Incorporating Abbreviated EEGs in the Initial Workup of Patients Who Present to the Emergency Room With Mental Status Changes of Unknown Etiology

Ramon Edmundo D. Bautista, Steven Godwin,† and David Caro†*

Summary: Patients frequently present to the emergency room (ER) with mental status changes without obvious cause. The EEG is underused in this population. The authors investigated whether an abbreviated EEG (AbEEG) can be incorporated in the early evaluation of these patients to provide useful information. A 5-minute AbEEG was performed using a preformed electrode placement system on 25 patients who presented to the ER with mental status changes of unknown cause. AbEEG findings were categorized as normal, showing diffuse abnormalities, focal abnormalities, electrographic seizures, or uninterpretable. Using retrospective chart review, the authors determined if the cause of mental status change was a diffuse encephalopathy or a nonneurologic event (DENNE), a focal brain abnormality, nonconvulsive status epilepticus (NCSE), psychogenic, or unknown, and if particular AbEEG findings were associated with specific causes of altered sensorium. The AbEEG identified NCSE in two patients who presented with new-onset seizures. The presence of diffuse slowing on the AbEEG was highly suggestive of mental status changes due to DENNE. AbEEGs can be successfully incorporated in the early evaluation of patients who present to the ER with mental status changes of unknown cause and provide useful information in this setting.

Key Words: Coma, Confusion, Electroencephalogram, Emergency room, Mental status change, Nonconvulsive status epilepticus.

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Patients commonly present to the emergency room (ER) with changes in mental status. It is estimated that up to 2% of all ER patients have some degree of confusion (Huff and Brady, 2002). There are many causes for acute changes in mental status (Sabin, 1981), some of which may not be immediately obvious. In the ER setting, it is important to arrive quickly at the correct diagnosis.

The EEG remains the most widely used test of electrocerebral activity (Hoosmand and Maloney, 1980). The EEG can help distinguish among the different forms of coma (Brenner, 1985) and is also the test of choice in establishing the diagnosis of nonconvulsive status epilepticus (NCSE) (Brenner, 1985; Kaplan, 1996; Kaplan, 1999; Niedermeyer and Ribeiro, 2000) and evaluating response to therapy.

Despite this, the EEG remains an underused test in the ER setting (Hoosmand and Maloney, 1980; Kaplan, 1999). In fact, there is no agreed on criteria for the use of emergent EEGs (Quigg et al., 2001). Although part of the reason for its underuse may be the lack of awareness among ER physicians of the utility of EEG when assessing patients with mental status changes, we believe that another factor limiting its use is the amount of time and preparation needed to perform a routine EEG (ACNS, 2006). This can potentially interfere with the established workup of patients who are simultaneously being assessed with other imaging and diagnostic modalities.

The delay in obtaining EEGs in the ER in patients in NCSE also leads to a delay in the diagnosis and treatment of this condition. In Kaplan's study (1999), the majority of NCSE cases were diagnosed more than 24 hours after arrival in the ER.

In this study, we attempt to incorporate a 5-minute abbreviated EEG (AbEEG) using a preformed electrode placement system in the initial workup of patients who present to the ER with changes in mental status of unknown cause. This technique shortens the duration and amount of preparation needed to obtain electrophysiologic data and generally does not interfere with other ongoing diagnostic procedures. We sought to determine if an AbEEG can be successfully incorporated in this scenario to provide useful information.

METHODS

The Institutional Review Board of the University of Florida HSC/Jacksonville and Shands Hospital approved this study.

We incorporated AbEEGs in the routine initial workup of 25 consecutive patients who presented to a tertiary care

Departments of *Neurology and †Emergency Medicine, University of Florida Health Sciences Center, Jacksonville, Florida.

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Address correspondences and reprint requests to Ramon Edmundo D. Bautista, M.D., Department of Neurology, University of Florida Health Sciences Center/Jacksonville, 580 West Eighth Street, Tower 1, Jacksonville, FL 32209; e-mail: ramon.bautista@jax.ufl.edu.

