

Review Article

EEG in ICU: A monitoring tool for critically ill patient

Selina Husna Banu¹

Abstract:

Electroencephalographic monitoring provides dynamic information about the brain function that permits early detection of changes in neurologic status, which is especially useful when the clinical examination is limited. Identification of ongoing electrographic seizures, non-convulsive status epilepticus (NCSE), periodic epileptogenic discharges (PED), irreversible cerebral dysfunction i.e., isoelectric tracing would help the care providers in appropriate decision making regarding the management. Non-convulsive seizures (NCSz) are more common than previously recognized and are associated with worse outcome if not treated in time. Majority of seizures at the ICU are not clinically identified because of the disease phenomena or as the patient may remain under sedation. Studies revealed the first NCSz within 1 to 24 hours of EEG monitoring; longer period of monitoring is required in comatose patient and those with PED. Factors associated with an increased risk of NCSz and NCSE include coma, prior clinical seizures, CNS infection, trauma, stroke, hypoxic ischemic encephalopathy, brain tumor, recent neurosurgery, and PED. In resource-poor situation, EEG is frequently requested to confirm brain death, particularly where there is limited information on neurological examination or inconclusive apnea test; or when the patient is in prolonged state of coma. Presence of isoelectric tracing for at least 30 minutes in the EEG along with other clinical evidences is helpful in such situations.

Extreme care should be taken for recording and reviewing continuous EEG (cEEG) monitoring at the ICU where sources of electrical noise are present. Patients identified with electrographic seizures and mild to moderate degree encephalopathy, with presence of normal background activities had better outcome compared to those with PED, monorhythmic alpha beta coma and severe generalized encephalopathy.

Real-time detection of ischemia at a reversible state is technologically feasible with cEEG and should be developed into a practical form for prevention of in-hospital infarction.

Brain function monitoring with EEG is useful and this is in great demand at the ICU of present time. Such monitoring can help to improve neurological outcome in a variety of ICU settings.

Key Words: EEG, Electroencephalogram, Subclinical Seizure, Intensive Care.

Introduction:

Critical care unit (CCU) or intensive care unit (ICU), is a special department of a health care facility. Common conditions treated at the ICUs include those in the state of coma with or without an immediate history of overt seizure, acute stroke, head injury, multiple organ failure, post operative slow or non recovery, cardio-respiratory failure, cardiac arrest and sepsis¹. Patients are transferred from an emergency department, or from a ward if they rapidly deteriorate; or immediately after surgery if the surgery is majorly invasive and the patient is at high risk of complications². First ICU was established in Copenhagen (1953) in response to a polio epidemic where many patients required constant ventilation³. The first application in the United States was in 1955⁴.

There was limited facility to assess the brain function at the

ICU until recently. Electro-encephalographic (EEG) monitoring is introduced recently that explored the fact that non-convulsive seizure attacks are common, that remain unrecognized on physical examination^{5,6}. The use of electroencephalography (EEG) in the ICU is not widely discussed or evaluated even in advanced countries.

Why it is important to identify the electrographic seizures or non-convulsive seizures?

Delayed recovery or deteriorated clinical condition in a critically ill patient is the major consequence of unidentified electrographic seizure. In addition, later negative effect on the speech-communication, attention and -behavior can be presumed through extrapolated information. Studies have suggested that these are affected by continuous spike wave of slow sleep (CSWSS), a specific EEG findings that had been identified long ago^{7,8}.

Technical aspect of EEG:

Electro-encephalography is the recording of the difference in voltage between at least 2 electrode sites on the scalp to detect brain activity, in a conscious or an unconscious patient. It involves multiple electrode placement on the scalp, connection of the electrode wires to an amplifier (head box), which is then connected to the monitor to display the wave pattern. Routine test (rEEG) is performed for a minimum period of 30 minutes. Emergency EEG (emEEG) is required in acutely ill patients, with an objective to prognostic evaluation, to assess the level of sedation, identify ongoing

1. Selina Husna Banu, MBBS, DCH, PhD, specialist Fellow in clinical Neurophysiology, ICH and GOSH, University College London, UK. Presbyterian Hospital, NY, USA. Dept of Clinical Neurophysiology and Sleep Clinic in Oxford University College Hospital, UK, Clinical Neurophysiologist and Child Neurologist. Associate professor Neurosciences dept. Institute of Child Health and Shishu Sasthya Foundation Hospital, Mirpur, Dhaka.

