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

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I. INTRODUCTION AND PURPOSE

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. Electroencephalography (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 four-fold increase in the number of CEEGs performed in intensive care units (ICUs) (Ney, van der Goes et al. 2013). Recent surveys, however, show variability in why and how CEEG is performed in the ICU (Abend, Dlugos et al. 2010, Sanchez, Arndt et al. 2013, Gavvala, Abend et al. 2014), 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. CCEEG 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. CCEEG typically includes simultaneous video recording, and may include graphical displays of quantitative EEG trends (QEEG). 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, in order 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 (LTME), sleep monitoring, or intraoperative monitoring. Separate recommendations have been developed by the American Clinical Neurophysiology Society (ACNS) for CCEEG in critically ill neonates (Shellhaas, Chang et al. 2011).

The ACNS CCEEG Guidelines Committee 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. CCEEG is often requested as an urgent or emergency study in critically ill patients. Current staffing models may not support 24-hour 7-day per week in-house NDTs. This consensus statement therefore addresses minimum techniques for CCEEG under emergency circumstances, as well as optimal techniques when qualified NDTs are available.

CCEEG 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 section III. Indications). 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 section III. Indications). Subsequent CCEEG recordings can then be compared to 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.

CCEEG 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 neurodiagnostic technologists (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.

II. 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 non-neonatal 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 prior to approval by ACNS Council. Recommendations are based on expert opinion and should not be used for performance measurements or competency purposes.

III. INDICATIONS FOR CCEEG

A. Diagnosis of Nonconvulsive Seizures (NCS), Nonconvulsive Status Epilepticus (NCSE), and Other Paroxysmal Events

1. ***CCEEG is recommended to identify NCS and NCSE in critically ill patients with:***

   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, Meyers et al. 1998). During 24 hours of CCEEG after GCSE, NCS were recorded in 48% and NCSE in 14% (DeLorenzo, Waterhouse et al. 1998). Similarly, NCS were seen in 43% of patients who had convulsive seizures before monitoring (Claassen, Mayer et al. 2004). Children with convulsive seizures (McCoy, Sharma et al. 2011, Greiner, Holland et al. 2012, Abend, Arndt et al. 2013) or GCSE (Williams, Jarrar et al. 2011) prior to CCEEG are at higher risk for NCS. Thirty-three percent of 98 children undergoing CCEEG after GCSE terminated had ongoing electrographic seizures (Sanchez Fernandez, Abend et al. 2014). Impaired consciousness after clinical seizures end can be secondary to prolonged postictal effects, sedative effects of antiseizure drugs, 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, Bell et al. 2012, Claassen, Taccone et al. 2013).

   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 (TBI), subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), encephalitis, acute ischemic stroke, and during and after therapeutic hypothermia post cardiac arrest (Claassen, Taccone et al. 2013). Patients less than 18 years of age may be at higher risk than adults for NCS and NCSE (Claassen, Mayer et al. 2004), and within the pediatrics age group, neonates and infants may be at higher risk than older children (Hosain, Solomon et al. 2005, Jette, Claassen et al. 2006, Saengpattrachai, Sharma et al. 2006, Tay, Hirsch et al. 2006, Abend and Dlugos 2007, Abend, Topjian et al. 2009, Shahwan, Bailey et al. 2010, Abend, Gutierrez-Colina et al. 2011, McCoy, Sharma et al. 2011, Williams, Jarrar et al. 2011, Greiner, Holland et al. 2012, Kirkham, Wade et al. 2012, Schreiber, Zelleke et al. 2012, Abend, Arndt et al. 2013, Arndt, Lerner et al. 2013, Hasbani, Topjian et al. 2013).

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

d. ***Generalized periodic discharges (GPDs), lateralized periodic discharges (LPDs), or BIPDs (bilateral independent periodic discharges) on routine or emergent EEG***. Adults and children with generalized or lateralized periodic discharges are more likely to develop NCS or NCSE (Jette, Claassen et al. 2006, Foreman, Claassen et al. 2012, Ong, Gilmore et al. 2012, Akman, Abou Khaled et al. 2013, Gaspard, Manganas et al. 2013, Pedersen, Rasmussen et al. 2013). The presence of lateralized rhythmic delta activity (LRDA) appears to have the same high association with seizures as LPDs and is also a reasonable indication for CCEEG (Gaspard, Manganas et al. 2013).

e. ***Requirement for pharmacological paralysis (e.g. therapeutic hypothermia protocols, extracorporeal membrane oxygenation (ECMO)) and risk for seizures***. 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 if they are ictal or non-ictal.*** Critically ill adults and children may have a variety of episodic abnormal movements or other clinical events which raise concern for seizures (Benbadis, Chen et al. 2010, Boesebeck, Freermann et al. 2010, Williams, Jarrar et al. 2011). Antiseizure drugs (ASDs) 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, Kelley 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 (Waziri, Claassen et al. 2009, Claassen, Perotte et al. 2013). 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, Perotte et al. 2013).

