying and managing ES improves outcomes.

Quantitative EEG for Electrographic Seizure Identification

Identifying seizures in critically ill children requires CEEG since most seizures in this population remain subclinical.[31, 35, 37–42, 70] Large numbers of critically ill children are at risk for subclinical seizures and stand to benefit from CEEG, however the availability of CEEG remains limited because of a scarcity of expert neurophysiologists required to interpret raw CEEG data. Furthermore, although CEEG is recorded continuously, real time review is seldom available, [2] potentially resulting in delays between seizure occurrence, seizure identification, and treatment.[71] When CEEG is being used to screen for NCS, neurologists report that it is reviewed once per day by 21%, twice per day by 29%, three or four times per day by 17%, and almost continuously by only 18%.[2]

Quantitative EEG (QEEG) algorithms separate the raw EEG into its component parts and compress several hours of EEG onto a single display. This technique may permit more rapid CEEG analysis by expert neurophysiologists and may facilitate seizure recognition by bedside caregivers without formal EEG training. Various QEEG tools, also referred to as digital trend analysis (DTA) since QEEG trends are displayed over time,[72] are now commercially available [73], but surveys indicate they are rarely used in current practice.[2]

Three commonly used QEEG tools are amplitude integrated EEG (aEEG), color density spectral array (CDSA or CSA), and envelope trend (ET). Because seizures typically contain higher frequency and higher amplitude activity than the background, seizures are apparent on aEEG and ET as arch-shaped elevations in the tracing (reflecting increased amplitude), and on CDSA as bright bands of color (reflecting increased power at higher frequencies) (Figure 1). Limitations of QEEG include missing seizures which are brief, low frequency or amplitude, or cover a small spatial area (particularly when a reduced number of channels are used). Conversely, high-amplitude or high-frequency artifacts may be misinterpreted as seizures on QEEG. Therefore, although QEEG may be used to identify regions of interest, review of the raw CEEG remains important to minimize over-interpretation.[72]

aEEG is currently used for prognostication and seizure identification in many neonatal ICUs,[74, 75] particularly among infants with hypoxic-ischemic encephalopathy undergoing therapeutic hypothermia.[76] While novice aEEG users identify seizures with a specificity of below 50%, experienced users can achieve a sensitivity and specificity of almost 85%. [77–80] However the sensitivity of aEEG for identifying individual seizures ranges from 12–96%.[78, 79, 81] Despite these imperfections, the use of aEEG can improve the precision of neonatal seizure diagnosis [82] and reduce ES burden.[83] However, with a false-positive rate using aEEG alone approaching 50%, [80] treatment decisions based solely on aEEG may result in over-diagnosis of seizures and over-use of anticonvulsants. The American Clinical Neurophysiology Society's Guidelines on EEG Monitoring in Neonates state that aEEG is a "useful, initial complementary tool" to CEEG, which remains the gold standard.[84]

Relatively few studies have investigated the utility of QEEG tools among critically ill non-neonatal children. In one study, the median sensitivity for seizure identification was 83% using CDSA and 82% using aEEG, but in individual EEG tracings sensitivity varied from 0–100%. False positive rates for both aEEG and CDSA were quite low. [85]. Another study applying CSA and ET demonstrated that sensitivity for seizure identification depends on user experience, display size, and inherent seizure characteristics, like seizure duration and spike amplitude [86]. In both of these studies, only brief training was required.

In summary, QEEG displays require minimal training to use and appear to have acceptable accuracy for seizure identification in some cases; however false-negatives and false-positives remain common enough that QEEG cannot yet replace review of conventional

EEG. Further work is required to optimize QEEG display parameters, for example by combining multiple QEEG trends to improve accuracy.[86, 87] Finally, although QEEG sensitivity remains imperfect, it must be remembered that inter-rater reliability for seizure identification using the “gold standard” conventional EEG is also imperfect [27, 88].

