
Continuous EEG Monitoring in the Intensive Care Unit

John Kay

ABSTRACT: Background: Electroencephalography (EEG) is playing an increasingly important role in the management of comatose patients in the intensive care unit. **Methods:** The techniques of EEG monitoring are reviewed. Initially, standard, discontinuous recordings were performed in intensive care units (ICUs). Later, continuous displays of "raw EEG" (CEEG) were used. More recently, the addition of quantitative techniques allowed for more effective reading. **Results and Conclusions:** Applications of continuous EEG to clinical problems are discussed. The most useful role of CEEG appears to be the detection and management of nonconvulsive seizures. There is a need for controlled studies to assess the role for CEEG in neuro-ICUs and general ICUs.

RÉSUMÉ: Surveillance EEG continue à l'unité de soins intensifs. Introduction: L'électroencéphalographie (EEG) joue un rôle de plus en plus important dans la prise en charge des patients comateux à l'unité de soins intensifs. **Méthodes:** Nous revoyons les techniques de surveillance EEG. Au départ, des enregistrements standards discontinus étaient pratiqués à l'unité de soins intensifs (USI). Par la suite, des moniteurs d'EEG "brut" (EEGC) étaient utilisés. Plus récemment, l'ajout de techniques quantitatives permet une meilleure évaluation. **Résultats et conclusions:** Nous discutons de l'application de l'EEG continu aux problèmes cliniques. Le rôle le plus utile de l'EEGC semble la détection et la prise en charge des crises épileptiques non convulsives. Des études contrôlées doivent être faites pour évaluer le rôle de l'EEGC à l'USI neurologique et à l'USI générale.

Can. J. Neurol. Sci. 1998; 25: S12-S15

Monitoring of cerebral cortical activity in the intensive care unit is challenging, particularly in patients with a depressed level of consciousness. Bedside clinical testing in comatose patients primarily addresses brain stem reflex function and is limited by the effects of sedative drugs and neuromuscular junction blocking agents. As outlined by Jordan,¹ EEG is a useful monitor of brain activity for five reasons. EEG activity is linked to cerebral metabolism; EEG is sensitive to the effects of hypoxia and ischemia, the most common causes of cerebral injury; EEG correlates with cerebral topography and can localize abnormalities; EEG deteriorates prior to irreversible cell damage, allowing for identification of deterioration and intervention prior to call death; and EEG is the best available method for detecting seizures.

Computerized digital EEG has made EEG practical for the ICU setting, and networking software has allowed EEG to be monitored both at the patient's bedside and at one or more remote sites. The ICU nurse is the person who has the single greatest contact with the patient, and experience at our institution and others has shown that the ICU nurse can learn to identify important features on the EEG accurately, thus providing real time monitoring 24 hours per day.¹ Digital data can also be stored for later review and analysis of trends. Digitization allows for quantitative techniques, e.g., power spectral analysis and the compressed spectral array, and provides better visualization of subtle frequency changes and trends in amplitude and frequency over time.^{2,3} This application should, however, be viewed as an adjunct to the continuous display of "raw EEG", as

qualitative changes can be missed and artifact may not be recognized as such.⁴ In contrast to many neurodiagnostic tests, EEG has the advantage of being non-invasive, relatively inexpensive, and available at the bedside without the need for patient transport.

EEG and Detection of Non-convulsive Seizures

EEG is the best available means of detecting seizures. In the intensive care unit, clinical recognition of seizures can be difficult because of the variable presentation of seizures in patients with altered levels of consciousness and in patients treated with neuromuscular blockade. The detection of non-convulsive seizures is one of the major clinical uses of continuous EEG monitoring (CEEG). Criteria defining the electrographic diagnosis of seizures in comatose patients have been described.⁵ Non-convulsive seizures may present as the cause of coma, simulating conditions such as metabolic encephalopathy, and may be undiagnosed until documented on EEG. Non-convulsive seizures may also complicate the course of patients with known acute cerebral injuries such as trauma, stroke, tumor, infection, and anoxia (Figure). Retrospective studies of CEEG monitored patients in a neurologic intensive care unit identified

From the Department of Clinical Neurological Sciences, University of Western Ontario, London.

