

COUNTERPOINT:

Emergency (“Stat”) EEG in the Era of Nonconvulsive Status Epilepticus

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ABSTRACT. *The most widely accepted indication for a stat EEG (stEEG) is the suspicion of nonconvulsive status epilepticus (NCSE). NCSE has been reported with surprising frequency in a wide variety of acute structural and metabolic brain injuries and significantly increases the risk of permanent brain damage and death. This risk rises and the effectiveness of treatment decreases with delays in diagnosis and increased duration of NCSE. Recent evidence confirms that more than half of NCSE patients improve with anti-seizure treatment. The emergence of NCSE as a common, dangerous, time-urgent, and treatable problem has positioned it as a target for emergency therapeutic intervention. NCSE can only be diagnosed by EEG testing, and stEEG has demonstrated value in improving NCSE management. As a result, in the near future, EEG laboratories will see increasing demands for stEEG related to NCSE. The two main obstacles to an effective stEEG program are EEG technologist coverage and electroencephalographer availability after work hours. We recommend three simple but fundamental changes in the traditional approach to stEEGs in order to overcome these obstacles: the use of EEG set-up templates by onsite personnel, easy access to EEG instruments after hours, and remote stEEG connectivity for real-time, off-site electroencephalographer interpretation.*

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KEY WORDS. *Acute brain injuries, emergency EEG, nonconvulsive status epilepticus, stat EEG.*

INTRODUCTION AND BACKGROUND

The most compelling and broadly accepted indication for an emergency EEG, often referred to as a “stat” EEG (stEEG), is the suspicion of nonconvulsive status epilepticus (NCSE) (Jordan 1995, Firosh et al. 2005, Praline et al. 2007, Benbadis 2008). As defined according to published criteria (Young et al. 1996, Claassen et al. 2004), NCSE has been reported with surprising frequency in patients with a wide variety of acute structural and metabolic causes of acute brain injury (ABI) (Jordan 1993, Privatera 1994, Vespa et al. 2005, Jirsch and Hirsch 2007) (Table 1). In fact, the overall reported incidence of NCSE in ABI is higher than that of ventricular tachycardia in acute myocardial infarction (13% versus 8%) (Al-Khatib et al. 2003, Hirsch 2004, Jordan 2008).

Although no class I studies have been performed to date, there is abundant evidence that when complicating ABI, NCSE significantly increases the risk of permanent brain damage and death (Vespa 2005, Hirsch 2008). This evidence includes:

Table 1. *Percentage of the incidence of nonconvulsive status epilepticus (NCSE) in reported series. CSE — convulsive status epilepticus, INF — meningitis and encephalitis, TBI — traumatic brain injury, ICH — intracerebral hemorrhage, SAH — subarachnoid hemorrhage, NSG — post craniotomy, ALOC — cryptogenic reduced consciousness, ASBL — unspecified acute structural brain lesions, ME — metabolic encephalopathy, NCSE — nonconvulsive status epilepticus, NCS — nonconvulsive seizures.*

	CSE	INF	TBI	ICH	SAH	NSG	ALOC	ASBL	ME
Alroughani et al. 2008							9		
Claassen et al. 2007				7					
Claassen et al. 2005	20	17	8	9	13	8	5		8
Young and Doig 2005								13*	
Vespa et al. 1999, 2003			11	11					
Towne et al. 2000							8		
DeLorenzo et al. 1998	14								
Trieman et al. 1998	32								

*Estimate based upon reported NCSE/NCS ratio in ASBL of 36 to 45%.

- NCSE is a predictor of worse outcome in multiple patient populations (Jirsch and Hirsch 2007).
- Neuron-specific enolase, a marker of neuronal injury, is elevated after NCSE and is highest in patients with combined NCSE and ABI (Rabinowicz et al. 1995).
- In patients with intracerebral hemorrhage (ICH), NCSE is associated with increased mass effect, midline shift, and clinical worsening (Vespa et al. 2003), as well as with expanding hemorrhages and worse outcome (Claassen et al. 2007).
- NCSE causes increases in brain glutamate above excitotoxic levels and glycerol levels consistent with cell membrane breakdown (Vespa et al. 1999, Vespa 2005).
- NCSE after acute traumatic brain injuries (TBI) produces prolonged elevations in intracranial pressure and elevated lactate/pyruvate ratios (Vespa et al. 2007).
- In animal models of acute cerebral infarction (ACI), NCSE causes a three-fold increase in mortality independent of infarct size (Williams et al. 2004).