Practical Considerations

Performing CEEG in the PICU requires the collaboration of neurophysiologists, neurologists, EEG technologists, intensivists, nurses, and information technology specialists. Around-the-clock staffing may be required, EEG technologists must collaborate with critical care personnel, physicians may rely on EEG technologists to aid in frequent screening of EEG, and physicians may require more remote access.[89] A standard for competency from the American Society of Electroneurodiagnostic Technologists addresses both knowledge and procedural issues as they pertain to the EEG technologist. The standard indicates that advanced training and continuing education is required to ensure that the EEG technologist “attained the advanced level of technical knowledge and skills as well as the cognitive ability necessary to interact with the critical care patient and staff to ensure a high quality ICU/cEEG recording that provides reliable information about the continuous electrophysiology of the brain.”[90] Providing EEG technologists with the opportunity to learn and develop such skills is vital to the success of CEEG in the PICU.

PICUs are often staffed by a large number of nurses, many of whom have very little experience with EEG technology. Providing nursing education via formal lectures and informal bedside training during CEEG hook-up may encourage nurses to become active participants in the CEEG process, in turn improving the data obtained from CEEG. Collaboration between nurses and EEG technologists is critical in positioning patients, performing reactivity testing, and in avoiding and trouble-shooting artifacts. EEG interpretation is much improved when bedside caregivers communicate information regarding patient state, medication administration, and clinical events to EEG readers.

While more types of providers are being asked to assist in various aspects of CEEG data acquisition in the PICU, certain advances in technology have reduced the amount of time required from each provider. CEEG in the PICU is generally performed with disc electrodes applied with collodion adhesive, decreasing the need for frequent reapplication. Although conventional EEG electrodes are not compatible with CT or MRI, newly-available MRI and CT ‘friendly’ electrodes are reported to be safe and generate only minimal imaging artifact. Individual institutions must decide based on their practice and EEG staffing whether it is more time and cost efficient to remove and reapply electrodes when neuroimaging is needed or to employ imaging-friendly electrodes.

Generally, a full array of EEG electrodes is applied according to the international 10–20 system. Reduced electrode montages may reduce workload, but at least in adults can fail to identify some seizures.[91] Video recording time-locked to the EEG allows readers accurately identify and classify artifacts and permits detailed clinical-electrographic correlation of events, which may help bedside caregivers determine whether future events are epileptic or non-epileptic. The advent and modernization of networked EEG monitoring systems allows data to be conveyed via a hospital network to the main neurophysiology reading system, thereby allowing EEG readers the ability to review EEG data from computers throughout the hospital or remotely.

Since performing CEEG monitoring is resource intensive,[92] institutions need to adopt clinical pathways to guide appropriate CEEG utilization. Neurophysiologists generally need to review the CEEG data at least twice per day to provide the treatment team with up to date, actionable information. Since no guidelines or consensus statements currently exist

regarding CEEG in the PICU, each institution will have to determine the most effective and efficient ways in which they can utilize their own resources.

Historical Notes and Future Directions

A routine one-hour EEG captures only about 4% of a day's encephalographic data and unfortunately, unusual, odd, clinical "spells" and unexplained autonomic attacks rarely seem to occur during the regular daytime schedule of most EEG laboratories. In the not-so-distant era of paper EEG recording, long term EEG monitoring was rarely done since it required continuous, round-the-clock presence of a technologist at the bedside to judge the quality of the tracing and to continuously replenish the paper and refill and realign the ink pens. At the end of a 24 hour recording session, a small mountain of paper had to be laboriously reviewed and then stored on microfilm. The technical innovations of high speed digital electronic data processing, high capacity digital storage media and the ability to seamlessly fuse digital images of the patient simultaneously with the CEEG data stream has transformed the field by allowing continuous video EEG monitoring to be performed on a regular basis and by expediting review.

Now that the technical issues of long term conventional CEEG have largely been solved, a new set of challenging questions arise. Is there agreement among different investigators about the definitions used to characterize the results of CEEG? Which patients require long term EEG monitoring and how long should CEEG be continued? How can we most efficiently and rapidly identify ES? And most importantly, building upon the answers to all these questions, does CEEG alter management a way that improves long-term neurodevelopmental outcome?

Answering such questions will likely require large, prospective, multi-center studies. The Critical Care EEG Monitoring Consortium, which contains a pediatric subgroup, has been laying the groundwork for these studies for nearly a decade. Current work includes refinement of EEG terminology, which will be essential in performing studies across sites or with groups of EEG readers, and development of a centralized database that will lay the foundation for standardized multi-center data collection. Within about a year of initiation, the Pediatric Critical Care EEG Group has already described the current state of EEG monitoring in critically ill children [1] and performed a retrospective study of about 500 critically ill children who underwent CEEG.