Reprint requests to: G. Bryan Young, Department of Clinical Neurological Sciences, London Health Sciences Centre, 375 South Street, London, Ontario, Canada, N6A 4G5

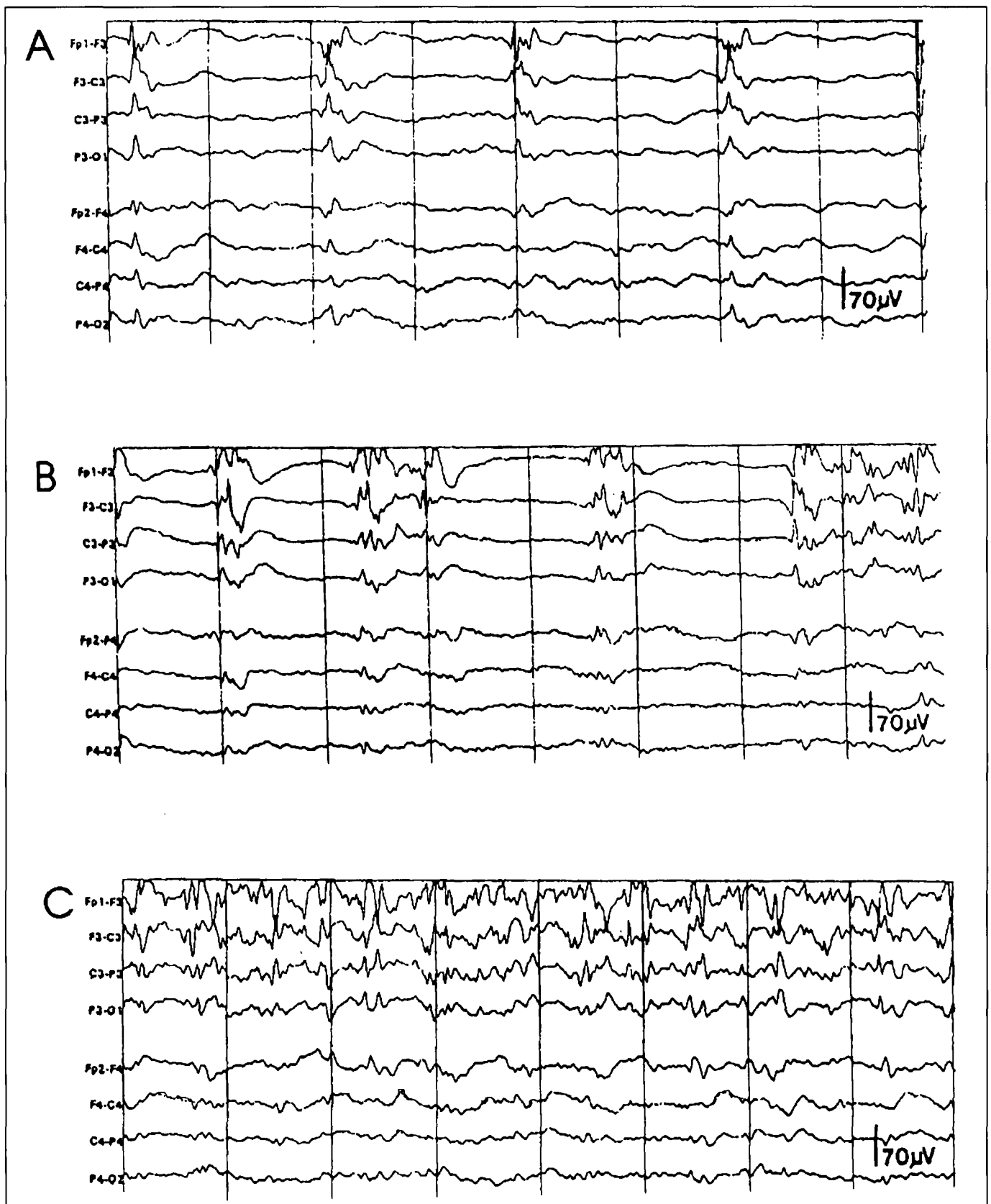


Figure: The evolution of periodic lateralized epileptiform discharges (PLEDs) to a seizure in a man with a left temporal glioma. PLEDs initially occur as single then polyphasic spikes every 2 seconds (A). The polyphasic component (after discharges) gradually lengthens and the interval between complexes shortens (B). Finally, continuous seizure activity occurs in the left hemisphere (C) Vertical lines are 1 second apart.