2. Evidence supporting use of CCEEG to identify seizures. Evaluation for suspected NCS is the most common indication for CCEEG (Abend, Dlugos et al. 2010, Sanchez, Carpenter et al. 2013, Gavvala, Abend et al. 2014). NCS, 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. NCSE, 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, Bell et al. 2012), and continuous or recurrent NCS for more than 50% of an EEG epoch.

    a. NCS occur in 8–48% of critically ill adults (Privitera, Hoffman et al. 1994, Jordan 1995, DeLorenzo, Waterhouse et al. 1998, Vespa, Nenov et al. 1999, Towne, Waterhouse et al. 2000, Claassen, Mayer et al. 2004, Pandian, Cascino et al. 2004, Oddo, Carrera et al. 2009) and 6–47% of children with altered mental status(Hosain, Solomon et al. 2005, Jette, Claassen et al. 2006, Tay, Hirsch et al. 2006, Abend and Dlugos 2007, Abend, Topjian et al. 2009, Shahwan, Bailey et al. 2010, Abend, Gutierrez-Colina et al. 2011, McCoy, Sharma et al. 2011, Williams, Jarrar et al. 2011, Greiner, Holland et al. 2012, Kirkham, Wade et al. 2012, Schreiber, Zelleke et al. 2012, Abend, Arndt et al. 2013, Arndt, Lerner et al. 2013, Hasbani, Topjian et al. 2013). Table 1 summarizes the percentage of critically ill patients with seizures by etiology.

    b. NCS 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 (Vespa, Prins et al. 1998, Vespa, Nuwer et al. 1999, Vespa, Martin et al. 2002, Vespa, O'Phelan et al. 2003, Vespa, Miller et al. 2007, Fabricius, Fuhr et al. 2008, Hartings, Watanabe et al. 2011, Dreier, Major et al. 2012).

    c. Prolonged NCS or NCSE are associated with increased mortality and increased risk for poor neurologic outcome (Young, Jordan et al. 1996, Abend, Arndt et al. 2013, Topjian, Gutierrez-Colina et al. 2013, Claassen, Albers et al. 2014, Payne, Zhao et al. 2014, Wagenman, Blake et al. 2014), 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, Dlugos 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, Costello et al. 2009) and children (Abend, Topjian et al. 2011).

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 exam.*** 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. ***CCEEG 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, Bell et al. 2012). The length of a CCEEG depends on the pre-test 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 30–60 minute EEG recordings identify NCS in only 45–58% of patients in whom seizures are eventually recorded (Claassen, Mayer et al. 2004, Pandian, Cascino et al. 2004, Abend, Gutierrez-Colina et al. 2011). About 80–95% of patients with NCS can be identified within 24–48 hours (Claassen, Mayer et al. 2004, Jette, Claassen et al. 2006, Abend and Dlugos 2007, Shahwan, Bailey et al. 2010, Abend, Gutierrez-Colina et al. 2011). ***In specific populations, such as patients who are comatose, have periodic discharges, or are pharmacologically sedated, NCS may occur later, so more prolonged monitoring (48 hours or more) may be needed*** (Claassen, Mayer et al. 2004, Abend, Topjian et al. 2009). Early EEG findings may help to refine the required period of recording (Westover, Shafi et al. 2014). Patients without early epileptiform discharges (within the first two hours) had less than a 5% chance of seizures in the next 72 hours. Brief (30 minute) serial EEGs have been demonstrated to have similar yield to CCEEG in adult post cardiac arrest patients undergoing hypothermia (Crepeau, Fugate 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. ***CCEEG 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 if it was ictal or non-ictal.

B. Assessment of Efficacy of Therapy for Seizures and Status Epilepticus

1. ***CCEEG is recommended to monitor the response of seizures and status epilepticus (SE) to treatment.***

   a. NCS and NCSE are common after apparently successful treatment of clinical seizures and SE (see section III.A.1.a) and cannot be diagnosed without EEG.

   b. ***For patients with refractory status epilepticus (RSE), 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. RSE is defined as clinical or electrographic seizures which continue after initial treatment for SE, typically with a benzodiazepine and at least one other acceptable ASD (Brophy, Bell 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. CCEEG 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, Hirsch et al. 2001, Claassen, Hirsch et al. 2002). CCEEG 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 (Krishnamurthy and Drislane 1999, Jordan and Hirsch 2006, Prins, de Hoog et al. 2007, Rossetti, Milligan et al. 2011).

3. Assessment of clinical behavior. ***Concurrent video recording is strongly recommended as a supplement to the clinical exam.*** 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 following administration of ASDs.