Corresponding Author:

Selina Husna Banu
Associate professor, Neurosciences Department
ICH and SSF Hospital, Neurosciences center
6/2 Barabag, Mirpur, Dhaka.
Email: selinabanu17@gmail.com

neuronal discharges or electrographic seizures and would assist in medical treatment. In some situation, EEG can assist the confirmation of brain death. Most of the critically ill patients would need a continuous EEG monitoring (cEEG), i.e., continuous digital EEG recorded for hours, days or weeks⁹. Duration of cEEG varied from hours to several days depending on the problem and clinical suspicion in different studies^{5,6,10}. To address the question, “how long is enough time for monitoring in cEEG and whether a routine EEG is adequate”, Pandian et al¹⁰ performed a rEEG for 30-minuted before their prolonged, digital VIDEO EEGs in 105 patients; seizures were detected in 11% and 27% with rEEG, and cEEG (median duration 2.9 days, $p=0.01$) respectively. Therefore, rEEG may detect less than half of seizures eventually identified by longer cEEG recording. One study⁵ identified the first seizure on cEEG in the first hour of recording in 50% among total 110 patients (56 non-comatose and 54 comatose). Studies in both adults⁵ and children¹¹ have reported that 80–95% of seizures are detected within 24 hours, slightly longer durations are needed in comatose patients. Longer recording period is suggested to detect NCSzs in comatose patient or if periodic epileptiform discharges are seen¹².

EEG findings in a critically ill patient: The EEG could reveal any-thing between normal cerebral activities for the patient's age and state (Fig 1 & 2) to severe dysfunctions. It

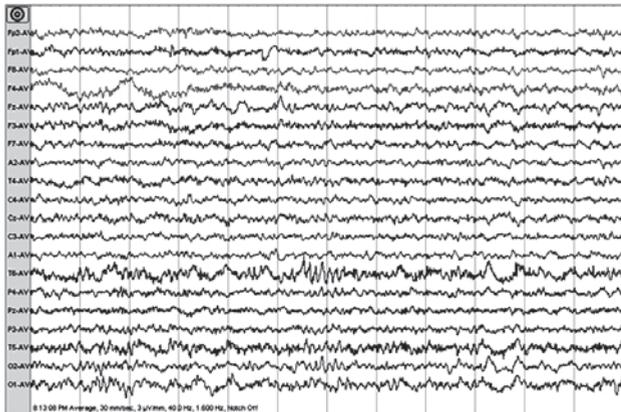


Fig-1

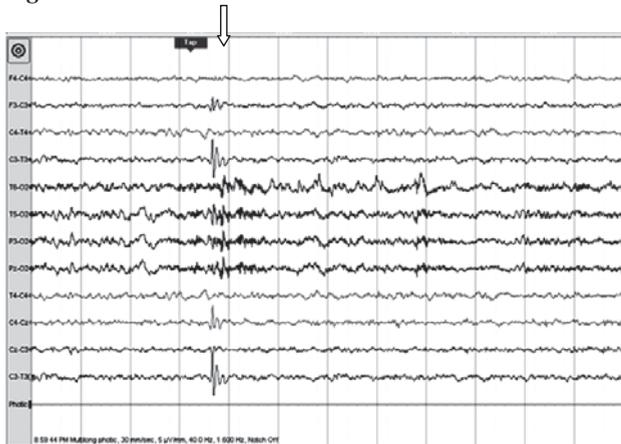


Fig-2

Tactile stimulation

may show distortion of normal background activities or abnormal pattern without any normal background activities in between e.g., burst suppression pattern, periodic complexes. Figure 1,2: A 25 year male, in unexplained non-improving coma state for 16 days, emEEG was called to find any supportive evidence of brain death. Note cerebral activity and reactivity to tactile stimulation. Occasional epileptogenic activities were noted over the temporal parietal area predominantly over the left side of the brain. The EEG excluded cerebral death at this stage. The patient was discharged with partial recovery, farther recovery later.