The mortality rate of untreated NCSE increases by 1 to 2% every hour. The longer the delay to diagnosis and duration of NCSE, the greater the risk of permanent brain damage and death and the less the chance of successful treatment (Young et al. 1996, Drislane et al. 2008). Although some reports have considered NCSE an epiphenomenon of severe ABI and have questioned the value of intervention, recent evidence confirms that more than half the patients do respond to treatment. With anti-seizure medication, 56% of intensive care unit (ICU) NCSE patients improved in alertness, two-thirds of whom were comatose. Improvement highly correlated with survival (Drislane et al. 2008). Among intracerebral hemorrhage patients with NCSE, 60% of patients treated for status regained consciousness (Claassen et al. 2007).

Clinicians ordering stEEGs consider it an important tool for diagnosing and managing patients with suspected NCSE. In a prospective study of 111 patients, NCSE and “subtle NCSE” were suspected in 34% and stEEG confirmed the diagnosis in 44% of patients tested. Overall, clinicians indicated that the test contributed to making the diagnosis in 77.5% of cases prompting stEEG (Praline et al. 2007). In another study, stEEG was ordered because of suspected NCSE in 54 patients, and was classified as useful in 96% of the cases (Firosh et al. 2005).

The emergence of NCSE as a common, dangerous, time-urgent, and treatable problem positions it as a target for therapeutic intervention in a wide spectrum of ABIs (Vespa et al. 2003, Vespa 2005, Williams et al. 2006, Drislane et al. 2008). Since NCSE can only be diagnosed by EEG testing, and stEEG has demonstrated value in NCSE, it is highly likely that EEG laboratories will see a significant increase in orders for stEEGs in the near future. These orders will come from all areas of the hospital, including the emergency department (ED), ICUs, and general hospital

floors. This will place new demands on hospital EEG laboratories to provide stEEG services. Pressure to meet these demands will be driven primarily by good patient care, but also by interest in patients' lengths of stay, resource utilization, and potential liability risks. Providing rapid, efficient, and cost-effective stEEG services on a 24 hour, 7-day a week, 365-day a year basis (24/7/365) will require EEG laboratories to rethink their traditional approaches to stEEG and find new solutions to long standing obstacles (Quigg 2001, Benbadis 2008).

The balance of this article suggests indications for stEEG in this "Era of NCSE," reviews the obstacles to stEEGs, and provides recommendations to help hospital-based EEG laboratories meet the anticipated increased demand for stEEGs.

INDICATIONS FOR stEEG IN THE ERA OF NCSE

We suggest that a succinct and appropriate indication for considering stEEG is "an unexpected decline in the patient's consciousness." A pathological reduction in awareness is always a worrisome clinical event and, though far from specific, it is the most common sign of generalized NCSE. While some authors have suggested that clinical features, such as facial or ocular twitching, can be used to "screen" for NCSE, these are neither sensitive nor specific enough to guide diagnostic decisions, including ordering a stEEG (Husain 2003, Claassen et al. 2004, Kaplan 2005, Riggio 2005, Claassen et al. 2008). Continuous video-EEG monitoring in the ICU has shown that the absence of movements does not exclude NCSE and that convulsive-like movements can occur on a nonepileptic basis, such as dystonic drug reactions, ischemic spasms, or metabolic mini-myoclonus (Jordan 1995, Claassen 2007).

In addition, identifying structural injuries or metabolic insults such as subarachnoid hemorrhage (SAH), ICH, illicit drugs, or hyponatremia in these patients does not exclude concurrent NCSE as the primary or a compounding cause of the decline in consciousness (Drislane et al. 2008). In fact, most reported cases of NCSE are in patients with primary structural or metabolic ABI (Jordan 1993, Hirsch 2004). NCSE occurs in 20 to 32% of patients with convulsive status epilepticus who stop convulsing, 17% of patients with meningitis/encephalitis, 8 to 11% with acute TBI, 10% with ICH, 13% with SAH, and 8% with metabolic encephalopathies or cryptogenic altered consciousness. Among general hospital patients with unexplained altered awareness, 9.3% had NCSE (Table 1).

While we agree with the statement that, "Not every patient with coma or unresponsiveness should be suspected of being in NCSE." (Benbadis 2008), we believe that the available evidence favors and we do recommend that in every patient with an *unexpected* decline in consciousness, consideration be given to obtaining a stEEG to confirm or exclude NCSE.

