

**UCLA**

**UCLA Previously Published Works**

**Title**

Consensus Statement on Continuous EEG in Critically Ill Adults and Children, Part I

**Permalink**

<https://escholarship.org/uc/item/10m75068>

**Journal**

Journal of Clinical Neurophysiology, 32(2)

**ISSN**

0736-0258

**Authors**

Herman, Susan T  
Abend, Nicholas S  
Bleck, Thomas P  
[et al.](#)

**Publication Date**

2015-04-01

**DOI**

10.1097/wnp.0000000000000166

Peer reviewed

# Consensus Statement on Continuous EEG in Critically Ill Adults and Children, Part I: Indications

Susan T. Herman,\* Nicholas S. Abend,† Thomas P. Bleck,‡ Kevin E. Chapman,§ Frank W. Drislane,\* Ronald G. Emerson,|| Elizabeth E. Gerard,¶ Cecil D. Hahn,# Aatif M. Husain,\*\*†† Peter W. Kaplan,‡‡ Suzette M. LaRoche,§§ Marc R. Nuwer,||| Mark Quigg,¶¶ James J. Riviello,### Sarah E. Schmitt,\*\*\* Liberty A. Simmons,††† Tammy N. Tsuchida,‡‡‡ and Lawrence J. Hirsch§§§

**Introduction:** Critical Care Continuous EEG (CCEEG) is a common procedure to monitor brain function in patients with altered mental status in intensive care units. There is significant variability in patient populations undergoing CCEEG and in technical specifications for CCEEG performance.

**Methods:** The Critical Care Continuous EEG Task Force of the American Clinical Neurophysiology Society developed expert consensus recommendations on the use of CCEEG in critically ill adults and children.

**Recommendations:** The consensus panel recommends CCEEG for diagnosis of nonconvulsive seizures, nonconvulsive status epilepticus, and other paroxysmal events, and for assessment of the efficacy of therapy for seizures and status epilepticus. The consensus panel suggests CCEEG for identification of ischemia in patients at high risk for cerebral ischemia; for assessment of level of consciousness in patients receiving intravenous sedation or pharmacologically induced coma; and for prognostication in patients after cardiac arrest. For each indication, the consensus panel describes the patient populations for which CCEEG is

indicated, evidence supporting use of CCEEG, utility of video and quantitative EEG trends, suggested timing and duration of CCEEG, and suggested frequency of review and interpretation.

**Conclusion:** CCEEG has an important role in detection of secondary injuries such as seizures and ischemia in critically ill adults and children with altered mental status.

**Key Words:** EEG, EEG monitoring, Quantitative EEG, Seizure, Nonconvulsive seizure, Status epilepticus, Nonconvulsive status, epilepticus, Intensive care unit, Critical care, Adults, Children.

(*J Clin Neurophysiol* 2015;32: 87–95)

Critically ill patients are at high risk for a variety of neurologic insults, including seizures, ischemia, edema, infection, and increased intracranial pressure, which can result in permanent neurologic disability if untreated. Despite these risks, there are few techniques for continuously monitoring brain function. EEG measures the brain's electrical activity, can be recorded continuously at the bedside, has good spatial and excellent temporal resolution, and is sensitive to changes in both brain structure and function (Nuwer, 1994). Over the past decade, technical advances have improved the efficiency of continuous EEG (CEEG) recording and remote review, leading to a greater than fourfold increase in the number of CEEGs performed in intensive care units (ICUs) (Ney et al., 2013). Recent surveys, however, show variability in why and how CEEG is performed in the ICU (Abend et al., 2010; Gavvala et al., 2014; Sanchez et al., 2013a), highlighting the need for clinical guidance on this expensive and labor-intensive procedure.

Critical care continuous EEG (CCEEG) refers to the simultaneous recording of EEG and clinical behavior (video) over extended time periods (hours to weeks) in critically ill patients at risk for secondary brain injury and neurologic deterioration. Critical care continuous EEG is usually performed in an ICU setting, but this varies by hospital and some patients may be in step-down units or general medical or surgical units. Critical care continuous EEG typically includes simultaneous video recording and may include graphical displays of quantitative EEG (QEEG) trends. The goal of CCEEG is to identify changes in brain function, such as nonconvulsive seizures (NCS) or ischemia, which may not be evident by neurological examination alone, to facilitate early identification and management of these abnormalities.

This consensus statement applies only to critically ill adult and pediatric patients. It does not apply to long-term monitoring of awake and alert patients with epilepsy, sleep monitoring, or intraoperative monitoring. Separate recommendations have been developed by the

From the \*Comprehensive Epilepsy Program, Department of Neurology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, U.S.A.; †Departments of Neurology and Pediatrics, Perelman School of Medicine, Children's Hospital of Philadelphia, University of Pennsylvania, Philadelphia, Pennsylvania, U.S.A.; ‡Departments of Neurological Sciences, Neurosurgery, Medicine, and Anesthesiology, Rush Medical College, Chicago, Illinois, U.S.A.; §Department of Pediatrics and Neurology, University of Colorado, Boulder, Colorado, U.S.A.; ||Department of Neurology, Hospital for Special Surgery, New York-Presbyterian Hospital/Weill Cornell Medical College, New York, New York, U.S.A.; ¶Department of Neurology, Feinberg School of Medicine, Northwestern University, Chicago, Illinois, U.S.A. #Division of Neurology, Department of Paediatrics, The Hospital for Sick Children, University of Toronto, Toronto, ON, Canada; \*\*Department of Neurology, Duke University Medical Center, Durham, North Carolina, U.S.A.; ††Neurodiagnostic Center, Veterans Affairs Medical Center, Durham, North Carolina, U.S.A.; ‡‡Department of Neurology, Johns Hopkins Bayview Medical Center, Baltimore, Maryland, U.S.A.; §§Department of Neurology, Emory University School of Medicine, Atlanta, Georgia, U.S.A.; |||Department of Neurology, David Geffen School of Medicine and Clinical Neurophysiology, Ronald Reagan UCLA Medical Center, Los Angeles, California, U.S.A.; ¶¶FE Dreibuss Comprehensive Epilepsy Program, Department of Neurology, University of Virginia, Charlottesville, Virginia, U.S.A.; ###NYU Division of Child Neurology, Columbia University Medical Center, New York, New York, U.S.A.; \*\*\*Department of Neurology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, U.S.A.; †††School of Clinical Neurophysiology, Crozer-Chester Medical Center, Upland, Pennsylvania, U.S.A.; ‡‡‡Department of Neurology and Pediatrics, Children's National Medical Center, George Washington University School of Medicine, Washington, District of Columbia, U.S.A.; and §§§Department of Neurology, Yale Comprehensive Epilepsy Center, Yale School of Medicine, New Haven, Connecticut, U.S.A.

Address correspondence and reprint requests to Susan T. Herman, MD, Beth Israel Deaconess Medical Center, Baker 5, 330 Brookline Avenue, Boston, MA 02215, U.S.A.; e-mail: sherman2@bidmc.harvard.edu.

Copyright © 2015 by the American Clinical Neurophysiology Society  
ISSN: 0736-0258/15/3202-0087

American Clinical Neurophysiology Society (ACNS) for CCEEG in critically ill neonates (Shellhaas et al., 2011).