The immediately treatable electrical condition is “continuous or frequent spike –wave discharges or electrographic seizures (Fig- 3) without any overt seizure in a comatose patient.



Figure – 3: NCSE, Post ictal non-recovery, 5 year boy, unconscious for 5 days, no recognizable seizure for 5 days. Note continuous, high amplitude, 2 c/s spike-wave complexes involving all the channels. sensitivity – 20 μ V/mm.

Other dysfunctions include non-reactive-monorhythmic activities (e.g., alpha-beta coma) (Fig 4); localized or generalized delta wave activities; or iso-electric tracing (Fig 5).

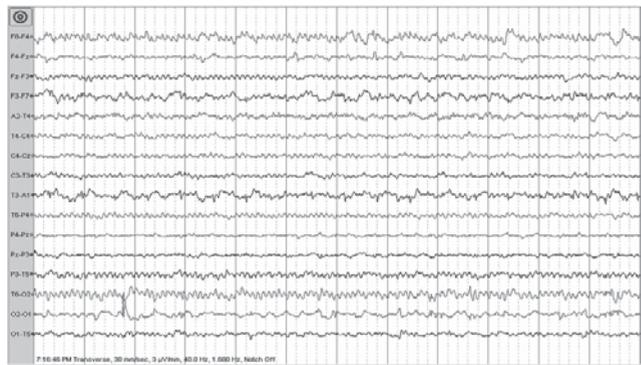


Figure 4: A 22 yr male, on artificial Ventilator for 2 wks. Note, the EEG showing non-reactive, very low amplitude 9-11c/s, monorhythmic activities in the background (alpha coma state). The patient expired on the next day.

Correlating with other evidence this may indicates brain death. The EEG findings have diagnostic and prognostic value and may help in the treatment plan of a critically ill patient.

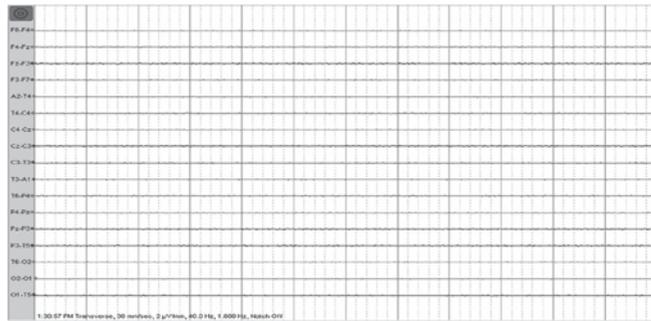


Figure 5: Isoelectric, non-reactive EEG tracing in all the channels in a 3 ½ month boy, post-operative non-recovery, unconscious for 4 days (sensitivity= 2 µV/mm) .

Electro-clinical correlation involves appropriate timing and duration of the EEG recording and is important for brain function monitoring. During data analysis the neurophysiologist should consider the previous history of seizure, primary etiology of the present illness, present medication with dose and recent clinical change⁶.

Pitfalls and challenges:

The EEG is a non-invasive way to assess the brain functions with certain limitations and challenge. On the first instance, patient selection, optimum time for the brain function monitoring is important. High quality cEEG recording in the ICU is a significant challenge. It is necessary to have adequately trained technologist to connect patients with the monitoring equipment and maintain those connections for many hours. Critically ill patients are frequently repositioned, and often undergo multiple procedure and diagnostic tests, including MRI ordered simultaneously. Choice of electrode (MRI compatible electrodes), paste to keep the electrodes in position (collodion, a durable nitrocellulose-based paste to secure disk electrodes) and checking the electrodes are suggested. Newer electrodes, such as subdural wires, which may be more secure and lead to less skin breakdown, may be appropriate for comatose patients^{13,14,15}. The next challenge is the labor intensive EEG data collection and interpretation.