WHO SHOULD ORDER stEEGS?

It is an accepted and long-standing practice for non-specialist physicians and other medical providers to order diagnostic studies that require interpretation by their specialist colleagues, such as CT and MRI scans, EKGs, ultrasound tests, nuclear medicine studies, as well as EEGs. In fact, specialists who are expert in interpreting diagnostic tests may be removed from the bedside practice of clinical medicine. Therefore, we believe that the physician attending the patient, the one who has taken the patient's medical history, examined the patient, and reviewed the relevant ancillary studies is in the best position to decide whether or not to order a stEEG. As with other diagnostic tests, the physician's decisions will be improved by some basic education in stEEG and an approved stEEG protocol. This protocol should be developed by the EEG Laboratory Director with contributions from fellow epileptologists, electroencephalographers, neurologists, and technologists. The protocol should go through the requisite hospital committees for review and then become standard practice for hospital clinicians. A stEEG protocol is also a good platform for driving clinical, organizational, staffing, and logistical decisions for the EEG laboratory (Figure 1).

We do not think patients are well served by interposing neurologists, technologists, fellows, or residents in "screening" or "gatekeeper" positions. This implies a responsibility and authority to pass judgment on or second-guess our clinician colleagues' decisions. We do not require cardiologists to screen EKG requests nor to do consultations before an EKG can be ordered. We understand that many EEG laboratories struggle with scarce resources and that such "screening" policies for stEEGs are intended to ease this burden (although they add a significant burden to the "screeners") (Quigg 2001, Benbadis 2008). From a patient-centered viewpoint, "screening" requirements can become barriers that delay obtaining stEEGs, while the patient's NCSE may be taking an increasing toll. We believe there are better solutions (see below: "Recommendations for stEEG Services").

OBSTACLES TO stEEG

There are two main obstacles to establishing a stEEG service:

- 1) **Technologist Coverage:** An on-call and call-back system for EEG technologists is a major expense, an organizational challenge, and a common source of employee burn-out. For this reason, many U.S. hospitals, including major medical centers, do not have EEG technologists available for nights, weekends, or holidays. As of this writing, most U.S. hospitals do not offer the option for stEEG after work hours. Even with a tech on-call system in place,

the delay from “ordering to recording” a stEEG after work hours can be lengthy. A time-motion study (Jordan and Schneider 2005) found this delay to be from 1.5 to 2.3 hours (Table 2). An informal survey taken by the authors among technologists and neurologists at professional meetings suggest this range may be on the low side.

- 2) **Electroencephalographer Availability:** The need for stEEGs to be read and reported emergently by a qualified electroencephalographer is often an intrusive burden for the on-call neurologist, who must travel to the hospital to interpret the study. In addition, a neurologist with EEG reading skills may not always be available. In most hospitals, neurologists rotate coverage for emergencies, but only 30% of board certified neurologists read EEGs. There is limited interest or incentive for electroencephalographers to take on this additional responsibility. There is usually no on-call pay and there is no additional reimbursement for reading a study stat versus reading it at 3PM the next day. One survey of several large EEG laboratories reported an average delay of 4 hours, and up to 24 hours for a stEEG to be “officially” read and another found a *mean* delay of 27 hours (Quigg et al. 2001, Firosh et al. 2005).

RECOMMENDATIONS FOR stEEG SERVICES

The following recommendations are based on the authors’ successful implementation of a 24/7/365 wide area network EEG service to three regional hospitals from 1999 to 2004, including a Level 1 trauma center, a tertiary care community hospital, and a small rural hospital. During this time, we performed over a thousand stEEG studies on patients in emergency departments, intensive care units, and hospital floors. We previously reported results from this program (Jordan and Schneider 2004, Jordan and Schneider 2005).

We believe these recommendations can be generalized and implemented by the majority of EEG laboratories that desire to develop stEEG services. We advise three simple but fundamental changes in the way stEEGs have traditionally been provided:

1. Train onsite staff to use EEG set-up templates for stEEGs. There are several commercial models of EEG set-up templates available, most of which are simple enough for non-expert onsite medical personnel to be trained to use with little difficulty. Onsite staff may include nurse practitioners, physician assistants, nurses, residents, medical students, respiratory therapists, nurse assistants, as well as EEG technologists if available. EEG set-up templates vary in specific features, but most provide acceptable accuracy and reliability within the International 10/20 Electrode Placement System guidelines. These

Sample Stat EEG Protocol

1. General Indication for stat EEG (stEEG)

The patient has suffered an unexpected decline in consciousness.