The ACNS CCEEG Task Force describes a variety of models for CCEEG. Some techniques are available in only a few specialized centers and represent an “idealized” system for CCEEG. The committee recognizes that many CCEEG programs do not have full access to all equipment, technical staff, and interpreting staff described below but should use these recommendations as a guide for program development and improvement. Each center should provide CCEEG at the highest level that local resources allow. Transferring patients to more specialized centers should be considered when local resources are insufficient for patient care needs and when the advantages of CCEEG outweigh the potential risks of transfer. Critical care continuous EEG is often requested as an urgent or emergency study in critically ill patients. Current staffing models may not support 24/7 in-house neurodiagnostic technologists (NDTs). This consensus statement therefore addresses minimum techniques for CCEEG under emergency circumstances, as well as optimal techniques when qualified NDTs are available.

Critical care continuous EEG is longer than routine EEG, but the required duration varies depending on individual patient characteristics, indications for monitoring and EEG findings. For most indications, recording for a minimum of 24 hours is recommended, but shorter or longer recording may be needed for selected populations (see INDICATIONS FOR CRITICAL CARE CONTINUOUS EEG). To optimally identify neurological deterioration in critically ill patients, CCEEG should be started as soon as feasible in selected patient groups with acute brain injuries, altered mental status, or risk for brain ischemia (see INDICATIONS FOR CRITICAL CARE CONTINUOUS EEG). Subsequent CCEEG recordings can then be compared with this initial “baseline” recording to identify secondary neurological insults.

Part I of this consensus statement describes the most common indications for CCEEG in adults and children. Part II covers technical aspects of CCEEG, such as qualifications of personnel performing and interpreting CCEEG, equipment, documentation, and safety. Part II also addresses commonly used CCEEG techniques for specific indications in adults and children.

Critical care continuous EEG is a rapidly evolving technology, and this statement addresses only current consensus-based recommendations for CCEEG. At this time, there is inadequate data on the impact of CCEEG on clinical outcomes to develop practice standards based on strong evidence, but existing evidence is summarized within this document. Because NCS and other secondary brain injuries are often completely unrecognized without CCEEG, this document emphasizes that delayed recognition is better than no recognition. In particular, the term “monitoring” usually does not imply continuous real-time analysis and reporting of the EEG. Due to resource limitations, CCEEG is typically *acquired continuously* and *reviewed intermittently* by NDTs for technical quality and changes in EEG patterns and also intermittently by electroencephalographers for interpretation and clinical correlation. The decision to initiate CCEEG, frequency of review, and communication of results to ICU caregivers are determined by local resources, local monitoring indications, CCEEG findings, and the patient’s clinical status.

## METHODS

The Critical Care Continuous EEG Task Force was assembled by ACNS to address clinical use of continuous video-EEG

monitoring in critically ill adults and nonneonatal children. Initial review of the literature identified no randomized trials examining the impact of CCEEG on seizure burden or patient outcomes; observational trials were often small, retrospective, and subject to bias. Since only low- or very low-quality evidence was available for most areas of CCEEG, a consensus statement was determined to be more appropriate than evidence-based guidelines.

The Task Force convened at annual ACNS meetings and conferred by conference call and e-mail. Agreements were achieved through iterative discussion and debate. Recommendations were unanimously agreed upon before approval by ACNS Council. Recommendations are based on expert opinion and should not be used for performance measurements or competency purposes.

## INDICATIONS FOR CRITICAL CARE CONTINUOUS EEG

### Diagnosis of Nonconvulsive Seizures, Nonconvulsive Status Epilepticus, and Other Paroxysmal Events

1. Critical care continuous EEG is *recommended* to identify NCS and nonconvulsive status epilepticus (NCSE) in critically ill patients with the following:
  - a. Persistently abnormal mental status following generalized convulsive status epilepticus (GCSE) or other *clinically-evident seizures*: After apparently successful treatment of GCSE, many patients remain comatose, obtunded, or confused (Treiman et al., 1998). During 24 hours of CCEEG after GCSE, NCS were recorded in 48% and NCSE in 14% (DeLorenzo et al., 1998). Similarly, NCS were seen in 43% of patients who had convulsive seizures before monitoring (Claassen et al., 2004a). Children with convulsive seizures (Abend et al., 2013a; Greiner et al., 2012; McCoy et al., 2011) or GCSE (Williams et al., 2011) before CCEEG are at higher risk for NCS. Thirty-three percent of 98 children undergoing CCEEG after GCSE terminated had ongoing electrographic seizures (Sanchez Fernandez et al., 2014). Impaired consciousness after clinical seizures end can be secondary to prolonged postictal effects, sedative effects of antiseizure drugs (ASDs), or continued NCS. If a patient is not showing clear signs of improvement alertness within 10 minutes or still has any impairment of consciousness for more than 30 minutes after cessation of motor or other clinically-evident seizure activity, CCEEG should be considered to assess for ongoing seizure activity (Brophy et al., 2012; Claassen et al., 2013b).
  - b. *Acute supratentorial brain injury with altered mental status*: Table 1 lists types of acute brain injuries in which NCS are commonly seen, including traumatic brain injury, subarachnoid hemorrhage, intracerebral hemorrhage, encephalitis, acute ischemic stroke, and during and after therapeutic hypothermia following cardiac arrest (Claassen et al., 2013b). Patients younger than 18 years may be at higher risk than adults for NCS and NCSE (Claassen et al., 2004a), and within the pediatrics age group, neonates and infants may be at higher risk than older children (Abend and Dlugos, 2007; Abend et al., 2009, 2011b, 2013b; Arndt et al., 2013; Greiner et al., 2012; Hasbani et al., 2013; Hosain et al., 2005; Jette et al., 2006; Kirkham et al., 2012; McCoy et al., 2011;

**TABLE 1.** Common Neurological, Medical, and Surgical Conditions Associated With High Likelihood of Recording Seizures on Critical Care Continuous EEG