Conclusions

ES are common among critically ill children with acute encephalopathy of diverse causes. The majority of ES would go unnoticed even with careful clinical observation, and therefore require CEEG for their identification. There is growing evidence from animal and human studies that ES may contribute to brain injury and worsen outcome. CEEG monitoring is an essential tool for advancing the care of critically ill children with encephalopathy. CEEG monitoring is required for accurate investigation of seizure epidemiology, seizure risk factors, optimal seizure management, and the impact of seizure identification on outcome.

Acknowledgments

N. S. Abend: NIH K23 Grant NS076550; K. E. Chapman: none; W. B. Gallentine: none; J. Goldstein: none; A. E. Hyslop: none; T. Loddenkemper: Supported by a Career Development Fellowship Award from Harvard Medical School and Boston Children's Hospital, the Program for Quality and Safety at Boston Children's Hospital, the Payer Provider Quality Initiative, The Epilepsy Foundation of America (EF-213583 and EF-213882), the Center for Integration of Medicine and Innovative Technology, the Epilepsy Therapy Project, and an investigator initiated research grant from Lundbeck; K. B. Nash: none; J. J. Riviello, Jr.: none; C. D. Hahn: SickKids Foundation, Canadian Institutes of Health Research, PSI Foundation.

References

1. Sanchez SM, Carpenter J, Chapman KE, et al. Pediatric ICU EEG Monitoring: Current Resources and Practice in the United States and Canada. *Journal of Clinical Neurophysiology*. In press A survey of large hospitals in the United States and Canada regarding EEG monitoring in the pediatric ICU. EEG monitoring use is increasing, and the main indication is identification of subclinical seizures.
2. Abend NS, Dlugos DJ, Hahn CD, et al. Use of EEG monitoring and management of non-convulsive seizures in critically ill patients: a survey of neurologists. *Neurocrit Care*. 2010; 12:382–9. [PubMed: 20198513]
3. Abend NS, Topjian AA, Gutierrez-Colina AM, et al. Impact of continuous EEG monitoring on clinical management in critically ill children. *Neurocrit Care*. 2011; 15:70–5. [PubMed: 20499208]
4. Firosh Khan S, Ashalatha R, Thomas SV, Sarma PS. Emergent EEG is helpful in neurology critical care practice. *Clin Neurophysiol*. 2005; 116:2454–9. [PubMed: 16122983]
5. Praline J, Grujic J, Corcia P, et al. Emergent EEG in clinical practice. *Clin Neurophysiol*. 2007; 118:2149–55. [PubMed: 17709288]
6. Bautista RE, Godwin S, Caro D. Incorporating abbreviated EEGs in the initial workup of patients who present to the emergency room with mental status changes of unknown etiology. *J Clin Neurophysiol*. 2007; 24:16–21. [PubMed: 17277572]