There are numerous sources of artifacts make EEG interpretation difficult, some are easy to identify, such as 60 Hz (50Hz in Europe) line noise from nearby electrical equipment. Artifacts from dialysis machine, cooling blankets, pacemakers, chest percussion, vibrating beds and IV drips may be difficult to avoid and sometimes mimic seizure-like phenomena or PEDs. cEEG with VIDEO recording is strongly recommended, that helps to recognize subtle seizures and clinical events that mimic seizures, also useful for artifact recognition.

Challenge for the countries with limited resources also includes the cost and time management for the cEEG. This problem could be managed through some modification i.e., emergency EEG with repeated brief period (30 – 60 minutes) of VIDEO- EEG recording and judicious patient selection. A research group has reported a study result on emergency EEG (25 EEGs) on 20 critically ill patients of mean age 14 years ranging from 1 month to 68 year (st deviation 20.2) performed

in different ICUs of Dhaka city¹⁶. Clinical conditions categorized as “patients in unexplained coma” for over 2 to 4 wks period in 40% ; “post-convulsive non-recovery” in 35%, “post operative complication” in 20%, neonatal “hypoxic ischemic encephalopathy” (HIE) 5%. None had recognizable seizures during EEG recording. The EEG recording was performed for 30 to 60 minutes. EEG features were categorized as “severe generalized encephalopathy with non-reactive delta waves” (40%), “isoelectric tracing” (27%); “epileptogenic discharges” in 20%; “alpha-beta coma” (13%). For correlation analysis with the clinical outcome, the EEG findings were re-categorized as 1. “irreversible cerebral dysfunction(ICD)” (isoelectric tracing and alpha-beta coma); 2. Severe generalized encephalopathy (SGE) and 3. Localized and/ or generalized epileptiform discharges with other dysfunction (LGEOD). Clinical outcome revealed significant correlation (p<0.05) with 100% mortality in those with ICD and 9% with SGE. Recovery ‘partial’ and ‘total’ was reported in 67% and 33% in those showing LGEOD; and 55% and 10% of those with SGE respectively. The researchers concluded that emEEG is useful to take appropriate decision at the ICU, particularly regarding continuation of ventilator support in a resource poor situation¹⁶.

Detection of non-convulsive seizures (NCSz) and non-convulsive status epilepticus (NCSE):

NCSz are electrographic seizures with little or no overt clinical manifestations commonly found in neonates, may occur in apparently well functioning children or adults, increasingly detected in comatose patients. NCSE is a condition with continuous or near continuous electrographic seizures of at least 30 minutes duration. Presence of NCSz or NCSE would delay the recovery process or may deteriorate the condition even when the primary cause of coma is treated well. Diagnosis of NCSz and NCSE are possible by the EEG test and are increasingly recognized as common occurrences in the critically ill patients. Over 8% - 48% of the comatose patients may have NCSz, depending on which patients are studied^{5,6,9,10,11,12,17,18, 19,20,21,48}.

Clinical feature: Common manifestation of NCSE or NCSz in critically ill patients is a depressed level of consciousness or non-improving, static condition²¹. Most patients with NCSz have purely electrographic seizures (figure 3),⁵, but subtle signs such as face and limb myoclonous, stereotyped movement, nystagmus, eye deviation, pupillary abnormalities (including hippus), and autonomic instability can be identified²³⁻³⁰. None of these clinical signs are highly specific of NCSz, and they are often noted under other circumstances in the critically ill patients; thus, EEG is necessary to diagnose NCSz and NCSE.