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academic medical center adult ER with changes in mental status of unknown cause. For purposes of this study, we limited our study sample to patients whose cause of mental status change could not be determined after initial clinical assessment by an ER physician but before use of imaging and/or laboratory tests except for bedside capillary glucose determination using fingerstick testing. Patients could present with different manifestations of altered sensorium (such as lethargy, coma, aphasia, or psychosis) and could have had resolved, improving, or ongoing lapses in sensorium at the time the AbEEG was performed. The decision to perform the AbEEG was made by the ER attending physician without neurologic consultation.

Patients were excluded from the study because of the following: 1. Findings in the initial patient history and/or examination (as determined by the ER attending physician) that already indicate the cause for the mental status change; 2. A capillary glucose level greater than 200 mg/dL or less than 40 mg/dL as measured by fingerstick testing; or 3. ER physician determination that the patient was clinically unstable to have the AbEEG.

This protocol models realistic ER scenarios where the ER physicians perform the initial clinical assessment and neurologists are not immediately present when patients arrive in the ER. Capillary glucose determination was included because this can be performed quickly at bedside. In our study, the need for AbEEGs could be determined within a few minutes after the patient arrived in the ER.

Experienced EEG technicians performed the AbEEG and were in the ER within 10 minutes after they were called. The AbEEG consisted of a 5-minute 16-channel portable digital EEG recording (Nihon Kohden, Foothill Ranch, CA) using a preformed electrode placement system that consisted of a commercially available stretch cap with 20 tin electrodes attached to the fabric (Electro-Cap, Eaton, OH). The cap was placed on the patient's head and the placement of the electrodes conformed to the International 10 to 20 System. Electrode gel was applied to each electrode although impedances were not measured and activating procedures were not performed. The amount of time and preparation needed to place the electrode cap on and perform the AbEEG was less than 10 minutes. The 5-minute recording time was chosen so as not to interfere with the standard ER workup of these patients.

Although the AbEEGs were not performed with the intention of influencing patient care, the EEG technicians were instructed to inform the ER attending if the AbEEG findings were consistent with status epilepticus. Also, the ER/attending physicians had the discretion of ordering standard EEGs either in the ER and/or later during the hospital stay that were used to influence patient care.

A board-certified electroencephalographer (R.B.) blinded to the clinical scenario reviewed the AbEEGs only after patient discharge. The AbEEG findings were categorized as follows:

1. Diffuse abnormalities without electrographic seizures (including background slowing). Diffuse, bisynchronous or multifocal interictal epileptiform discharges were included in this category so long as they did not evolve into electrographic seizure activity.
2. Focal abnormalities without electrographic seizures. Focal interictal epileptiform discharges were also included in this category as long as they did not evolve into electrographic seizure activity.
3. Electrographic seizures (including both generalized and focal seizures).
4. Normal.
5. Uninterpretable.

The AbEEG findings could be listed under different categories if it contained mixed features (i.e., diffuse slowing admixed with focal abnormalities or the presence of focal abnormalities admixed with electrographic seizures).

Patient chart review was later done to obtain the discharge diagnosis. Based on this, we determined if the cause of the patient's change in mental status was due to

1. A diffuse encephalopathy or a nonneurologic event (DENNE) that occurred without ongoing clinical seizures. Included in this category were metabolic/systemic causes of altered mental status, meningoencephalitis, neurodegenerative conditions, syncope, and hypoxic/anoxic causes. Also included in this category were patients who presented with altered sensorium due to a postictal state but were not actively having seizures in the ER.
2. A focal brain abnormality without ongoing clinical seizures (i.e., tumor, CVA).
3. NCSE regardless of etiology.
4. Psychogenic causes.
5. Unknown causes.

Data Analysis

We determined if the different categories of AbEEG findings discriminate among the various causes of mental status change. In particular, we wanted to know if patients with diffuse slowing on the AbEEG had changes in sensorium due to DENNE and if focal abnormalities on AbEEG were due to a focal brain abnormality. We also wanted to know if the presence of electrographic seizures on a 5-minute AbEEG was sufficient to diagnose NCSE and whether normal AbEEGs were indicative of mental status changes due to psychogenic causes.