7. Varelas PN, Spanaki MV, Hacin-Bey L, et al. Emergent EEG: indications and diagnostic yield. *Neurology*. 2003; 61:702–4. [PubMed: 12963769]
8. Benbadis SR, Chen S, Melo M. What's shaking in the ICU? The differential diagnosis of seizures in the intensive care setting. *Epilepsia*. 2010; 51:2338–2340. [PubMed: 20738384]
9. Vespa PM, Nenov V, Nuwer MR. Continuous EEG monitoring in the intensive care unit: early findings and clinical efficacy. *J Clin Neurophysiol*. 1999; 16:1–13. [PubMed: 10082088]
10. Kilbride RD, Costello DJ, Chiappa KH. How seizure detection by continuous electroencephalographic monitoring affects the prescribing of antiepileptic medications. *Arch Neurol*. 2009; 66:723–8. [PubMed: 19506131]
11. Guerit JM, Amantini A, Amodio P, et al. Consensus on the use of neurophysiological tests in the intensive care unit (ICU): electroencephalogram (EEG), evoked potentials (EP), and electroneuromyography (ENMG). *Neurophysiol Clin*. 2009; 39:71–83. [PubMed: 19467437]
12. Abend NS, Licht DJ. Predicting outcome in children with hypoxic ischemic encephalopathy. *Pediatr Crit Care Med*. 2008; 9:32–9. [PubMed: 18477911]
13. Nishisaki A, Sullivan J 3rd, Steger B, et al. Retrospective analysis of the prognostic value of electroencephalography patterns obtained in pediatric in-hospital cardiac arrest survivors during three years. *Pediatr Crit Care Med*. 2007; 8:10–7. [PubMed: 17251876]
14. Kessler S, Topjian AA, Gutierrez-Colina AM, et al. Short-term outcome prediction by electroencephalographic features in children treated with therapeutic hypothermia after cardiac arrest. *Neurocritical Care*. 2011; 14:37–43. [PubMed: 20890677]
15. Kravljanc R, Jovic N, Djuric M, et al. Outcome of status epilepticus in children treated in the intensive care unit: a study of 302 cases. *Epilepsia*. 2011; 52:358–63. [PubMed: 21269297]
16. Pampiglione G, Harden A. Resuscitation after cardiocirculatory arrest. Prognostic evaluation of early electroencephalographic findings. *Lancet*. 1968; 1:1261–5. [PubMed: 4172132]
17. Tasker RC, Boyd S, Harden A, Matthew DJ. Monitoring in non-traumatic coma. Part II: Electroencephalography. *Arch Dis Child*. 1988; 63:895–9. [PubMed: 3415324]
18. Cheliout-Heraut F, Sale-Franque F, Hubert P, Bataille J. Cerebral anoxia in near-drowning of children. The prognostic value of EEG. *Neurophysiol Clin*. 1991; 21:121–32. [PubMed: 1921939]
19. Ramachandran R, Sharma R, Weiss SK, Cortez MA. Reactive EEG patterns in pediatric coma. *Pediatr Neurol*. 2005; 33:345–9. [PubMed: 16243222]
20. Mandel R, Martinot A, Delepouille F, et al. Prediction of outcome after hypoxic-ischemic encephalopathy: a prospective clinical and electrophysiologic study. *J Pediatr*. 2002; 141:45–50. [PubMed: 12091850]