	Adults	Children	References
Following convulsive status epilepticus	48%	26%–57%	Abend et al., 2011b, 2013b; DeLorenzo et al., 1998; Sanchez Fernandez et al., 2014; Tay et al., 2006; Williams et al., 2011
Aneurysmal subarachnoid hemorrhage	Any seizure: 10%–19% NCSE: 3%–13%		Claassen et al., 2004a, 2006, 2014; Dennis et al., 2002; Little et al., 2007; O'Connor et al., 2014; Westover et al., 2014
Intraparenchymal hemorrhage	16%–23%	11%–100%	Claassen et al., 2007; Greiner et al., 2012; Jette et al., 2006; Kirkham et al., 2012; Kurtz et al., 2014; McCoy et al., 2011; Payne et al., 2014; Saengpatrachai et al., 2006; Tay et al., 2006; Vespa et al., 2003; Westover et al., 2014
Moderate-to-severe traumatic brain injury	18%–33%	14%–70%	Abend et al., 2011b, 2013b; Arndt et al., 2013; Claassen et al., 2004a; Hasbani et al., 2013; Jette et al., 2006; Payne et al., 2014; Ronne-Engstrom and Winkler, 2006; Sanchez et al., 2013a; Schreiber et al., 2012; Vespa et al., 1999b; Williams et al., 2011
Central nervous system infections	10%–33%	16%–100%	Abend et al., 2011b, 2013b; Carrera et al., 2008; Claassen et al., 2004a; Gwer et al., 2012; Jette et al., 2006; Payne et al., 2014; Saengpatrachai et al., 2006; Schreiber et al., 2012; Tay et al., 2006; Westover et al., 2014; Williams et al., 2011
Recent neurosurgical procedures	23%	71%	Claassen et al., 2004a; Payne et al., 2014; Westover et al., 2014
Brain tumors	Any seizure: 23–37% NCSE: 9–12%	19%–66%	Abend et al., 2011b, 2013b; Greiner et al., 2012; Jette et al., 2006; Kirkham et al., 2012; Marcuse et al., 2014; Westover et al., 2014
Acute ischemic stroke	6%–27%	20%–71%	Abend et al., 2011b; Claassen et al., 2004a; Greiner et al., 2012; Jette et al., 2006; Kirkham et al., 2012; Kurtz et al., 2014; McCoy et al., 2011; Payne et al., 2014; Saengpatrachai et al., 2006; Sanchez et al., 2013b; Vespa et al., 2003; Westover et al., 2014
Hypoxic–ischemic injury following cardiac or respiratory arrest, with or without therapeutic hypothermia	10%–59%	16%–79%	Abend et al., 2009, 2011b, 2013b; Claassen et al., 2004a; Crepeau et al., 2013; Jette et al., 2006; Kawai et al., 2011; Knight et al., 2013; Legriel et al., 2013; Mani et al., 2012; Payne et al., 2014; Rittenberger et al., 2012; Sadaka et al., 2014; Sanchez et al., 2013a; Tay et al., 2006; Westover et al., 2014; Williams et al., 2011
Sepsis-associated encephalopathy	32%	58%	Abend et al., 2013a; Oddo et al., 2009
Extracorporeal membrane oxygenation		21%	Piantino et al., 2013
Epilepsy related	33%–39%	11%–71%	Abend et al., 2011b, 2013b; Claassen et al., 2004a; Hyllienmark and Amark, 2007; Jette et al., 2006; McCoy et al., 2011; Saengpatrachai et al., 2006; Tay et al., 2006; Westover et al., 2014; McCoy et al., 2011

NCSE, nonconvulsive status epilepticus.

Saengpatrachai et al., 2006; Schreiber et al., 2012; Shahwan et al., 2010; Tay et al., 2006; Williams et al., 2011).

- c. *Fluctuating mental status or unexplained alteration of mental status without known acute brain injury*: Mental status abnormalities can include agitation, lethargy, fixed or fluctuating neurologic deficits such as aphasia or neglect, obtundation, and coma. Nonconvulsive

seizures have been reported in 8% to 10% of patients with unexplained coma or altered consciousness who did not have prior clinical seizures (Kurtz et al., 2014; Oddo et al., 2009; Towne et al., 2000).

- d. *Generalized periodic discharges (GPDs), lateralized periodic discharges (LPDs), or bilateral independent periodic discharges (BIPDs) on routine or emergent EEG*: Adults and children with generalized or lateralized

periodic discharges are more likely to develop NCS or NCSE (Akman et al., 2013; Foreman et al., 2012; Gaspard et al., 2013; Jette et al., 2006; Ong et al., 2012; Pedersen et al., 2013). The presence of lateralized rhythmic delta activity (LRDA) seems to have the same high association with seizures as LPDs and is also a reasonable indication for CCEEG (Gaspard et al., 2013).

- e. *Requirement for pharmacological paralysis and risk for seizures* (e.g., therapeutic hypothermia protocols, extracorporeal membrane oxygenation). Paralytic agents will prevent any clinical manifestations of seizures, making CCEEG recording essential to identify seizures in high risk patients.
  - f. *Clinical paroxysmal events suspected to be seizures, to determine whether they are ictal or nonictal*: Critically ill adults and children may have a variety of episodic abnormal movements or other clinical events that raise concern for seizures (Benbadis et al., 2010; Boesebeck et al., 2010; Williams et al., 2011). Antiseizure drugs may be initiated for these events but carry a risk for sedation, hypersensitivity reactions, and other adverse effects including cardiac and respiratory dysfunction. Exclusion of seizures may prevent initiation of or facilitate withdrawal of unnecessary ASDs. Episodic events which may benefit from evaluation with video CCEEG include (1) motor movements such as subtle face or limb twitching, nystagmus, gaze deviation, eyelid fluttering, chewing, myoclonus, tremors, rigors, episodic posturing, and other paroxysmal or repetitive face, limb, or trunk movements; (2) paroxysmal autonomic spells such as unexplained apnea, tachycardia, flushing, or blood pressure changes; or (3) unexplained paroxysmal increases in intracranial pressure or lactate or lactate/pyruvate ratio on microdialysis. EEG may not identify seizures with a small field or deep location. Because only approximately 21% of simple partial seizures show changes on scalp EEG (Devinsky et al., 1988), a normal EEG during a clinical event does not exclude an ictal etiology. Intracranial EEG recordings of critically ill patients may show seizures that are not identified on the scalp EEG (Claassen et al., 2013a; Waziri et al., 2009). Although these intracranial seizures typically have no clinical manifestations, they may be associated with systemic effects including increases in blood pressure and heart rate (Claassen et al., 2013a).
2. Evidence supporting use of CCEEG to identify seizures: Evaluation for suspected NCS is the most common indication for CCEEG (Abend et al., 2010; Gavvala et al., 2014; Sanchez et al., 2013b). Nonconvulsive seizures, also called subclinical, electrographic-only, subtle, occult, or silent seizures, have minimal or no overt clinical signs and can only be reliably diagnosed using CCEEG. Nonconvulsive status epilepticus, in which NCS are prolonged or repetitive, is variably defined as NCS lasting more than 30 minutes or recurrent over 30 minutes without return to normal consciousness; continuous or recurrent NCS lasting more than 5 minutes (Brophy et al., 2012), and continuous or recurrent NCS for more than 50% of an EEG epoch.
    - a. Nonconvulsive seizures occur in 8% to 48% of critically ill adults (Claassen et al., 2004a; DeLorenzo et al., 1998; Jordan, 1995; Oddo et al., 2009; Pandian et al., 2004; Privitera et al., 1994; Towne et al., 2000; Vespa et al., 1999a) and 6% to 47% of children with altered mental status (Abend and Dlugos, 2007; Abend et al., 2009, 2011b, 2013b; Arndt et al., 2013; Greiner et al., 2012; Hasbani et al., 2013; Hosain et al., 2005; Jette et al., 2006; Kirkham et al., 2012; McCoy et al., 2011; Schreiber et al., 2012; Shahwan et al., 2010; Tay et al., 2006; Williams et al., 2011). Table 1 summarizes the percentage of critically ill patients with seizures by etiology.
    - b. Nonconvulsive seizures are associated with other signs of neurologic injury, such as increased intracranial pressure, increased edema and mass effect, changes in tissue oxygenation, and local increases in lactate, lactate/pyruvate ratio, and glutamate, suggesting that NCS play a role in secondary brain injury (Dreier et al., 2012; Fabricius et al., 2008; Hartings et al., 2011; Vespa et al., 1998, 1999b, 2002a, 2003, 2007).
    - c. Prolonged NCS or NCSE are associated with increased mortality and increased risk for poor neurologic outcome (Abend et al., 2013b; Claassen et al., 2014; Payne et al., 2014; Topjian et al., 2013; Wagenman et al., 2014; Young et al., 1996), so rapid diagnosis is encouraged. Seventy-nine percent of physicians responding to a survey of CCEEG practice responded that CCEEG should be initiated immediately if NCS or NCSE are suspected (Abend et al., 2010).
    - d. The use of CCEEG in ICU patients at risk for NCS leads to changes in treatment in the majority of both adults (Kilbride et al., 2009) and children (Abend et al., 2011a).
    - e. The impact of NCS identification and management on outcome has not yet been established, and may differ based on the NCS etiology, duration, and management approach.
  3. Assessment of clinical behavior: Concurrent video recording is *strongly recommended* as a supplement to the clinical examination. The CCEEG team should establish, by direct observation or video review, whether electrographic seizures are associated with discrete clinical changes. Testing at the bedside is superior to video for identification of subtle seizure manifestations, but video allows post hoc review of events which were not directly observed.
  4. Timing and duration: Critical care continuous EEG should be initiated as soon as possible when NCS are suspected, since prolonged NCS and NCSE are associated with higher morbidity and mortality and treatment is likely to be more effective earlier in the course (Brophy et al., 2012). The length of a CCEEG depends on the pretest probability for seizures and the patient's clinical course. Recording for at least 24 hours is *recommended*, but there may be situations in which shorter or longer periods of recording are necessary. Typical 30- to 60-minute EEG recordings identify NCS in only 45% to 58% of patients in whom seizures are eventually recorded (Abend et al., 2011b; Claassen et al., 2004a; Pandian et al., 2004). About 80% to 95% of patients with NCS can be identified within 24 to 48 hours (Abend and Dlugos, 2007; Abend et al., 2011b; Claassen et al., 2004a; Jette et al., 2006; Shahwan et al., 2010). In specific populations, such as patients who are comatose, have periodic discharges, or are pharmacologically sedated, NCS may occur later and more prolonged monitoring (48 hours or more) may be needed (Abend et al., 2009; Claassen et al., 2004a). Early EEG findings may help to refine the required period of recording (Westover et al., 2014). Patients without early epileptiform