Examples:

- The patient does not awaken within 30 minutes of ending a convulsive seizure.
- The patient does not awaken from anesthesia following a craniotomy.
- The patient is comatose with a mild lithium overdose.
- The patient has a stable intracranial hemorrhage but declines from a Glasgow Coma Score (GCS) of 12 to a GCS of 7.

2. Ordering the stEEG

- The physician attending the patient orders the stEEG.
- Use verbal order to RN, written order sheet, or physician electronic order.
- Confirm stEEG order is processed.

3. Setting-Up the stEEG

- Designated on-site medical personnel (RN, NP, RT, Resident, or Fellow, etc. who have gone through training, or EEG technologist if available) are notified to set-up stEEG.
- EEG instrument moved to patient's bedside.
- EEG set-up template used to apply electrodes.
- EEG instrument connections established to remote network.
- EEG reader contacted to connect remotely to EEG equipment.
- "Plug and play" EEG recording started using pre-programmed stEEG settings and montage.

4. Reading the stEEG

- EEG reader connects to EEG data (via in-house network or remote VPN).
- EEG reader contacts ordering physician with interpretation via text, email, or phone.
- Report is printed out from EEG instrument for documentation.

5. After stEEG completed

- EEG electrodes and template are removed and discarded (if disposable) or stored for cleaning if re-usable.
- EEG instrument is returned to storage area.
- If continuous EEG is ordered, electrodes can be secured and monitoring continued.

FIG. 1. Sample of a stat EEG (stEEG) protocol incorporating recommendations in the text

templates are an easy and efficient way for stEEGs to be set up without additional labor expenses or scheduling problems. If continuous EEG is needed, during the next work day, the EEG technologists can check the template electrodes and replace them with manually measured electrodes if desired. This option not only saves on labor costs and staff "wear and tear," but can reduce stEEG "ordering to recording" time from more than two hours

Table 2. *On-call EEG technologist time-motion study (Jordan and Schneider 2005).*

Task	Duration (minutes)
Page . . . get ready. . . leave home	15 to 30
Drive time/parking	15 to 30
To Lab for supplies	15
Set-up equipment at bedside	15
Chart review/enter history	10 to 20
Measure head: 10/20 method	10 to 20
Apply electrodes	10
Total Set-Up Time	1.5 hour to 2.3 hours

to less than 15 minutes, markedly shortening the time to treatment decisions (Jordan and Schneider 2005).

2. Provide ready access to one or more EEG instruments after work hours. A portable or mobile EEG instrument that is conveniently located to the ED and ICU may be all that is needed, depending on the demand for stEEGs. A regular daytime instrument can be set aside during off-work hours for this purpose and wheeled to the patient's bedside. It is easy to train onsite medical personnel to turn on the equipment, put in basic patient data, and begin the recording with a pre-set montage. Most commercial EEG jackboxes have either color-coded or common electrode plug-ins that make it easy to connect the electrodes to the instrument. In our program, we conducted 2-hour training classes, provided easy-to-follow handouts, and attached a simple step-by-step instruction list to each stEEG instrument.
3. Use remote access software for the electroencephalographer to read the stEEG from outside the hospital. Almost all commercial EEG systems have software to read EEG studies by remote connectivity either by T-1/T-10 lines or similar hardwired access, or by Virtual Private Network Internet access. Health Insurance Portability and Accountability Act (HIPPA) compliance can be accomplished using patient data encryption and other security measures. This is analogous to after-hours remote radiological interpretation, which has become routine in many centers. A number of hospitals are currently using EEG local area networks for routine reading and for monitoring ICU and epilepsy patients within the hospital campus, and several are beginning to allow access from outside the campus confines (Hirsch LJ, Personal Communication). Hospitals and EEG vendors can cooperate to provide the electroencephalographer with a laptop computer and reading software. Medicare currently reimburses remote interpretation of EEGs at the same rate as standard readings. This option not only improves electroencephalographer convenience, but more importantly, it dramatically reduces the response time to stEEG interpretation.