21. Pampiglione G, Chaloner J, Harden A, O'Brien J. Transitory ischemia/anoxia in young children and the prediction of quality of survival. *Ann N Y Acad Sci.* 1978; 315:281–92. [PubMed: 284740]
22. Evans BM, Bartlett JR. Prediction of outcome in severe head injury based on recognition of sleep related activity in the polygraphic electroencephalogram. *J Neurol Neurosurg Psychiatry.* 1995; 59:17–25. [PubMed: 7608703]
23. Rossetti AO, Carrera E, Oddo M. Early EEG correlates of neuronal injury after brain anoxia. *Neurology.* 2012; 78:796–802. [PubMed: 22323758]
24. Chong DJ, Hirsch LJ. 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; 22:79–91. [PubMed: 15805807]
25. Young GB, Jordan KG, Doig GS. An assessment of nonconvulsive seizures in the intensive care unit using continuous EEG monitoring: an investigation of variables associated with mortality. *Neurology.* 1996; 47:83–9. [PubMed: 8710130]
26. Drislane FW. Presentation, Evaluation, and Treatment of Nonconvulsive Status Epilepticus. *Epilepsy Behav.* 2000; 1:301–314. [PubMed: 12609161]
27. Ronner HE, Ponten SC, Stam CJ, Uitdehaag BM. Inter-observer variability of the EEG diagnosis of seizures in comatose patients. *Seizure.* 2009; 18:257–63. [PubMed: 19046902]
28. Cross JH. When is epileptic encephalopathy nonconvulsive status epilepticus? *Epilepsia.* 2007; 48 (Suppl 8):42–3. [PubMed: 18329996]
29. Abend NS, Gutierrez-Colina AM, Topjian AA, et al. Non-convulsive seizures are common in critically ill children. *Neurology.* 2011; 76:1071–1077. [PubMed: 21307352]
30. Hosain SA, Solomon GE, Kobylarz EJ. Electroencephalographic patterns in unresponsive pediatric patients. *Pediatr Neurol.* 2005; 32:162–5. [PubMed: 15730895]
31. 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. [PubMed: 17172615]
32. Abend NS, Dlugos DJ. Nonconvulsive status epilepticus in a pediatric intensive care unit. *Pediatr Neurol.* 2007; 37:165–70. [PubMed: 17765803]
33. Alehan FK, Morton LD, Pellock JM. Utility of electroencephalography in the pediatric emergency department. *J Child Neurol.* 2001; 16:484–7. [PubMed: 11453443]
34. Tay SK, Hirsch LJ, Leary L, et al. Nonconvulsive status epilepticus in children: clinical and EEG characteristics. *Epilepsia.* 2006; 47:1504–9. [PubMed: 16981867]
35. Shahwan A, Bailey C, Shekerdemian L, Harvey AS. The prevalence of seizures in comatose children in the pediatric intensive care unit: A prospective video-EEG study. *Epilepsia.* 2010; 51:1198–1204. [PubMed: 20163439]
36. Abend NS, Topjian A, Ichord R, et al. Electroencephalographic monitoring during hypothermia after pediatric cardiac arrest. *Neurology.* 2009; 72:1931–1940. [PubMed: 19487651]
37. Williams K, Jarrar R, Buchhalter J. Continuous video-EEG monitoring in pediatric intensive care units. *Epilepsia.* 2011; 52:1130–6. [PubMed: 21671924]
38. Greiner HM, Holland K, Leach JL, et al. Nonconvulsive status epilepticus: the encephalopathic pediatric patient. *Pediatrics.* 2012; 129:e748–55. [PubMed: 22331332]
39. Kirkham FJ, Wade AM, McElduff F, et al. Seizures in 204 comatose children: incidence and outcome. *Intensive Care Med.* 2012; 38:853–62. A study of 204 critically ill comatose children and neonates who underwent EEG monitoring. Worse outcome was associated with clinically evident seizures, electrographic seizures, higher number and longer duration electrographic seizures, and a worse EEG background score. Multivariate analysis indicated even after accounting for some variables related to encephalopathy etiology and severity, electrographic seizures were associated with worse outcome. [PubMed: 22491938]
40. Abend NS, Gutierrez-Colina AM, Topjian AA, et al. Nonconvulsive seizures are common in critically ill children. *Neurology.* 2011; 76:1071–7. [PubMed: 21307352]
41. McCoy B, Sharma R, Ochi A, et al. Predictors of nonconvulsive seizures among critically ill children. *Epilepsia.* 2011; 52:1973–8. [PubMed: 22003955]