Patients with NCSzs are not exclusively in the neurology ICUs; studies on comatose patients from any ICU¹², pediatric ICU¹⁸, or patient having unexplained altered mental status anywhere in the hospital have identified ongoing NCSz in 8%-37%, suggesting that at-risk patients can be found in any critical care setting^{8,21,22,23,24,25,26}. It is important to note that many of the studies are retrospective and included some

patient for whom there was a high suspicion for NCSz based on previous history of seizures, rhythmic movements or a possibly epileptogenic injury potentially contributing to the high rate of NCSz observed in some studies.

In a prevalence study¹², EEG evaluation of 236 comatose patients of all ages has concluded that NCSE is an under-recognized cause of coma, occurring in 8% of all comatose patients without clinical manifestation. Therefore, EEG should be included in the routine evaluation of comatose patients even if clinical seizure activity is not apparent.

The underlying cause or etiologies for the NCSz and NCSE in ICU patients are not exactly identified, they have some common associations. These include acute structural lesions, intracranial hemorrhage, head injury, infections, infarctions, stroke, metabolic derangements, toxins, withdrawal and epilepsy.

NCSzs following convulsive status epilepticus (SE)

Presence of continuous electrographic seizures has been identified in many cases after control of convulsive SE^{21,27,28,29,32,33,61}. In most of the patients coma was the only clinical manifestation. The mortality rate was also more than two fold higher among those patients identified with NCSE compared to those who recovered with cessation of convulsion³². Therefore, cEEG monitoring should be performed on any patient who does not quickly regain consciousness after a convulsive seizure to detect ongoing seizure activity.

Cerebral hemorrhage

Cerebral hemorrhage, including intra-cerebral, subarachnoid, subdural hematoma, from any underlying cause, e.g., stroke, ruptured aneurysm, tumor, trauma, etc. can be irritating to the surrounding neurons. If the neurons become very irritated and/or hyperactive, seizures can occur. In such case seizure may remain clinically undetectable because of the fact that the patient may remain under deep sedation or in deep coma. NCSzs were identified in 18% - 21% of patients with intracerebral hemorrhage (ICH)^{19,20}. cEEG findings may also predict outcome after ICH. Vespa et al³⁴ and Claassen et al¹⁹ found that NCSz were associated with increased mid-line shift and with expansion of hemorrhage volume that led to worse outcomes. Periodic epileptiform discharges (PEDs) in cEEG was found to be an independent predictor of poor outcome (death, vegetative state, minimally conscious on discharge)²².

Traumatic Brain injury (TBI):

Early post traumatic seizures (EPTS) is a common occurrence found in previous studies^{20, 35,36}, however, because of the widespread use of seizure prophylaxis after TBI, acute clinical seizures have become less common, occurring in <1% in one large study³⁷. In 96 consecutive patients with moderate to severe TBI underwent cEEG, 22% of the patients had seizures, half of them had only NCSz³⁸. Some studies have shown that EPTS is an independent risk factor for adults³⁹ and children⁴⁰ with severe TBI.

Post-operative complications:

Postoperative clinical seizures are common association with neuro-surgical procedures, especially those involving the supratentorial lesions (in 4%-17% cases)⁴¹⁻⁴⁴ and in patients, with history of presurgical epilepsy (34%)⁴⁴. Incidence of NCSz and NCSE in post operative patients has not been studied, however, should be considered as contributing factor in post-operative unusual behavior, movement or delayed recovery.