4. Timing and duration. ***CCEEG should be initiated as soon as possible when persistent NCS are suspected after treatment of clinical seizures or SE. CCEEG should be recorded until seizures have been controlled for at least 24 hours. CCEEG should be recorded during the entire period that cIV-ASDs are utilized.*** Seizures may recur despite EEG-confirmed burst-suppression or complete suppression (Claassen, Hirsch et al. 2001, Claassen, Hirsch 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, Dlugos 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 section III.A.5, rapid identification of NCS allows appropriate ASD treatment to be initiated quickly and may reduce morbidity and mortality associated with NCS and NCSE. ***CCEEG 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 if it was ictal or non-ictal.

C. Identification of Cerebral Ischemia

1. ***CCEEG 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). CCEEG, and particularly quantitative EEG trends, can be used to identify changes in cortical perfusion before irreversible infarct occurs (Vespa, Nuwer et al. 1997, Claassen, Hirsch et al. 2004).

    a. EEG and quantitative EEG techniques have been used to identify ischemia during neurosurgical and interventional neuroradiology vascular procedures (Plestis, Loubser et al. 1997, Laman, van der Reijden et al. 2001, Pinkerton 2002, van Putten, Peters et al. 2004, Botes, Le Roux et al. 2007, Ballotta, Saladini et al. 2010, Mishra, Banday et al. 2011, Skordilis, Rich et al. 2011).

    b. Retrospective studies have shown that CCEEG and quantitative trends can identify delayed cerebral ischemia (DCI) during vasospasm after SAH, but no prospective studies have been performed (Vespa, Nuwer et al. 1997, Claassen, Hirsch et al. 2004). Most centers using CCEEG for identification of vasospasm monitor patients at highest risk (severe SAH 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. MR/CT perfusion or angiography, TCDs or conventional angiography) to identify DCI and may predict which patients are at risk for DCI earlier than other studies.

    c. CCEEG holds promise for ischemia identification in patients with hemodynamic lesions and borderline flow or those at high risk for acute ischemic stroke (Sheorajpanday, Nagels 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 quantitative EEG. This may change as resources increase and automated EEG analysis improves, and if intracranial EEG recordings are utilized more often.

3. Assessment of clinical behavior. ***Concurrent video recording is recommended as a supplement to the clinical exam.*** Review of video can help to identify artifacts as well as changes in EEG and QEEG related to state changes.

4. Timing and duration. ***CCEEG should be recorded during the period of time when the patient is at highest risk for ischemia.***

a. SAH. CCEEG 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. CCEEG 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 (TIA) or 24 hours following 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, while 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 quantitative EEG analysis,*** such as graphical displays of power ratios over time.

D. Monitoring of Sedation and High-Dose Suppressive Therapy

1. CCEEG is suggested, in conjunction with the clinical examination, to assess level of consciousness in patients requiring intravenous sedation or pharmacologically-induced coma (Walder, Suter et al. 2001, Ypparila, Korhonen et al. 2002, de Wit and Epstein 2003, Doenicke, Kugler et al. 2007, Mirski and Hemstreet 2007). 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 II.B.2.b). 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 quantitative EEG methods have been utilized for monitoring analgesia and sedation in the ICU (Sessler, Jo Grap 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.

E. 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, Synek 1990, Young, Kreeft et al. 1999, Young, Wang et al. 2004). Favorable prognostic features include background continuity, spontaneous variability, reactivity to stimulation, and presence of normal sleep patterns (Synek 1988, Synek 1990, Young, Kreeft et al. 1999, Young, Wang et al. 2004). Clinical populations in which EEG may aid in prognosis include:

    a. Severe traumatic brain injury, including abusive traumatic brain injury in infants (Vespa, Nuwer et al. 1999, Vespa, Boscardin et al. 2002, Stevens and Sutter 2013).

    b. Hypoxic ischemic encephalopathy following cardiac arrest (without or with therapeutic hypothermia) (Rossetti, Oddo et al. 2010, Kessler, Topjian et al. 2011, Sandroni, Cavallaro et al. 2013a, Sandroni, Cavallaro et al. 2013b)

    c. SAH (Claassen, Hirsch 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, Wang 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, since many medications can produce EEG changes that are identical to changes seen with brain injury.

3. 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, Oddo et al. 2010).

4. 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.

IV. CONCLUSION

CCEEG 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.