42. Schreiber JM, Zelleke T, Gaillard WD, et al. Continuous video EEG for patients with acute encephalopathy in a pediatric intensive care unit. *Neurocrit Care*. 2012; 17:31–8. [PubMed: 22565632]
43. Dan B, Boyd SG. Nonconvulsive (Dialeptic) Status Epilepticus in Children. *Current Pediatric Reviews*. 2005; 1:7–16.
44. Claassen J, Mayer SA, Kowalski RG, et al. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. *Neurology*. 2004; 62:1743–8. [PubMed: 15159471]
45. Abend NS, Beslow LA, Smith SE, et al. Seizures as a Presenting Symptom of Acute Arterial Ischemic Stroke in Childhood. *J Pediatr*. 2011; 159:479–483. [PubMed: 21429519]
46. Beslow-Kaye LA, Abend NS, Gindville MC, et al. Pediatric Intracerebral Hemorrhage: Acute Symptomatic Seizures and Epilepsy. *Archives of Neurology*. In press.
47. Hahn JS, Vaucher Y, Bejar R, Coen RW. Electroencephalographic and neuroimaging findings in neonates undergoing extracorporeal membrane oxygenation. *Neuropediatrics*. 1993; 24:19–24. [PubMed: 8474607]
48. Horan M, Azzopardi D, Edwards AD, et al. Lack of influence of mild hypothermia on amplitude integrated-electroencephalography in neonates receiving extracorporeal membrane oxygenation. *Early Hum Dev*. 2007; 83:69–75. [PubMed: 16814962]
49. Helmers SL, Wypij D, Constantinou JE, et al. Perioperative electroencephalographic seizures in infants undergoing repair of complex congenital cardiac defects. *Electroencephalogr Clin Neurophysiol*. 1997; 102:27–36. [PubMed: 9060852]
50. Clancy RR, McGaurn SA, Wernovsky G, et al. Risk of seizures in survivors of newborn heart surgery using deep hypothermic circulatory arrest. *Pediatrics*. 2003; 111:592–601. [PubMed: 12612242]
51. Gaynor JW, Nicolson SC, Jarvik GP, et al. Increasing duration of deep hypothermic circulatory arrest is associated with an increased incidence of postoperative electroencephalographic seizures. *J Thorac Cardiovasc Surg*. 2005; 130:1278–86. [PubMed: 16256779]
52. Clancy RR, Sharif U, Ichord R, et al. Electrographic neonatal seizures after infant heart surgery. *Epilepsia*. 2005; 46:84–90. [PubMed: 15660772]
53. Chock VY, Reddy VM, Bernstein D, Madan A. Neurologic events in neonates treated surgically for congenital heart disease. *J Perinatol*. 2006; 26:237–42. [PubMed: 16496014]
54. Schmitt B, Finckh B, Christen S, et al. Electroencephalographic changes after pediatric cardiac surgery with cardiopulmonary bypass: is slow wave activity unfavorable? *Pediatr Res*. 2005; 58:771–8. [PubMed: 16189208]
55. Gunn JK, Beca J, Penny DJ, et al. Amplitude-integrated electroencephalography and brain injury in infants undergoing Norwood-type operations. *Ann Thorac Surg*. 2012; 93:170–6. [PubMed: 22075220]
56. Hyllienmark L, Amark P. Continuous EEG monitoring in a paediatric intensive care unit. *Eur J Paediatr Neurol*. 2007; 11:70–5. [PubMed: 17188917]
57. Gutierrez-Colina AM, Topjian AA, Dlugos DJ, Abend NS. EEG Monitoring in Critically Ill Children: Indications and Strategies. *Pediatric Neurology*. 2012; 46:158–161. [PubMed: 22353290]
58. Topjian AA, Gutierrez-Colina AM, Sanchez SM, et al. Electrographic Status Epilepticus is Associated with Mortality and Worse Short-Term Outcome in Critically Ill Children. *Critical Care Medicine*. In press A prospective study of critically ill children who underwent EEG monitoring for altered mental status and an acute neurologic problem. Electrographic seizures occurred in 21% and electrographic status epilepticus occurred in 22%. Multivariate analysis indicated even after accounting for some variables related to encephalopathy etiology and severity, electrographic status epilepticus (but not electrographic seizures) was associated with an increased risk of mortality and neurologic morbidity.
59. Lambrechtsen FA, Buchhalter JR. Aborted and refractory status epilepticus in children: a comparative analysis. *Epilepsia*. 2008; 49:615–25. [PubMed: 18093148]
60. Gaynor JW, Jarvik GP, Bernbaum J, et al. The relationship of postoperative electrographic seizures to neurodevelopmental outcome at 1 year of age after neonatal and infant cardiac surgery. *J Thorac Cardiovasc Surg*. 2006; 131:181–9. [PubMed: 16399310]