- discharges (within the first 2 hours) had less than a 5% chance of seizures in the next 72 hours. Brief (30 minutes) serial EEGs have been demonstrated to have similar yield to CCEEG in adult postcardiac arrest patients undergoing hypothermia (Crepeau et al., 2014). Additional studies are needed to confirm the utility and cost effectiveness of CCEEG versus serial or briefer EEG in other populations.
5. Frequency of review and interpretation: Rapid diagnosis of NCS allows appropriate treatment to be initiated quickly. Optimally, CCEEG would be reviewed continuously by qualified personnel to identify seizures in real time, but current staffing models rarely support this level of monitoring. Critical care continuous EEG should be reviewed as often as logistically and technically feasible and interpreted by electroencephalographers at least twice daily (i.e., about every 12 hours). If frequent NCS or NCSE are identified, more frequent interpretation should be provided until seizures are controlled. If clinical events are recorded, CCEEG should be interpreted as soon as possible after the event to determine whether it was ictal or nonictal.

### Assessment of Efficacy of Therapy for Seizures and Status Epilepticus

1. Critical care continuous EEG is *recommended* to monitor the response of seizures and status epilepticus to treatment.
  - a. Nonconvulsive seizures and NCSE are common after apparently successful treatment of clinical seizures and status epilepticus (see 1a under Diagnosis of Nonconvulsive Seizures, Nonconvulsive Status Epilepticus, and Other Paroxysmal Events) and cannot be diagnosed without EEG.
  - b. For patients with refractory status epilepticus, CCEEG should be used to monitor the efficacy of continuous intravenous antiseizure drugs (cIV-ASDs) such as midazolam, propofol, or pentobarbital, for seizure suppression, burst suppression, or complete EEG suppression. Refractory status epilepticus is defined as clinical or electrographic seizures that continue after initial treatment for status epilepticus, typically with a benzodiazepine and at least one other acceptable ASD (Brophy et al., 2012).
  - c. Recurrence of altered consciousness in a patient with known NCS should prompt consideration of repeat CCEEG to exclude recurrent NCS.
2. Evidence: Critical care continuous EEG can confirm seizure cessation and absence of seizure recurrence. Most seizures during treatment with IV-ASDs are subclinical and would not be identified without CCEEG (Claassen et al., 2001; Claassen et al., 2002). Critical care continuous EEG can also be used to monitor the adequacy of burst suppression (duration of burst and interburst periods) or complete EEG suppression induced by cIV-ASDs (Jordan and Hirsch, 2006; Krishnamurthy and Drislane, 1999; Prins et al., 2007; Rossetti et al., 2011).
3. Assessment of clinical behavior: Concurrent video recording is *strongly recommended* as a supplement to the clinical examination. In addition to recording subtle clinical manifestations, bedside testing and/or video recording can help to document the response to treatment, such as improvement in mental status after administration of ASDs.

4. Timing and duration: Critical care continuous EEG should be initiated as soon as possible when persistent NCS are suspected after treatment of clinical seizures or status epilepticus. Critical care continuous EEG should be recorded until seizures have been controlled for at least 24 hours. Critical care continuous EEG should be recorded during the entire period that cIV-ASDs are used. Seizures may recur despite EEG-confirmed burst suppression or complete suppression (Claassen et al., 2001; Claassen et al., 2002), so intermittent monitoring for burst suppression alone may be insufficient to confirm complete seizure control. Because there is a high risk of seizure recurrence after withdrawal of cIV-ASDs, CCEEG is often continued for at least 24 hours after cIV-ASDs are withdrawn (Abend et al., 2010). For cIV-ASDs with long half-lives, more prolonged recording may be necessary, but the required duration of monitoring has not been standardized.
5. Frequency of review and interpretation: As in point 5 under Diagnosis of Nonconvulsive Seizures, Nonconvulsive Status Epilepticus, and Other Paroxysmal Events, rapid identification of NCS allows appropriate ASD treatment to be initiated quickly and may reduce morbidity and mortality associated with NCS and NCSE. Critical care continuous EEG should be reviewed as often as logistically and technically feasible, and interpreted by electroencephalographers at least twice daily (i.e., about every 12 hours). If frequent NCS or NCSE are identified, more frequent interpretation should be provided until seizures are controlled. If clinical events are recorded, CCEEG should be interpreted as soon as possible after the event to determine whether it was ictal or nonictal.