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Table 1

Common neurological, medical, and surgical conditions associated with high likelihood of recording seizures on CCEEG

	Adults	Children	References
Post convulsive status epilepticus	48%	26–57%	(DeLorenzo, Waterhouse et al. 1998, Tay, Hirsch et al. 2006, Abend, Gutierrez-Colina et al. 2011, Williams, Jarrar et al. 2011, Abend, Wusthoff et al. 2013, Sanchez Fernandez, Abend et al. 2014)
Aneurysmal subarachnoid hemorrhage	Any seizure: 10–19% NCSE: 3–13%		(Dennis, Claassen et al. 2002, Claassen, Mayer et al. 2004, Claassen, Hirsch et al. 2006, Little, Kerrigan et al. 2007, Claassen, Albers et al. 2014, O'Connor, Westover et al. 2014, Westover, Shafi et al. 2014)
Intraparenchymal hemorrhage	16–23%	11–100%	(Vespa, O'Phelan et al. 2003, Jette, Claassen et al. 2006, Saengpattrachai, Sharma et al. 2006, Tay, Hirsch et al. 2006, Claassen, Jette et al. 2007, McCoy, Sharma et al. 2011, Greiner, Holland et al. 2012, Kirkham, Wade et al. 2012, Kurtz, Gaspard et al. 2014, Payne, Zhao et al. 2014, Westover, Shafi et al. 2014)
Moderate to severe traumatic brain injury	18–33%	14–70%	(Vespa, Nuwer et al. 1999, Claassen, Mayer et al. 2004, Jette, Claassen et al. 2006, Ronne-Engstrom and Winkler 2006, Abend, Gutierrez-Colina et al. 2011, Williams, Jarrar et al. 2011, Schreiber, Zelleke et al. 2012, Abend, Arndt et al. 2013, Arndt, Lerner et al. 2013, Hasbani, Topjian et al. 2013, Sanchez, Arndt et al. 2013, Payne, Zhao et al. 2014)
Central nervous system infections	10–33%	16–100%	(Claassen, Mayer et al. 2004, Jette, Claassen et al. 2006, Saengpattrachai, Sharma et al. 2006, Tay, Hirsch et al. 2006, Carrera, Claassen et al. 2008, Abend, Gutierrez-Colina et al. 2011, Williams, Jarrar et al. 2011, Gwer, Idro et al. 2012, Schreiber, Zelleke et al. 2012, Abend, Arndt et al. 2013, Payne, Zhao et al. 2014, Westover, Shafi et al. 2014)
Recent neurosurgical procedures	23%	71%	(Claassen, Mayer et al. 2004, Payne, Zhao et al. 2014, Westover, Shafi et al. 2014)
Brain tumors	Any seizure: 23–37% NCSE: 9–12%	19–66%	(Jette, Claassen et al. 2006, Abend, Gutierrez-Colina et al. 2011, Greiner, Holland et al. 2012, Kirkham, Wade et al. 2012, Abend, Arndt et al. 2013, Marcuse, Lancman et al. 2014, Westover, Shafi et al. 2014)
Acute ischemic stroke	6–27%	20–71%	(Vespa, O'Phelan et al. 2003, Claassen, Mayer et al. 2004, Jette, Claassen et al. 2006, Saengpattrachai, Sharma et al. 2006, Abend, Gutierrez-Colina et al. 2011, McCoy, Sharma et al. 2011, Greiner, Holland et al. 2012, Kirkham, Wade et al. 2012, Sanchez, Carpenter et al. 2013, Kurtz, Gaspard et al. 2014, Payne, Zhao et al. 2014, Westover, Shafi et al. 2014)
Hypoxic-ischemic injury following cardiac or respiratory arrest, with or without therapeutic hypothermia	10–59%	16–79%	(Claassen, Mayer et al. 2004, Jette, Claassen et al. 2006, Tay, Hirsch et al. 2006, Abend, Topjian et al. 2009, Abend, Gutierrez- Colina et al. 2011, Kawai, Thapalia et al. 2011, Williams, Jarrar et al. 2011, Mani, Schmitt et al. 2012, Rittenberger, Popescu et al. 2012, Abend, Arndt et al. 2013, Crepeau, Rabinstein et al. 2013, Knight, Hart et al. 2013, Legriel, Hilly-Ginoux et al. 2013, Sanchez, Arndt et al. 2013, Payne, Zhao et al. 2014, Sadaka, Doerr et al. 2014, Westover, Shafi et al. 2014)
Sepsis-associated encephalopathy	32%	58%	(Oddo, Carrera et al. 2009, Abend, Arndt et al. 2013)
Extracorporeal membrane oxygenation (ECMO)		21%	(Piantino, Wainwright et al. 2013)
Epilepsy Related	33–39%	11–71%	(Claassen, Mayer et al. 2004, Jette, Claassen et al. 2006, Saengpattrachai, Sharma et al. 2006, Tay, Hirsch et al. 2006, Hyllienmark and Amark 2007, Abend, Gutierrez-Colina et al. 2011, McCoy, Sharma et al. 2011, Abend, Arndt et al. 2013, Westover, Shafi et al. 2014) Hyllienmark 2007 McCoy 2011