SUMMARY

- We have entered the “Era of NCSE,” where abundant and compelling data indicate that NCSE is a common, time-urgent, dangerous, and treatable process; that it commonly complicates ABI; and that it can only be diagnosed by EEG testing. This combination of factors makes NCSE an emergency therapeutic target and will likely result in dramatically increased demand for stEEGs. The authors believe it is incumbent upon hospitals and EEG laboratories to review their stEEG programs so this demand can be met in our patients’ interests.
- The cardinal clinical feature of generalized NCSE is an unexpected decline in consciousness, which we recommend be the threshold indication for considering stEEG. NCSE often occurs in the context of structural or metabolic brain insults, and can cause or compound brain damage.
- The physician in charge of the patient’s clinical assessment should have the autonomy to order stEEGs according to his/her best judgment guided by an approved stEEG protocol.
- Onsite medical personnel can set-up stEEGs on a 24/7 basis using EEG set-up templates. Hospitals can provide easy access to EEG instruments during off-work hours. It is relatively easy to give these personnel basic training to use the set-up templates and initiate the EEG recording. The electroencephalographer on call can read stEEGs promptly by remote connectivity.
- These simple and inexpensive alternatives to traditional stEEG approaches will allow hospitals to offer stEEG services at lower cost, greater efficiency, with increased availability, and with greater staff satisfaction. Most importantly, our patients with previously undiagnosed NCSE will be rapidly identified and given timely and appropriate treatment.

DISCLOSURES

Dr. Jordan and Ms. Schneider are principals in Jordan NeuroScience, Inc, a medical device manufacturer specializing in acute EEG monitoring. They have received no direct or indirect financial support for this article.

REFERENCES

- Al-Khatib SM, Stebbins AL, Califf RM, Lee KL, Granger CB, White HD, Armstrong PW, Topol EJ, Ohman EM, GUSTO-III trial. Sustained ventricular arrhythmias and mortality among patients with acute myocardial infarction: results from the GUSTO-III trial. *Am Heart J* 2003; 145(3):515–21.
- Alroughani R, Javidan M, Qasem A, Alotaibi N. Non-convulsive status epilepticus; the rate of occurrence in a general hospital. *Seizure* Aug 26, 2008; (Epub ahead of print).
- Benbadis SR. Use and abuse of stat EEG. *Expert Rev Neurother* 2008; 8(6):865–68.

- Claassen J, Mayer SA, Kowalski RG, Emerson RG, Hirsch LJ. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. *Neurology* 2004; 62:1743–48.
- Claassen J, Mayer SA, Hirsch LJ. Continuous EEG monitoring in patients with subarachnoid hemorrhage. *J Clin Neurophysiol* 2005; 22:92–98.
- Claassen J, Jetté N, Chum F, Green R, Schmidt M, Choi H, Jirsch J, Frontera JA, Connolly ES, Emerson RG, Mayer SA, Hirsch LJ. Electrographic seizures and periodic discharges after intracerebral hemorrhage. *Neurology* 2007; 69(13):1356–65.
- Claassen J, Jetté N, Chum F, Green R, Schmidt M, Choi H, Jirsch J, Frontera JA, Connolly ES, Emerson RG, Mayer SA, Hirsch LJ. Electrographic seizures and periodic discharges after intracerebral hemorrhage. *Neurology (Letter)* 2008:1555.
- Drislane FW, Lopez MR, Blum AS, Schomer DL. Detection and treatment of refractory status epilepticus in the intensive care unit. *J Clin Neurophysiol* 2008; 25(4):181–86.
- DeLorenzo RJ, Waterhouse EJ, Towne AR, Boggs JG, Ko D, DeLorenzo GA, Brown A, Garnett L. Persistent nonconvulsive status epilepticus after the control of convulsive status epilepticus. *Epilepsia* 1998; 39:833–40.
- Firosh Kahn S, Ashalatha R, Thomas SV, Sarma PS. Emergent EEG is helpful in neurology critical care practice. *Clin Neurophysiol* 2005; 116(10):2454–59.
- Hirsch LJ. Continuous EEG monitoring in the intensive care unit: an overview. *J Clin Neurophysiol* 2004; 21(5):332–40.
- Hirsch LJ. Nonconvulsive seizures in traumatic brain injury: what you don't see can hurt you. *Epilepsy Curr* 2008; 8(4):97–99.
- Husain AM, Horn GJ, Jacobson MP. Non-convulsive status epilepticus: usefulness of clinical features in selecting patients for urgent EEG. *J Neurol Neurosurg Psychiatry* 2003; 74(2):189–91.
- Jirsch J, Hirsch LJ. Nonconvulsive seizures: developing a rational approach to the diagnosis and management in the critically ill population. *Clin Neurophysiol* 2007; 118:1660–1670.
- Jordan KG. Continuous EEG and evoked potential monitoring in the neuroscience intensive care unit. *J Clin Neurophysiol* 1993; 10(4):445–75.
- Jordan KG. Neurophysiologic monitoring in the neuroscience intensive care unit. *Neurol Clin* 1995; 13:579–626.
- Jordan KG. Unpublished data. Presented at UCLA Neuromonitoring Course 2008. Los Angeles, California.
- Jordan KG, Schneider AL. Rapid, remote, realtime ER-EEG. *Neurocrit Care* 2004; 1:A125.
- Jordan KG, Schneider AL. Overcoming barriers to ICU emergency and continuous EEG. *Crit Care Med* 2005; 33:A104.
- Kaplan PW. The clinical features, diagnosis and prognosis of nonconvulsive status epilepticus. *Neurologist* 2005; 11:348–61.
- Praline J, Grujic J, Corcia P, Lucas B, Hommet C, Autret A, de Toffol B. Emergent EEG in clinical practice. *Clin Neurophysiol* 2007; 118:2149–55.
- Privitera M, Hoffman M, Moore JL, Jester D. EEG detection of nontonic-clonic status epilepticus in patients with altered consciousness. *Epilepsy Res* 1994; 18:155–66.
- Quigg M, Shneker B, Domer P. Current practice in administration and clinical criteria of emergent EEG. *J Clin Neurophysiol* 2001; 18:162–65.
- Rabinowicz AL, Correale JD, Bracht KA, Smith TD, DeGiorgio CM. Neuron-specific enolase is increased after nonconvulsive status epilepticus. *Epilepsia* 1995; 36(5):475–79.
- Riggio S. Nonconvulsive status epilepticus: clinical features and diagnostic challenges. *Psychiat Clin North Am* 2005; 28:653–64.
- Towne AR, Waterhouse EJ, Boggs JG, Garnett LK, Brown AJ, Smith JR Jr, DeLorenzo RJ. Prevalence of nonconvulsive status epilepticus in comatose patients. *Neurology* 2000; 54:340–45.
- Trieman DM, Meyers PD, Walton NY, Collins JF, Colling C, Rowan AJ, Handforth A, Faught E, Calabrese VP, Uthman BM, Ramsay RE, Mamdani MB. A comparison of four treatments for generalized convulsive status epilepticus. Veterans affairs status epilepticus cooperative study group. *N Engl J Med* 1998; 339:792–98.