### Identification of Cerebral Ischemia

1. Critical care continuous EEG is *suggested* as an adjunct method to identify ischemia in patients at high risk for ischemia.
2. Evidence: During ischemia, EEG shows a progressive sequence of changes involving loss of fast activity followed by increasing slow activity (Jordan, 2004). Critical care continuous EEG, and particularly QEEG trends, can be used to identify changes in cortical perfusion before irreversible infarct occurs (Claassen et al., 2004b; Vespa et al., 1997).
  - a. EEG and QEEG techniques have been used to identify ischemia during neurosurgical and interventional neuroradiology vascular procedures (Ballotta et al., 2010; Botes et al., 2007; Laman et al., 2001; Mishra et al., 2011; Pinkerton, 2002; Plestis et al., 1997; Skordilis et al., 2011; van Putten et al., 2004).
  - b. Retrospective studies have shown that CCEEG and QEEG trends can identify delayed cerebral ischemia during vasospasm after subarachnoid hemorrhage, but no prospective studies have been performed (Claassen et al., 2004b; Vespa et al., 1997). Most centers using CCEEG for identification of vasospasm monitor patients at highest risk (severe subarachnoid hemorrhage with Hunt and Hess grades 3–5 or large amounts of cisternal blood, Fisher grade 3). Because EEG is nonspecific as to etiology of changes, CCEEG is typically used in conjunction with other ancillary testing (e.g., MRI or computer tomography perfusion or angiography, transcranial doppler ultrasounds or conventional angiography)

to identify delayed cerebral ischemia and may predict which patients are at risk for delayed cerebral ischemia earlier than other studies.

- c. Critical care continuous EEG holds promise for ischemia identification in patients with hemodynamic lesions and borderline flow or those at high risk for acute ischemic stroke (Sheorajpanday et al., 2009), but at this time, real-time identification of ischemia is usually not feasible as it requires continuous real-time analysis, ideally of the raw and QEEG. This may change as resources increase and automated EEG analysis improves, and if intracranial EEG recordings are used more often.
3. Assessment of clinical behavior: Concurrent video recording is recommended as a supplement to the clinical examination. Review of video can help to identify artifacts as well as changes in EEG and QEEG related to state changes.
4. Timing and duration: Critical care continuous EEG should be recorded during the period of time when the patient is at the highest risk for ischemia.
  - a. Subarachnoid hemorrhage: Critical care continuous EEG should be started before the highest risk window for vasospasm begins (approximately day 3 post-SAH) to establish a baseline recording. Ideally, this should be as soon as the aneurysm is secured. Critical care continuous EEG should be continued until the window for vasospasm has passed (day 14) or the patient is considered no longer at risk for vasospasm.
  - b. The optimal duration of monitoring for ischemia in other patient groups has not been established and should be individualized for the specific clinical situation. A practical guide would be to continue CCEEG during the highest risk window for ischemia (e.g., 24–48 hours in a patient with crescendo transient ischemic attack or 24 hours after carotid endarterectomy).
5. Frequency of review and interpretation: When CCEEG is performed for ischemia identification, review by CCEEG personnel should be frequent enough to allow therapeutic intervention to prevent or reverse ischemic insults if CCEEG identifies changes potentially related to ischemia. The optimal frequency of review has not been determined and may vary for different indications. For vasospasm, in which ischemia typically develops over several hours, CCEEG should be reviewed at least three times daily, whereas for patients at risk for acute ischemic stroke, more frequent review may be necessary, especially while the patient is asleep and clinical symptoms/signs may not be noted.
6. Because it is difficult to identify changes from ischemia on raw EEG over prolonged time periods, CCEEG for ischemia should include QEEG analysis, such as graphical displays of power ratios over time.

### Monitoring of Sedation and High-Dose Suppressive Therapy

1. Critical care continuous EEG is *suggested*, in conjunction with the clinical examination, to assess level of consciousness in patients requiring intravenous sedation or pharmacologically induced coma (Doenicke et al., 2007; Mirski and Hemstreet, 2007; Walder et al., 2001; Ypparila et al., 2002;

de Wit and Epstein, 2003). The most common use is monitoring burst suppression induced by pentobarbital in patients with increased intracerebral pressure (Hyllienmark and Amark, 2007) or refractory status epilepticus (see 2b under Assessment of Efficacy of Therapy for Seizures and Status Epilepticus). In pharmacologically induced coma, the goal is to optimize seizure suppression, burst suppression, or complete suppression while avoiding oversedation, hemodynamic complications, and other adverse effects.

2. Evidence: Once a patient is unresponsive, it can be very difficult to judge the degree of sedation on clinical grounds alone. Although some QEEG methods have been used for monitoring analgesia and sedation in the ICU (Sessler et al., 2008), these methods have not been validated in patients with neurologic dysfunction. Similarly, no studies have prospectively examined the likelihood of adverse effects based on the degree of EEG suppression.
3. Timing and duration: No studies have addressed the optimal timing or duration of CCEEG for monitoring of sedation or pharmacologically induced coma; these should be individualized based on patient status and indication for CCEEG.
4. Frequency of review and interpretation: No studies have addressed the optimal frequency of review and interpretation of CCEEG for monitoring of sedation or pharmacologically induced coma. Since patients are often also at risk for NCS, twice a day review may be considered.

### Assessment of Severity of Encephalopathy and Prognostication

1. EEG can help to predict outcome in several neurologic conditions, although it is unclear whether prolonged monitoring is superior to briefer EEG recordings performed at specific times after brain injury. In addition, most EEG parameters used to predict good outcome have a fairly high false-positive rate (i.e., EEG shows favorable pattern, but patient still has poor clinical outcome). Unfavorable prognostic factors include isoelectric pattern, burst suppression pattern, periodic patterns, and electrographic seizures (Synek, 1988, 1990; Young et al., 1999, 2004). Favorable prognostic features include background continuity, spontaneous variability, reactivity to stimulation, and presence of normal sleep patterns (Synek, 1988, 1990; Young et al., 1999, 2004). Clinical populations in which EEG may aid in prognosis include:
  - a. Severe traumatic brain injury, including abusive traumatic brain injury in infants (Stevens and Sutter, 2013; Vespa et al., 1999b, 2002b).
  - b. Hypoxic ischemic encephalopathy after cardiac arrest (without or with therapeutic hypothermia) (Kessler et al., 2011; Rossetti et al., 2010; Sandroni et al., 2013a; Sandroni et al., 2013b).
  - c. Subarachnoid hemorrhage (Claassen et al., 2006).
2. Evidence: Several grading systems have been developed to describe the severity of EEG abnormalities and aid in prognosis (Synek, 1990; Young et al., 2004). The EEG grade or degree of abnormality correlates fairly well with the level of consciousness, although EEG changes may precede or lag clinical changes. Serial or continuous studies may therefore be helpful when following disease evolution. Ensuring accurate clinical information is provided regarding

the medications being administered is essential because many medications can produce EEG changes that are identical to changes seen with brain injury.

- Timing and duration: No studies have addressed the optimal timing or duration of CCEEG for encephalopathy severity assessment or prognostication; these should be individualized based on patient status and indication for CCEEG. At this time, CCEEG has not been demonstrated to be of greater utility than standard EEG at specified time points (Rossetti et al., 2010).
- Frequency of review and interpretation: No studies have addressed the optimal frequency of review and interpretation of CCEEG when being used for assessment of encephalopathy or prognostication. Since patients are often also at risk for NCS, twice a day review may be considered.