61. Bellinger DC, Jonas RA, Rappaport LA, et al. Developmental and neurologic status of children after heart surgery with hypothermic circulatory arrest or low-flow cardiopulmonary bypass. *N Engl J Med.* 1995; 332:549–55. [PubMed: 7838188]
62. Rappaport LA, Wypij D, Bellinger DC, et al. Relation of seizures after cardiac surgery in early infancy to neurodevelopmental outcome. Boston Circulatory Arrest Study Group. *Circulation.* 1998; 97:773–9. [PubMed: 9498541]
63. Bellinger DC, Wypij D, Kuban KC, et al. Developmental and neurological status of children at 4 years of age after heart surgery with hypothermic circulatory arrest or low-flow cardiopulmonary bypass. *Circulation.* 1999; 100:526–32. [PubMed: 10430767]
64. Bellinger DC, Wypij D, Rivkin MJ, et al. Adolescents with d-transposition of the great arteries corrected with the arterial switch procedure: neuropsychological assessment and structural brain imaging. *Circulation.* 2011; 124:1361–9. Long-term follow-up of prospectively enrolled children with D-transposition of the great arteries who underwent an arterial switch operation and postoperative EEG monitoring with successive neurodevelopmental assessments. The presence of postoperative electrographic seizures was the medical variable most consistently associated with worse outcome when evaluated in follow-up as adolescents. [PubMed: 21875911]
65. Carrera E, Claassen J, Oddo M, et al. Continuous electroencephalographic monitoring in critically ill patients with central nervous system infections. *Arch Neurol.* 2008; 65:1612–8. [PubMed: 19064748]
66. Claassen J, Jette N, Chum F, et al. Electrographic seizures and periodic discharges after intracerebral hemorrhage. *Neurology.* 2007; 69:1356–65. [PubMed: 17893296]
67. Oddo M, Carrera E, Claassen J, et al. Continuous electroencephalography in the medical intensive care unit. *Crit Care Med.* 2009; 37:2051–6. [PubMed: 19384197]
68. Vespa PM, Miller C, McArthur D, et al. Nonconvulsive electrographic seizures after traumatic brain injury result in a delayed, prolonged increase in intracranial pressure and metabolic crisis. *Crit Care Med.* 2007; 35:2830–6. [PubMed: 18074483]
69. Vespa PM, McArthur DL, Xu Y, et al. Nonconvulsive seizures after traumatic brain injury are associated with hippocampal atrophy. *Neurology.* 2010; 75:792–8. [PubMed: 20805525]
70. Gwer S, Idro R, Fegan G, et al. Continuous EEG monitoring in Kenyan children with non-traumatic coma. *Arch Dis Child.* 2012; 97:343–9. [PubMed: 22328741]
71. Trevathan E, Ellen R. Grass Lecture: Rapid EEG analysis for intensive care decisions in status epilepticus. *Am J Electroneurodiagnostic Technol.* 2006; 46:4–17. [PubMed: 16605169]
72. Riviello JJ. Digital trend analysis in the pediatric and neonatal intensive care units (ICU). *Journal of Clinical Neurophysiology.* In press.
73. Scheuer ML, Wilson SB. Data analysis for continuous EEG monitoring in the ICU: seeing the forest and the trees. *J Clin Neurophysiol.* 2004; 21:353–78. [PubMed: 15592009]
74. Boylan G, Burgoyne L, Moore C, et al. An international survey of EEG use in the neonatal intensive care unit. *Acta Paediatr.* 2010; 99:1150–5. [PubMed: 20353503]
75. Ponnusamy V, Nath P, Bissett L, et al. Current availability of cerebral function monitoring and hypothermia therapy in UK neonatal units. *Arch Dis Child Fetal Neonatal Ed.* 2010; 95:F383–4. [PubMed: 20530100]
76. Filippi L, Catarzi S, Gozzini E, et al. Hypothermia for neonatal hypoxic-ischemic encephalopathy: may an early amplitude-integrated EEG improve the selection of candidates for cooling? *J Matern Fetal Neonatal Med.* 2012; 25:2171–6. [PubMed: 22506547]
77. Shellhaas RA, Soaita AI, Clancy RR. Sensitivity of amplitude-integrated electroencephalography for neonatal seizure detection. *Pediatrics.* 2007; 120:770–7. [PubMed: 17908764]
78. Lawrence R, Mathur A, Nguyen The Tich S, et al. A pilot study of continuous limited-channel aEEG in term infants with encephalopathy. *J Pediatr.* 2009; 154:835–41. e1. [PubMed: 19230897]
79. Frenkel N, Friger M, Meledin I, et al. Neonatal seizure recognition - Comparative study of continuous-amplitude integrated EEG versus short conventional EEG recordings. *Clin Neurophysiol.* 2011; 122:1091–7. [PubMed: 21216190]
80. Evans E, Koh S, Lerner J, et al. Accuracy of amplitude integrated EEG in a neonatal cohort. *Arch Dis Child Fetal Neonatal Ed.* 2010; 95:F169–73. [PubMed: 20444809]