a 35% incidence of seizures, with 76% of these patients having non-convulsive status epilepticus.⁶ Preliminary data from our institution have shown a lower incidence of seizures in a population of comatose patients in a general intensive care unit.

In a study of 49 neuro-ICU patients with non-convulsive seizures,⁵ the overall mortality was 33%. Those who had non-convulsive status epilepticus had a mortality of 57%, where status was defined as EEG-recorded ictal episodes that were continuous or recurrent for greater than 30 minutes without improvement in clinical state or return to a pre-ictal EEG pattern between seizures. Mortality was associated with age, seizure duration, length of stay, delay to diagnosis and etiology, although in a multi-variant analysis only seizure duration and delayed diagnosis were independently associated with increased mortality. This study emphasizes the need for EEG monitoring of patients at risk for non-convulsive seizures to facilitate early diagnosis. Further studies are required to determine whether earlier diagnosis and effective treatment of non-convulsive seizures will improve patient outcomes.

CEEG and Convulsive Status Epilepticus

Continuous EEG is useful to monitor the treatment of convulsive status epilepticus, particularly in patients who are comatose or pharmacologically paralyzed. In patients treated for status epilepticus post-ictal or drug-induced depressed level of consciousness must be differentiated from ongoing non-convulsive status epilepticus.⁷ Continuous EEG monitoring can determine when treatment for status epilepticus has been effective,⁸ thus minimizing the dose of drugs administered and reducing the duration of iatrogenic coma. Protocols for the treatment of refractory status epilepticus with barbiturate⁹ or midazolam¹⁰ coma require EEG monitoring to determine the depth of coma and to assess whether ictal activity remains abolished as the drugs are withdrawn. Effective management of patients with status epilepticus requires the EEG monitoring be initiated promptly. Technical considerations may preclude the use of collodion secured disc electrodes and the 10-20 international system during the treatment of the acute phase of status epilepticus. We have used pre-gelled disposable adhesive electrodes and sub-hairline montage as described by Ebersole¹¹ as a means of initiating CEEG monitoring quickly during urgent situations.

Other Uses of CEEG

In addition to the detection of seizures, CEEG monitoring has several other uses. EEG can be used to guide the depth of medically induced coma. Serum and CSF drug levels correlate poorly with physiologic effect during pentobarbital-induced coma.¹² CEEG provides continuous monitoring of drug effect, allowing the barbiturate dose to be the lowest necessary to maintain burst suppression and minimize toxicity. CEEG can also be used to gauge the depth of sedation in patients treated with neuromuscular blocking agents.

EEG activity is related to cerebral blood flow. EEG has, therefore, been used following subarachnoid hemorrhage with focal electrographic changes being predictive of vasospasm.¹³ Quantitative EEG studies have shown decreased relative alpha variability in vasospasm.¹⁴ Furthermore, CEEG can be used to monitor the effects of therapy for vasospasm.¹⁵

EEG monitoring shows focal slowing in acute ischemic stroke. Interventions to treat ischemia can reverse EEG abnor-

malities,¹⁶ often before clinical change is observed.¹ EEG should be investigated as a means of bedside monitoring of emerging therapies in the treatment of acute ischemic stroke.

EEG patterns have prognostic importance following global hypoxic-ischemic cerebral injury. Serial EEG studies show evolution of electrographic change to prognostically definitive patterns.¹⁷ CEEG monitoring of comatose patients following cerebral anoxia may allow more rapid determination of prognosis, and aid in decisions regarding continuing life sustaining treatment.