RESULTS

Patients' ages ranged from 15 to 91 years and 13 were males. Patient demographics, discharge diagnosis, and AbEEG results are summarized in Table 1. Table 2 shows the number of patients with specific AbEEG and clinical findings. Two of the 25 AbEEGs were uninterpretable (patients 1 and 10) due to movement artifacts and were excluded from analysis. Two AbEEGs (patients 3 and 19) had mixed features and were listed under different categories.

Two patients (patients 2 and 17) presented in NCSE. In both cases the AbEEGs showed electrographic seizure activity (Fig. 1). These findings were later verified by standard EEGs also performed in the ER. New onset seizures of

TABLE 1. Patient Demographics, Discharge Diagnosis, and Abbreviated EEG Findings

Patient No.	Age (y) / Sex	Clinical Diagnosis	Abbreviated EEG Findings
1	77/M	Postictal confusion	Uninterpretable due to movement artifacts
2	56/F	NCSE	Left temporal rhythmic 1–2 Hz sharp and slow wave activity
3	49/M	Antiepileptic drug toxicity	Diffuse theta and delta, intermittent left hemisphere sharp and slow waves
4	74/F	Post-ictal confusion	Diffuse delta
5	44/M	Syncope	Normal
6	47/M	Urosepsis	Background slowing postictal confusion
7	65/M	Dementia	Normal
8	64/M	Syncope	Normal
9	15/M	TCA overdose	Diffuse delta and theta admixed with beta; bifrontal slow waves
10	91/F	Urosepsis	Uninterpretable due to movement artifacts
11	56/F	Postictal confusion	Diffuse polymorphic theta and delta admixed with beta
12	65/M	Anoxic encephalopathy	Diffuse low-voltage theta and delta
13	53/F	Hypertensive encephalopathy	Background slowing
14	25/F	Uncertain	Low-voltage diffuse delta
15	52/F	Uncertain	Normal
16	67/M	Postictal confusion	Background slowing
17	55/F	NCSE	Bifrontal rhythmic spike and wave activity
18	61/F	Uncertain	Normal
19	64/F	Left MCA stroke	Diffuse delta activity; accentuated slowing over left hemisphere
20	68/M	Postictal confusion	Mild background slowing
21	30/M	Cryptococcal meningitis	Normal
22	51/M	Postictal confusion	Background slowing
23	48/F	Postictal confusion	Diffuse theta activity
24	48/F	Dehydration/malnutrition	Normal
25	45/M	Uncertain	Normal

MCA, middle cerebral artery; NCSE, nonconvulsive status epilepticus; TCA, tricyclic antidepressants.

TABLE 2. Number of Patients With Specific Abbreviated EEG (AbEEG) and Clinical Findings

		Clinical Findings		
AbEEG Findings	DENNE	Focal Brain Abnormalities	Nonconvulsive Status Epilepticus	Unknown
Diffuse abnormalities	11	1		1
Focal abnormalities	1	1		
Electrographic Seizures			2	
Normal	5			3
Uninterpretable	2			

DENNE, diffuse encephalopathy or nonneurologic event.

cryptogenic etiology were later diagnosed in these patients. In this study, we did not encounter any situation wherein the AbEEG did not show electrographic seizure activity but NCSE was later diagnosed.

Sixteen patients had mental status changes due to DENNE. Of these, 10 had AbEEGs that showed diffuse/background slowing (Fig. 2) and 5 were normal. One AbEEG showed both diffuse and focal abnormalities (patient 3). Patients with normal AbEEG findings had a variety of discharge diagnoses that included syncope (patients 5 and 8), worsening dementia (patient 7), meningoencephalitis (patient 21), and dehydration (patient 24).

We obtained 13 EEGs that showed diffuse abnormalities (patients 3, 4, 6, 9, 11–14, 16, 19, 20, 22, and 23). Of these, 11 were obtained from patients with DENNE (positive predictive value 84%). However, the absence of diffuse abnormalities did not necessarily rule out DENNE (negative predictive value 50%). All patients whose cause of mental status changes were due to postictal confusion had diffuse slowing on their AbEEG and the absence of diffuse slowing on AbEEG ruled out postictal confusion as the cause of mental status changes. Two patients who were later diagnosed to have had a syncopal event as the cause of mental status change had normal AbEEGs.