## CONCLUSIONS

Critical care continuous EEG is an emerging technique to identify secondary brain injuries such as seizures and ischemia in critically ill patients. There is increasing evidence that these secondary injuries can worsen neurologic outcome, although no prospective studies have yet demonstrated that treatment of EEG-identified changes improves neurologic outcome. The most common indication for CCEEG is for identification of NCS and NCSE, with ischemia identification and prognostication as less common uses.

## ACKNOWLEDGMENTS

The authors wish to thank the Critical Care EEG Monitoring Research Consortium (CCEMRC) for the CCEEG research, which forms the basis of many of these recommendations and for helpful critiques of this statement.

## REFERENCES

- Abend NS, Dlugos DJ. Nonconvulsive status epilepticus in a pediatric intensive care unit. *Pediatr Neurol* 2007;37:165–170.
- Abend NS, Topjian A, Ichord R, et al. Electroencephalographic monitoring during hypothermia after pediatric cardiac arrest. *Neurology* 2009;72:1931–1940.
- 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;12:382–389.
- Abend NS, Topjian AA, Gutierrez-Colina AM, et al. Impact of continuous EEG monitoring on clinical management in critically ill children. *Neurocrit Care* 2011a;15:70–75.
- Abend NS, Gutierrez-Colina AM, Topjian AA, et al. Nonconvulsive seizures are common in critically ill children. *Neurology* 2011b;76:1071–1077.
- Abend NS, Wusthoff CJ, Goldberg EM, Dlugos DJ. Electrographic seizures and status epilepticus in critically ill children and neonates with encephalopathy. *Lancet Neurol* 2013a;12:1170–1179.
- Abend NS, Arndt DH, Carpenter JL, et al. Electrographic seizures in pediatric ICU patients: cohort study of risk factors and mortality. *Neurology* 2013b;81:383–391.
- Akman CI, Abou Khaled KJ, Segal E, et al. Generalized periodic epileptiform discharges in critically ill children: clinical features, and outcome. *Epilepsia Res* 2013;106:378–385.
- Arndt DH, Lerner JT, Matsumoto JH, et al. Subclinical early posttraumatic seizures detected by continuous EEG monitoring in a consecutive pediatric cohort. *Epilepsia* 2013;54:1780–1788.
- Ballotta E, Saladini M, Gruppo M, et al. Predictors of electroencephalographic changes needing shunting during carotid endarterectomy. *Ann Vasc Surg* 2010;24:1045–1052.
- 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.
- Boesebeck F, Freemann S, Kellinghaus C, Evers S. Misdiagnosis of epileptic and non-epileptic seizures in a neurological intensive care unit. *Acta Neurol Scand* 2010;122:189–195.
- Botes K, Le Roux DA, Van Marle J. Cerebral monitoring during carotid endarterectomy—a comparison between electroencephalography, transcranial cerebral oximetry and carotid stump pressure. *S Afr J Surg* 2007;45:43–46.
- Brophy GM, Bell R, Claassen J, et al. Guidelines for the evaluation and management of status epilepticus. *Neurocrit Care* 2012;17:3–23.
- Carrera E, Claassen J, Oddo M, et al. Continuous electroencephalographic monitoring in critically ill patients with central nervous system infections. *Arch Neurol* 2008;65:1612–1618.
- Claassen J, Hirsch LJ, Emerson RG, et al. Continuous EEG monitoring and midazolam infusion for refractory nonconvulsive status epilepticus. *Neurology* 2001;57:1036–1042.
- Claassen J, Hirsch LJ, Emerson RG, Mayer SA. Treatment of refractory status epilepticus with pentobarbital, propofol, or midazolam: a systematic review. *Epilepsia* 2002;43:146–153.
- Claassen J, Mayer SA, Kowalski RG, et al. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. *Neurology* 2004a;62:1743–1748.
- Claassen J, Hirsch LJ, Kreiter KT, et al. Quantitative continuous EEG for detecting delayed cerebral ischemia in patients with poor-grade subarachnoid hemorrhage. *Clin Neurophysiol* 2004b;115:2699–2710.
- Claassen J, Hirsch LJ, Frontera JA, et al. Prognostic significance of continuous EEG monitoring in patients with poor-grade subarachnoid hemorrhage. *Neurocrit Care* 2006;4:103–112.
- Claassen J, Jette N, Chum F, et al. Electrographic seizures and periodic discharges after intracerebral hemorrhage. *Neurology* 2007;69:1356–1365.
- Claassen J, Perotte A, Albers D, et al. Nonconvulsive seizures after subarachnoid hemorrhage: multimodal detection and outcomes. *Ann Neurol* 2013a;74:53–84.
- Claassen J, Taccone FS, Horn P, et al. Recommendations on the use of EEG monitoring in critically ill patients: consensus statement from the neurointensive care section of the ESICM. *Intensive Care Med* 2013b;39:1337–1351.
- Claassen J, Albers D, Schmidt JM, et al. Nonconvulsive seizures in subarachnoid hemorrhage link inflammation and outcome. *Ann Neurol* 2014;75:771–781.
- Crepeau AZ, Rabenstein AA, Fugate JE, et al. Continuous EEG in therapeutic hypothermia after cardiac arrest: prognostic and clinical value. *Neurology* 2013;80:339–344.
- Crepeau AZ, Fugate JE, Mandrekar J, et al. Value analysis of continuous EEG in patients during therapeutic hypothermia after cardiac arrest. *Resuscitation* 2014;85:785–789.
- de Wit M, Epstein SK. Administration of sedatives and level of sedation: comparative evaluation via the Sedation-Agitation Scale and the Bispectral Index. *Am J Crit Care* 2003;12:343–348.
- DeLorenzo RJ, Waterhouse EJ, Towne AR, et al. Persistent nonconvulsive status epilepticus after the control of convulsive status epilepticus. *Epilepsia* 1998;39:833–840.
- Dennis LJ, Claassen J, Hirsch LJ, et al. Nonconvulsive status epilepticus after subarachnoid hemorrhage. *Neurosurgery* 2002;51:1136–1143; discussion 1144.
- Devinsky O, Kelley K, Porter RJ, Theodore WH. Clinical and electroencephalographic features of simple partial seizures. *Neurology* 1988;38:1347–1352.
- Doenicke AW, Kugler J, Kochs E, et al. The Narcotrend monitor and the electroencephalogram in propofol-induced sedation. *Anesth Analg* 2007;105:982–992. table of contents.
- Dreier JP, Major S, Pannek HW, et al. Spreading convulsions, spreading depolarization and epileptogenesis in human cerebral cortex. *Brain* 2012;135:259–275.
- Fabricius M, Fuhr S, Willumsen L, et al. Association of seizures with cortical spreading depression and peri-infarct depolarisations in the acutely injured human brain. *Clin Neurophysiol* 2008;119:1973–1984.
- Foreman B, Claassen J, Abou Khaled K, et al. Generalized periodic discharges in the critically ill: a case-control study of 200 patients. *Neurology* 2012;79:1951–1960.
- Gaspard N, Manganas L, Rampal N, et al. Similarity of lateralized rhythmic delta activity to periodic lateralized epileptiform discharges in critically ill patients. *JAMA Neurol* 2013;70:1288–1295.
- Gavvala J, Abend N, LaRoche S, et al. Continuous EEG monitoring: a survey of neurophysiologists and neurointensivists. *Epilepsia* 2014;55:1864–1871.
- Greiner HM, Holland K, Leach JL, et al. Nonconvulsive status epilepticus: the encephalopathic pediatric patient. *Pediatrics* 2012;129:e748–e755.
- Gwer S, Idro R, Fegan G, et al. Continuous EEG monitoring in Kenyan children with non-traumatic coma. *Arch Dis Child* 2012;97:343–349.
- Hartings JA, Watanabe T, Bullock MR, et al. Spreading depolarizations have prolonged direct current shifts and are associated with poor outcome in brain trauma. *Brain* 2011;134:1529–1540.