81. Shah DK, Mackay MT, Lavery S, et al. Accuracy of bedside electroencephalographic monitoring in comparison with simultaneous continuous conventional electroencephalography for seizure detection in term infants. *Pediatrics*. 2008; 121:1146–54. [PubMed: 18519484]
82. Shellhaas RA, Barks AK. Impact of amplitude-integrated electroencephalograms on clinical care for neonates with seizures. *Pediatr Neurol*. 2012; 46:32–5. [PubMed: 22196488]
83. van Rooij LG, Toet MC, van Huffelen AC, et al. Effect of treatment of subclinical neonatal seizures detected with aEEG: randomized, controlled trial. *Pediatrics*. 2010; 125:e358–66. [PubMed: 20100767]
84. Shellhaas RA, Chang T, Tsuchida T, et al. The American Clinical Neurophysiology Society's Guideline on Continuous Electroencephalography Monitoring in Neonates. *J Clin Neurophysiol*. 2011
85. Stewart CP, Otsubo H, Ochi A, et al. Seizure identification in the ICU using quantitative EEG displays. *Neurology*. 2010; 75:1501–8. Study of the diagnostic accuracy of two quantitative EEG display tools for seizure identification in critically ill children by neurophysiologists. The median sensitivity for seizure identification across all recordings was 83% with color density spectral array and 82% with amplitude- integrated EEG, but for individual recordings the sensitivities ranged from 0–100%. False positive rates were low. [PubMed: 20861452]
86. Akman CI, Micic V, Thompson A, Riviello JJ Jr. Seizure detection using digital trend analysis: Factors affecting utility. *Epilepsy Res*. 2011; 93:66–72. [PubMed: 21146370]
87. Glaria AP, Murray A. Comparison of EEG monitoring techniques: an evaluation during cardiac surgery. *Electroencephalogr Clin Neurophysiol*. 1985; 61:323–30. [PubMed: 2411511]
88. Abend NS, Gutierrez-Colina AM, Zhao H, et al. Interobserver reproducibility of electroencephalogram interpretation in critically ill children. *Journal of Clinical Neurophysiology*. 2011; 28:15–19. [PubMed: 21221016]
89. Kull LL, Emerson RG. Continuous EEG monitoring in the intensive care unit: technical and staffing considerations. *J Clin Neurophysiol*. 2005; 22:107–18. [PubMed: 15805810]
90. American-Society-of-Electroneurodiagnostic-Technologists. National competency skill standards for ICU/cEEG monitoring. *Am J Electroneurodiagnostic Technol*. 2008; 48:258–64. [PubMed: 19203079]
91. Kolls BJ, Husain AM. Assessment of hairline EEG as a screening tool for nonconvulsive status epilepticus. *Epilepsia*. 2007; 48:959–65. [PubMed: 17433054]
92. Benbadis SR. Use and abuse of stat EEG. *Am J Electroneurodiagnostic Technol*. 2009; 49:87–93. [PubMed: 19388550]

Figure 1.

Appearance of seizures on a 4-hour quantitative EEG display. A) Timing of electrographic seizures identified by review of the raw EEG; B) Color Density Spectral Array (CDSA) trend depicts seizures as bright bands of color; C) Amplitude-integrated EEG (aEEG) trend depicts seizures as elevations in the lower and upper margins of the tracing. Note that not all electrographic seizures identified by raw EEG are equally recognizable on the CDSA or aEEG trends.

Table 1**Common Indications for CEEG Monitoring in the PICU**

Clinical scenarios in which CEEG monitoring should be considered
Refractory status epilepticus
Acute encephalopathy
With prior clinical seizures
With acute brain injury
Unexplained
Neuromuscular blockade, with acute brain injury
Characterization of clinical events suspected to be seizures
Intracranial pressure management
Specific diagnoses & treatments where CEEG monitoring should be considered
Status epilepticus
Traumatic brain injury (accidental, non-accidental)
Hypoxic-ischemic brain injury (neonatal, cardiac arrest, near drowning)
Extracorporeal membrane oxygenation (ECMO) therapy
Acute ischemic or hemorrhagic stroke
Post-cardiac surgery
Post-neurosurgery
Acute metabolic encephalopathy (sepsis, hepatic, renal)