One patient (patient 19) had a focal brain abnormality without seizures (left middle cerebral artery stroke). The AbEEG showed both diffuse slowing and focal abnormalities. Focal abnormalities were also seen in an epilepsy patient who developed mental status changes due to antiepileptic drug toxicity (patient 3).

In our series, there were no instances where mental status changes were thought to be due to psychogenic causes.

In four patients, the cause for mental status changes remained unknown even at the time of hospital discharge. The AbEEGs showed diffuse slowing in one (patient 14) and were normal in the rest (patients 15, 18, and 25). By the time of discharge, these patients returned to baseline mental state.

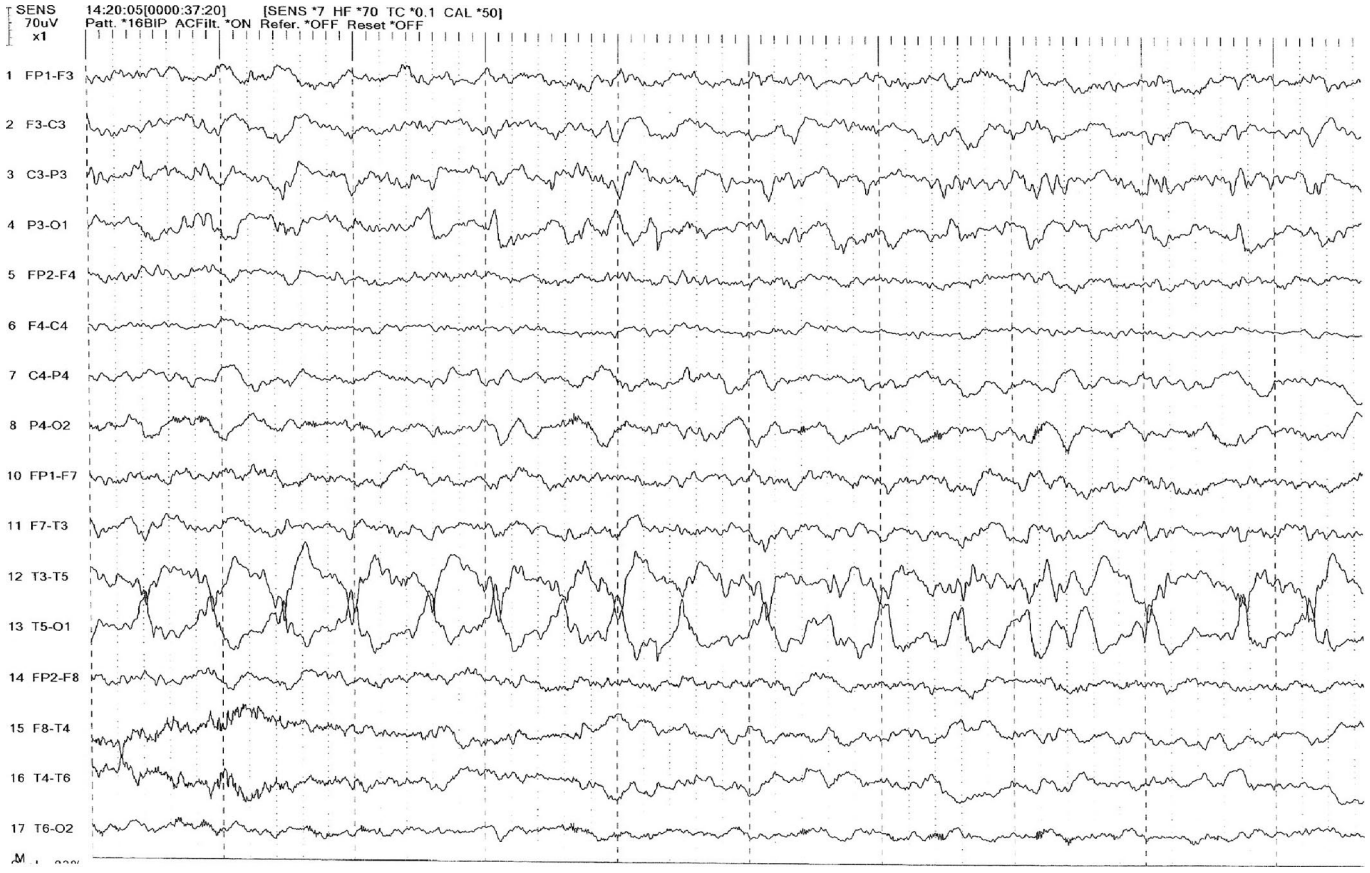

FIGURE 1. Abbreviated EEG shows continuous left temporal sharp and slow wave in a patient who presents in NCSE.

DISCUSSION

Our study indicates that a 5-minute AbEEG using a preformed electrode placement pattern can be successfully incorporated in the early assessment of patients who present to the ER with mental status changes of unknown cause and provides useful and immediate information in this scenario without interrupting the routine workup of these patients. Of the 25 patients enrolled in the study, 2 (8%) presented in NCSE initially detected by AbEEG and confirmed by routine EEGs. In our sample, the presence of diffuse slowing on AbEEG was also highly suggestive of mental status changes due to DENNE.

The incidence of NCSE in our study sample is similar to that obtained by Towne and others (2000) (although their study focused solely on standard EEG recordings obtained in comatose patients). However, our incidence is less than the 37% obtained by Privitera and others (1994). Interestingly, both