The Role of Continuous EEG

The role of continuous EEG monitoring is still evolving. The impact of CEEG on patient management has been reviewed retrospectively in a neurologic intensive care unit setting.¹ The impact on care was considered "decisive" when at least one major clinical decision, such as the initiation or alteration of anti-epileptic drug therapy or transporting the patient out of the intensive care unit, was made solely on the basis of CEEG findings. The impact was deemed contributory when at least one major decision was made on the basis of CEEG plus the clinical examination. CEEG had a decisive or contributory impact on the management of 82% of cases. However, before being widely accepted, CEEG will have to be shown not only to influence medical decisions, but also to influence patient outcomes. A prospective randomized control trial comparing CEEG monitoring to routine care for comatose patients in a general intensive care unit is currently underway in our institution with the aim of assessing whether CEEG monitoring improves patient care as measured by the length of stay in the intensive care unit, mortality, quality of life in surviving patients, and the cost of hospitalization.

REFERENCES

1. Jordan KG. Continuous EEG and evoked potential monitoring in the neurosciences intensive care unit. *J Clin Neurophysiol* 1993; 10: 445-475.
2. Nuwer MR. Quantitative EEG: II. Frequency analysis and topographic mapping in clinical settings. *J Clin Neurophysiol* 1988; 5: 45-85.
3. Cant BR, Shaw NA. Monitoring by compressed spectral array in prolonged coma. *Neurology* 1984; 34: 35-39.
4. Nuwer MR. Quantitative EEG I. Techniques and problems of frequency analysis and topographic mapping. *J Clin Neurophysiol* 1988; 5: 1-43.
5. 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.
6. Jordan KG. Non-convulsive status epilepticus in the neuro-ICU by continuous EEG monitoring. *Neurology* 1992; 42 (Suppl 1): 194.
7. Fagar KJ, Lee SI. Prolonged confusion following convulsions due to generalized non-convulsive status epilepticus. *Neurology* 1990; 40: 1689-1694.
8. VanNess PC. Pentobarbital and EEG burst suppression in treatment of status epilepticus refractory to benzodiazepines and phenytoin. *Epilepsia* 1990; 31: 61-67.
9. Lowenstein D, Aminoff M, Simon R. Barbiturate anesthesia in treatment of status epilepticus: clinical experience in the treatment of 14 patients. *Neurology* 1988; 38: 395-400.
10. Kumar A, Bleck T. Intravenous midazolam for the treatment of refractory status epilepticus. *Crit Care Med* 1992; 20: 483-488.
11. Ebersole JS, Bridgers SL. Montage design for Cassette EEG. In: Ebersole JS, ed. *Ambulatory EEG Monitoring*. New York: Raven Press, 1989: 64-66.

12. Winer JW, Rosenwasser RH, Jiminez F. Electroencephalographic activity and serum and cerebrospinal fluid pentobarbital level in determining the therapeutic end point during barbiturate coma. *Neurosurgery* 1991; 29: 739-741.
13. Rivierez M, Landau-Ferey J, Grob R, et al. Value of electroencephalogram in prediction and diagnosis of vasospasm after intracranial aneurysm rupture. *Acta Neurochirurgica* 1991; 110: 17-23.
14. Vespa PM, Nuwer MR, Nartin NA, Becker DP. Early detection of vasospasm after subarachnoid hemorrhage using continuous quantitative electroencephalogram. *Neurology* 1996; 46 (Suppl): A385.
15. Jordan KG. Neurophysiologic monitoring in the neuroscience intensive care unit. *Neurologic Clinics* 1995; 13: 579-626.
16. Wood JH, Polyzoidis KS, Epstein CM, Gibby GL, Tindall GT. Quantitative EEG alterations after isovolemic hemodilution augmentation of cerebral perfusion in stroke patients. *Neurology* 1984; 34: 764-768.
17. Young GB, Blume WT, Campbell VM, et al. Alpha, theta and alpha-theta coma: a clinical outcome study utilizing serial recordings. *Electroenceph Clin Neurophysiol* 1994; 91: 93-99.