Table 1. Indication for Emergency-EEG (American College of Emergency Physicians 2004)

- | |
|---|
| <ol style="list-style-type: none"> 1. Refractory SE 2. Persistent altered consciousness 3. Suspected NCSE after generalized convulsive SE (failing to return to the normal behavior or cognitive state after convulsive SE) 4. Pharmacological paralysis - deep sedation 5. Coma 6. Suspected Brain death |
|---|

Hypoxic Ischemic Injury (HIE):

A series of comatose patient, 42% identified with NCSE had hypoxic/anoxic injury¹², and 20% of the patient with hypoxic ischemic injury monitored by cEEG in Columbia series had seizures, most of which were NCSz⁵. Presence of clinical seizure or decreased mental status after cardiac arrest is suggested to be the indication for EEG monitoring⁴⁵. In addition, with recent use of hypothermia after cardiac arrest for neuroprotection cEEG might be a help to distinguish shivering from seizures especially during rewarming period⁴⁶

Toxic-metabolic encephalopathy:

Overt and subclinical seizures or change of mental status are not unusual consequences of hypo-, or hyperglycemia, hyponatremia, hypocalcemia, drug intoxication or withdrawal, hepatic failure, uremia, sepsis³¹. In the Columbia cEEG study 20% of the primary diagnosed cases of toxic-metabolic encephalopathy had NCSz⁵. In other series, 5%-25% of patients with NCSz had metabolic derangements as the likely etiology^{12,47}.

A study on 201 medical ICU patients revealed PED or seizures in 22%; sepsis and acute renal failure were significantly associated with electrographic seizures⁴⁸.

Patients in Pediatric ICU

Incidence of NCSz and NCSE are probably more frequent, however, less reported in younger age and infants^{5,49}. NCSz and NCSE was identified among 23%, 33% and 44% in critically ill children studied^{11,50, 52}. The most common associations and etiology identified were previous history of epilepsy, hypoxic ischemic injury and stroke^{11,51,52}. Out of 183 infants having cardiopulmonary operation for congenital heart defect 11.5% were identified with NCSE as post surgery complication⁵³.

Acute brain ischemia or acute stroke:

Patients with primary diagnosis of stroke may show the first supportive evidence in their EEG, where changes could be detected within seconds of reduction in cerebral blood flow (CBF)^{54,55}. This is the basis for intra-operative EEG monitoring for patients undergoing surgeries with a high risk for cerebral ischemia, such as carotid endarterectomy^{56,57,58}. As the CBF decreases below 25-30 mL·100g⁻¹·min⁻¹ there is a progressive loss of higher frequency and prominent slowing of background EEG activity noted. When CBF is below 8-10 mL·100g⁻¹·min⁻¹, low enough to cause cell death, all EEG frequencies are suppressed^{59,60}. EEG monitoring can detect ischemia at the early stage and provides a window of opportunity to prevent permanent brain injury. This is important as thrombolytic and endovascular therapies have been shown to be effective in acute stroke and vasospasm, especially when treatment is provided very early^{61,62}.

Table 2. Indication for continuous EEG monitoring (cEEG)

1. Detection of subclinical seizures (NCSz) and Characterization of spells in patients with **altered** mental status/ or conscious level
 - a. Particularly in patients with previous history of epilepsy/seizures
 - b. Recent convulsive status epilepticus
 - c. Acute brain injury with altered mental status
 - d. Fluctuating mental status
 - e. Unexplained alteration of mental status
 - f. Stereotyped, paroxysmal or repetitive movements or episodic posturing,
 - g. Subtle twitching, jerking, nystagmus, eye deviation, chewing
 - h. Paroxysmal automatic spells including tachycardia
2. Monitoring ongoing therapy
 - a. Assessment of level of sedation
 - b. Induced coma for elevated intracranial pressure or refractory status epilepticus
3. Management of burst-suppressions in anesthetic coma
4. Detection of Ischemia
 - a. After subarachnoid hemorrhage
 - b. During and After vascular neurosurgical or interventional neurocardiology procedures
 - c. In patients with hemodynamic lesions and borderline flow
 - d. In other patients at risk for in-hospital acute ischemia
5. Prognostication
 - a. Following cardiac arrest
 - b. Following acute brain injury
 - c. Encephalopathy of infective or other origin

Recent advances in EEG technique with real-time application of quantitative algorithms (qEEG) have allowed for extracting time-frequency data to measure change in the background EEG rhythms. Visual review of simple values produced by cEEG recording is useful to detect cerebral hypoperfusion, especially in comatose and sedated patients when clinical examination is limited. qEEG is used for the detection of ischemic stroke and delayed cerebral ischemia (DCI) due to vasospasm after subarachnoid hemorrhage (SAH). However, its value in timely detection of vasospasm and cerebral ischemia is well analyzed and reviewed in different retrospective studies^{63,64,65} with sensitivity 100% and specificity from 50% - 84%. cEEG with the specific algorithm (qEEG) is proved to be useful for ischemia detection and prognostification^{66,67}.