patients who presented in NCSE in this study did not have a history of prior seizures. This finding contrasts with the works of other authors (Kaplan, 1999; Tomson et al., 1992) that showed that NCSE occurs more often in individuals who already have a prior history of seizures.

A major concern is that a 5-minute AbEEG recording may not be sufficient to consistently detect cases of NCSE. In fact, Claassen and others (2004) demonstrated that up to 20% of patients with unexplained coma need to be monitored for more than 24 hours before electrographic seizures can be documented. In our study, there was no instance where the AbEEG did not show electrographic seizure activity yet it was found later that the patient was in fact in NCSE. However, further studies with larger sample sizes are still needed to determine whether a 5-minute AbEEG is generally sufficient to detect most cases of NCSE in the ER setting. Another concern is that the use of a preformed electrode placement setting and the failure to check impedances and perform activating procedures would result in poorer quality EEG studies. Admittedly, the overall quality of the AbEEGs in our study were lower and two recordings were frankly uninterpretable, but we were still able to obtain readable AbEEG recordings in 23 of 25 patients (92%).

This study has several limitations. The small sample size prevents more robust statistical and subgroup analysis. As stated, it is important to replicate this study in a larger number of patients across different medical institutions using standardized testing conditions to more accurately determine the clinical effectiveness and utility of this technique. Also, admission into the study was highly dependent on the ER physician's discretion, and this could