- Hasbani DM, Topjian AA, Friess SH, et al. Nonconvulsive electrographic seizures are common in children with abusive head trauma. *Pediatr Crit Care Med* 2013;14:709–715.
- Hosain SA, Solomon GE, Kobylarz EJ. Electroencephalographic patterns in unresponsive pediatric patients. *Pediatr Neurol* 2005;32:162–165.
- Hyllienmark L, Amark P. Continuous EEG monitoring in a paediatric intensive care unit. *Eur J Paediatr Neurol* 2007;11:70–75.
- 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;63:1750–1755.
- Jordan KG. Neurophysiologic monitoring in the neuroscience intensive care unit. *Neurol Clin* 1995;13:579–626.
- Jordan KG. Emergency EEG and continuous EEG monitoring in acute ischemic stroke. *J Clin Neurophysiol* 2004;21:341–352.
- Jordan KG, Hirsch LJ. In nonconvulsive status epilepticus (NCSE), treat to burst-suppression: pro and con. *Epilepsia* 2006;47(suppl 1):41–45.
- Kawai M, Thapalia U, Verma A. Outcome from therapeutic hypothermia and EEG. *J Clin Neurophysiol* 2011;28:483–488.
- Kessler SK, Topjian AA, Gutierrez-Colina AM, et al. Short-term outcome prediction by electroencephalographic features in children treated with therapeutic hypothermia after cardiac arrest. *Neurocrit Care* 2011;14:37–43.
- Kilbride RD, Costello DJ, Chiappa KH. How seizure detection by continuous electroencephalographic monitoring affects the prescribing of antiepileptic medications. *Arch Neurol* 2009;66:723–728.
- Kirkham FJ, Wade AM, McElduff F, et al. Seizures in 204 comatose children: incidence and outcome. *Intensive Care Med* 2012;38:853–862.
- Knight WA, Hart KW, Adeoye OM, et al. The incidence of seizures in patients undergoing therapeutic hypothermia after resuscitation from cardiac arrest. *Epilepsy Res* 2013;106:396–402.
- Krishnamurthy KB, Drislane FW. Depth of EEG suppression and outcome in barbiturate anesthetic treatment for refractory status epilepticus. *Epilepsia* 1999;40:759–762.
- Kurtz P, Gaspard N, Wahl AS, et al. Continuous electroencephalography in a surgical intensive care unit. *Intensive Care Med* 2014;40:228–234.
- Laman DM, van der Reijden CS, Wieneke GH, et al. EEG evidence for shunt requirement during carotid endarterectomy: optimal EEG derivations with respect to frequency bands and anesthetic regimen. *J Clin Neurophysiol* 2001;18:353–363.
- Legriel S, Hilly-Ginoux J, Resche-Rigon M, et al. Prognostic value of electrographic postanoxic status epilepticus in comatose cardiac-arrest survivors in the therapeutic hypothermia era. *Resuscitation* 2013;84:343–350.
- Little AS, Kerrigan JF, McDougall CG, et al. Nonconvulsive status epilepticus in patients suffering spontaneous subarachnoid hemorrhage. *J Neurosurg* 2007;106:805–811.
- Mani R, Schmitt SE, Mazer M, et al. The frequency and timing of epileptiform activity on continuous electroencephalogram in comatose post-cardiac arrest syndrome patients treated with therapeutic hypothermia. *Resuscitation* 2012;83:840–847.
- Marcuse LV, Lancman G, Demopoulos A, Fields M. Nonconvulsive status epilepticus in patients with brain tumors. *Seizure* 2014;23:542–547.
- McCoy B, Sharma R, Ochi A, et al. Predictors of nonconvulsive seizures among critically ill children. *Epilepsia* 2011;52:1973–1978.
- Mirski MA, Hemstreet MK. Critical care sedation for neuroscience patients. *J Neurol Sci* 2007;261:16–34.
- Mishra M, Bandy M, Derakhshani R, et al. A quantitative EEG method for detecting post clamp changes during carotid endarterectomy. *J Clin Monit Comput* 2011;25:295–308.
- Ney JP, van der Goes DN, Nuwer MR, et al. Continuous and routine EEG in intensive care: utilization and outcomes, United States 2005–2009. *Neurology* 2013;81:2002–2008.
- Nuwer MR. EEG and evoked potentials: monitoring cerebral function in the neurosurgical ICU. In: Martin NA, ed. *Neurosurgical Intensive Care*. Philadelphia, PA: W.B. Saunders, 1994: 647–659.
- O'Connor KL, Westover MB, Phillips MT, et al. High risk for seizures following subarachnoid hemorrhage regardless of referral bias. *Neurocrit Care* 2014;21:476–482.
- Oddo M, Carrera E, Claassen J, et al. Continuous electroencephalography in the medical intensive care unit. *Crit Care Med* 2009;37:2051–2056.
- Ong C, Gilmore E, Claassen J, et al. Impact of prolonged periodic epileptiform discharges on coma prognosis. *Neurocrit Care* 2012;17:39–44.
- Pandian JD, Cascino GD, So EL, et al. Digital video-electroencephalographic monitoring in the neurological-neurosurgical intensive care unit: clinical features and outcome. *Arch Neurol* 2004;61:1090–1094.
- Payne ET, Zhao XY, Frmdova H, et al. Seizure burden is independently associated with short term outcome in critically ill children. *Brain* 2014;137:1429–1438.
- Pedersen GL, Rasmussen SB, Gyllenborg J, et al. Prognostic value of periodic electroencephalographic discharges for neurological patients with profound disturbances of consciousness. *Clin Neurophysiol* 2013;124:44–51.
- Piantino JA, Wainwright MS, Grimson M, et al. Nonconvulsive seizures are common in children treated with extracorporeal cardiac life support. *Pediatr Crit Care Med* 2013;14:601–609.
- Pinkerton JA Jr. EEG as a criterion for shunt need in carotid endarterectomy. *Ann Vasc Surg* 2002;16:756–761.
- Plestis KA, Loubser P, Mizrahi EM, et al. Continuous electroencephalographic monitoring and selective shunting reduces neurologic morbidity rates in carotid endarterectomy. *J Vasc Surg* 1997;25:620–628.
- Prins SA, de Hoog M, Blok JH, et al. Continuous noninvasive monitoring of barbiturate coma in critically ill children using the bispectral index monitor. *Crit Care* 2007;11:R108.
- 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.
- Rittenberger JC, Popescu A, Brenner RP, et al. Frequency and timing of nonconvulsive status epilepticus in comatose post-cardiac arrest subjects treated with hypothermia. *Neurocrit Care* 2012;16:114–122.
- Ronne-Engstrom E, Winkler T. Continuous EEG monitoring in patients with traumatic brain injury reveals a high incidence of epileptiform activity. *Acta Neurol Scand* 2006;114:47–53.
- Rossetti AO, Oddo M, Logroscino G, Kaplan PW. Prognostication after cardiac arrest and hypothermia: a prospective study. *Ann Neurol* 2010;67:301–307.
- Rossetti AO, Milligan TA, Vulliemoz S, et al. A randomized trial for the treatment of refractory status epilepticus. *Neurocrit Care* 2011;14:4–10.
- Sadaka F, Doerr D, Hindia J, et al. Continuous electroencephalogram in comatose postcardiac arrest syndrome patients treated with therapeutic hypothermia: outcome prediction study. *J Intensive Care Med* 2014;Jan 7 [Epub ahead of print].
- Saengpatrachai M, Sharma R, Hunjan A, et al. Nonconvulsive seizures in the pediatric intensive care unit: etiology, EEG, and brain imaging findings. *Epilepsia* 2006;47:1510–1518.
- Sanchez Fernandez I, Abend NS, Arndt DH, et al. Electrographic seizures after convulsive status epilepticus in children and young adults: a retrospective multicenter study. *J Pediatr* 2014;164:339 e1–2–346 e1–2.
- Sanchez SM, Arndt DH, Carpenter JL, et al. Electroencephalography monitoring in critically ill children: current practice and implications for future study design. *Epilepsia* 2013a;54:1419–1427.
- Sanchez SM, Carpenter J, Chapman KE, et al. Pediatric ICU EEG monitoring: current resources and practice in the United States and Canada. *J Clin Neurophysiol* 2013b;30:156–160.
- Sandroni C, Cavallaro F, Callaway CW, et al. Predictors of poor neurological outcome in adult comatose survivors of cardiac arrest: a systematic review and meta-analysis. Part 1: patients not treated with therapeutic hypothermia. *Resuscitation* 2013a;84:1310–1323.
- Sandroni C, Cavallaro F, Callaway CW, et al. Predictors of poor neurological outcome in adult comatose survivors of cardiac arrest: a systematic review and meta-analysis. Part 2: patients treated with therapeutic hypothermia. *Resuscitation* 2013b;84:1324–1338.
- 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;17:31–38.
- Sessler CN, Jo Grap M, Ramsay MA. Evaluating and monitoring analgesia and sedation in the intensive care unit. *Crit Care* 2008;12(suppl 3):S2.
- 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.
- 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;28:611–617.
- Sheorajpanday RV, Nagels G, Weeren AJ, et al. Reproducibility and clinical relevance of quantitative EEG parameters in cerebral ischemia: a basic approach. *Clin Neurophysiol* 2009;120:845–855.
- Skordilis M, Rich N, Vilorio A, et al. Processed electroencephalogram response of patients undergoing carotid endarterectomy: a pilot study. *Ann Vasc Surg* 2011;25:909–912.
- Stevens RD, Sutter R. Prognosis in severe brain injury. *Crit Care Med* 2013;41:1104–1123.
- Synek VM. Prognostically important EEG coma patterns in diffuse anoxic and traumatic encephalopathies in adults. *J Clin Neurophysiol* 1988;5:161–174.
- Synek VM. Value of a revised EEG coma scale for prognosis after cerebral anoxia and diffuse head injury. *Clin Electroencephalogr* 1990;21:25–30.
- Tay SK, Hirsch LJ, Leary L, et al. Nonconvulsive status epilepticus in children: clinical and EEG characteristics. *Epilepsia* 2006;47:1504–1509.
- 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. *Crit Care Med* 2013;41:215–223.
- Towne AR, Waterhouse EJ, Boggs JG, et al. Prevalence of nonconvulsive status epilepticus in comatose patients. *Neurology* 2000;54:340–345.
- 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.