- Vespa PM. Continuous EEG monitoring for the detection of seizures in traumatic brain injury, infarction, and intracerebral hemorrhage: "To Detect and Protect." *J Clin Neurophysiol* 2005; 22(2):99–106.
- Vespa PM, Nuwer MR, Nenov V, Ronne-Engstrom E, Hovda DA, Bergsneider M, Kelly DR, Martin NA, Becker DP. Increased incidence and impact of nonconvulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring. *J Neurosurg* 1999; 91:750–60.
- Vespa PM, Miller C, McArthur D, Eliseo M, Etchepare M, Hirt D, Glenn TC, Martin N, Hovda D. Nonconvulsive electrographic seizures after traumatic brain injury result in a delayed, prolonged increase in intracranial pressure and metabolic crisis. *Crit Care Med* 2007; 35(120):2830–36.
- Vespa PM, O'Phelan K, Shah M, Mirabelli J, Starkman S, Kidwell C, Saver J, Nuwer MR, Frazee JG, McArthur DA, Martin NA. Acute seizures after intracerebral hemorrhage: a factor in progressive midline shift and outcome. *Neurology* 2003; 60:1441–46.
- Williams AJ, Tortella FC, Lu XM, Moreton JE, Hartings JA. Antiepileptic drug treatment of nonconvulsive seizures induced by experimental focal brain ischemia. *J Pharmacol Exp Ther* 2004; 311(1):220–27.
- Williams AJ, Hartings JA, Tortella FC. Non-convulsive seizures secondary to brain injury: an emerging clinical concern and potential implications for clinical trial design. Walter Reed Army Institute of Research White Paper 2006.
- Young GB, Jordan KG, Doig GS. An assessment of non-convulsive seizures in the intensive care unit using continuous EEG monitoring: an investigation of variables associated with mortality. *Neurology* 1996; 47:83–89.
- Young GB, Doig GS. Continuous EEG monitoring in comatose intensive care patients: epileptiform activity in etiologically distinct groups. *Neurocrit Care* 2005; 2:5–10.