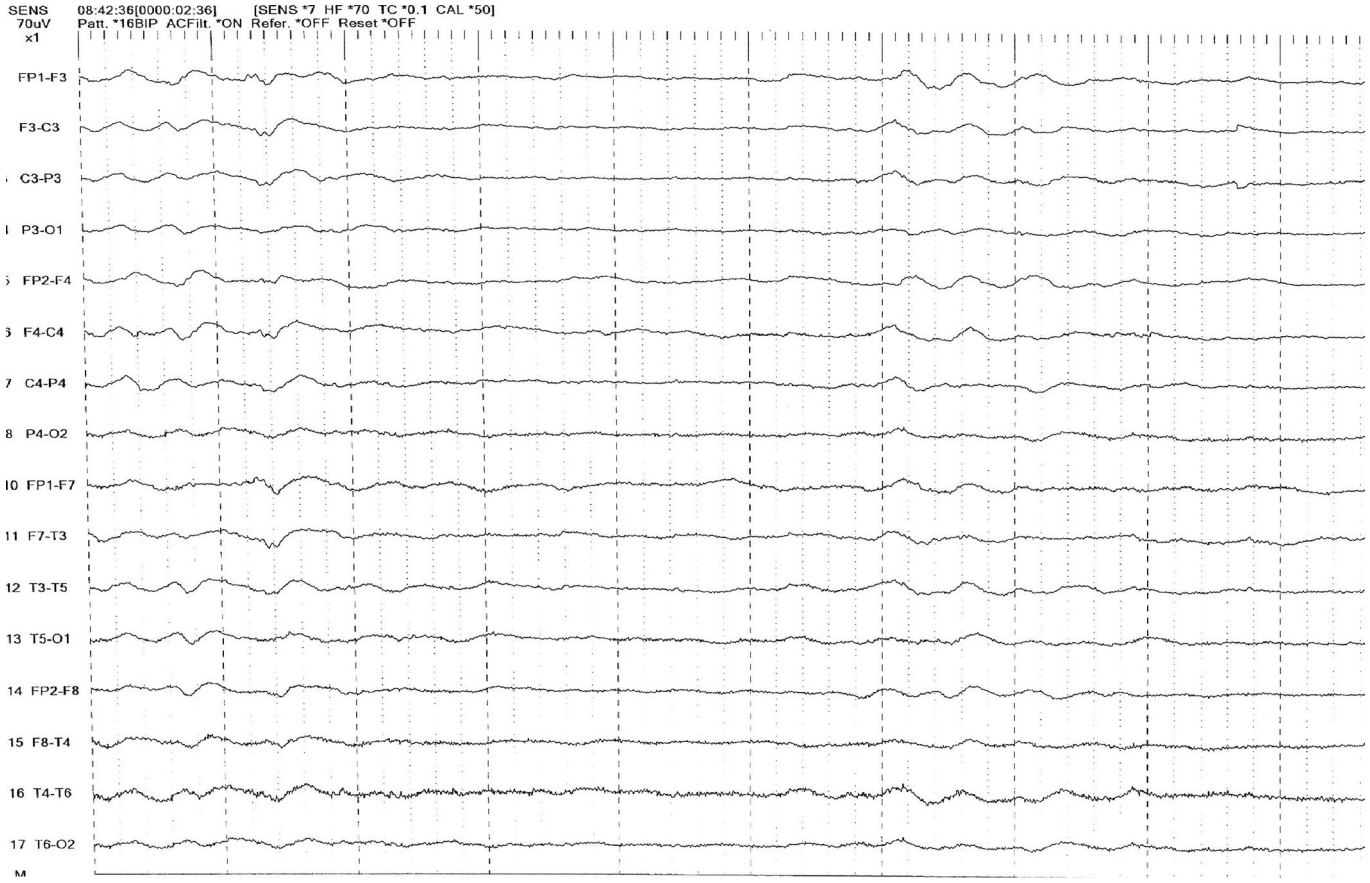

FIGURE 2. Abbreviated EEG shows diffuse slowing inpatient with anoxic encephalopathy.

have resulted in selection bias (Ransohoff and Feinstein, 1978). It is possible that some causes of altered mental status could have been immediately apparent to a neurologist and not to the ER physician. Also, clinical symptoms could have been resolving by the time the AbEEG was performed thereby affecting the recording. In this study, we strove to replicate realistic clinical scenarios where neurologists are not always immediately available to evaluate ER consults and the ER physician performs the initial evaluation and assessment.

The results of this study should stimulate further research leading to more frequent and earlier use of the EEG, especially in situations where immediate information can critically influence patient care. Further studies should also focus on the rigorous development of AbEEG protocols for select clinical scenarios.

With proper training, ER personnel can learn to apply the preformed electrodes and perform the AbEEG. It is also conceivable that through digital transmission of data, immediate interpretation of EEGs can be made by qualified neurologists, thus expediting patient care. Although the use of AbEEG in this scenario should not be made a substitute for neurologic consultation, many emergency rooms do not have immediate access to neurologic expertise. In these situations, the data obtained from AbEEG recordings may be useful in patient management.

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