- van Putten MJ, Peters JM, Mulder SM, et al. A brain symmetry index (BSI) for online EEG monitoring in carotid endarterectomy. *Clin Neurophysiol* 2004;115:1189–1194.
- Vespa PM, Nuwer MR, Juhasz C, et al. Early detection of vasospasm after acute subarachnoid hemorrhage using continuous EEG ICU monitoring. *Electroencephalogr Clin Neurophysiol* 1997;103:607–615.
- Vespa P, Prins M, Ronne-Engstrom E, et al. Increase in extracellular glutamate caused by reduced cerebral perfusion pressure and seizures after human traumatic brain injury: a microdialysis study. *J Neurosurg* 1998;89:971–982.
- Vespa PM, Nenov V, Nuwer MR. Continuous EEG monitoring in the intensive care unit: early findings and clinical efficacy. *J Clin Neurophysiol* 1999a;16:1–13.
- 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* 1999b;91:750–760.
- Vespa P, Martin NA, Nenov V, et al. Delayed increase in extracellular glycerol with post-traumatic electrographic epileptic activity: support for the theory that seizures induce secondary injury. *Acta Neurochir Suppl* 2002a;81:355–357.
- Vespa PM, Boscardin WJ, Hovda DA, et al. Early and persistent impaired percent alpha variability on continuous electroencephalography monitoring as predictive of poor outcome after traumatic brain injury. *J Neurosurg* 2002b;97:84–92.
- 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.
- 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;35:2830–2836.
- Wagenman KL, Blake TP, Sanchez SM, et al. Electrographic status epilepticus and long-term outcome in critically ill children. *Neurology* 2014;82:396–404.
- Walder B, Suter PM, Romand JA. Evaluation of two processed EEG analyzers for assessment of sedation after coronary artery bypass grafting. *Intensive Care Med* 2001;27:107–114.
- Waziri A, Claassen J, Stuart RM, et al. Intracortical electroencephalography in acute brain injury. *Ann Neurol* 2009;66:366–377.
- Westover MB, Shafi MM, Bianchi MT, et al. The probability of seizures during EEG monitoring in critically ill adults. *Clin Neurophysiol* 2014;126:463–471.
- Williams K, Jarrar R, Buchhalter J. Continuous video-EEG monitoring in pediatric intensive care units. *Epilepsia* 2011;52:1130–1136.
- 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;47:83–89.
- Young GB, Kreeft JH, McLachlan RS, Demelo J. EEG and clinical associations with mortality in comatose patients in a general intensive care unit. *J Clin Neurophysiol* 1999;16:354–360.
- Young GB, Wang JT, Connolly JF. Prognostic determination in anoxic-ischemic and traumatic encephalopathies. *J Clin Neurophysiol* 2004;21:379–390.
- Ypparila H, Korhonen I, Westeren-Punnonen S, et al. Assessment of postoperative sedation level with spectral EEG parameters. *Clin Neurophysiol* 2002;113:1633–1639.