Efficacy of therapy

Treatment of refractory status epilepticus (SE) with IV infusions i.e., midazolam, propofol, or pentobarbital under EEG monitoring is useful technique⁶⁸. qEEG based tools, such as Bispectral index⁶⁹, patient state index⁷⁰, and narcotrend⁷¹ have been in use in operating room and ICU for more than a decade to monitor depth of sedation.

Confirmation of brain death:

Brain death is referred to the complete, irreversible, permanent loss of all brain and brainstem functions. EEG might serve as an auxiliary and useful tool in the confirmatory tests for adults and children^{72,73,74}. Typically, isoelectric EEG recording is required at least for 30 min⁷⁵. Confirmation of brain death is urgent in certain ICU situation, particularly in resource poor condition where maintenance of artificial ventilation costs high. In addition, there is a need to diagnose brain death with utmost accuracy and urgency because of an increased awareness amongst the masses for an early diagnosis of brain death and the requirements of organ retrieval for transplantation.

The diagnosis of brain death is primarily clinical. Ancillary testing is ordered only if clinical neurological examination cannot be fully performed due to patient factors, or if apnea testing is inconclusive and aborted, or is not performed due to patient factors. Only one ancillary test among the five (cerebral angiogram included CT or MR angiogram, Nuclear brain scan HMPAO SPECT, EEG, cerebral perfusion scintigraphy (CPS) needs to be performed (step 5 of the guideline)⁷⁶. Considering the availability, time and cost effectiveness, EEG monitoring at least for 30 minutes is suitable in our situation. Visual evoked potentials (EP), somatosensory EPs, and brain stem auditory EPs (BAEPs) can also be used.

Conclusion:

On arrival of a critically ill or a comatose patient at the ICU, it is mandatory to monitor cardiopulmonary physiology, however, an equipment to monitor the brain physiology, a vital organ that is obviously dysfunctional in this case, is unavailable to the ICU staff in most of situation. In a comatose patient, there is hardly a few examinations that can be reliably followed to assess worsening brain injury. The

situation is worst in patients who are sedated and possibly paralyzed. Neuroimaging provides information about structural brain injury often after it is irreversible and cannot reveal functional changes, such as seizures and level of sedation. "Time is brain", therefore cerebral function monitoring through a non-invasive technique is necessary for patients at risk for neuroprotection. Recent advances in computer technology, networking and data storage have made cEEG monitoring practical and its use is common in many non-neuroscience ICUs. Methods of analyzing and compressing the vast amounts of data generated by cEEG have allowed neurophysiologists to more efficiently review recording from many patients monitored simultaneously and provide timely information for guiding treatment. This article reviewed the use of EEG at the ICU with limitations and pitfalls, discussed different study findings, current indications and potential uses for emEEG and cEEGs (table 1,2). We believe that EEG monitoring should be included in the ICU management protocol.

References:

1. "What is Intensive Care?". London: Intensive Care Society, 2011. Retrieved 2013-05-25.
2. Smith, S. E. (2013-03-24). "What is an ICU". *wiseGEEK*. Bronwyn Harris, ed. Sparks, Nevada: Conjecture Corporation. Retrieved 2012-06-15.
3. Takroui, M.S.M. (2004). "Intensive Care Unit". *Internet Journal of Health* (Sugar Land, Texas: Internet Scientific Publications) **3** (2). doi:10.5580/1c97. ISSN 1528-8315. OCLC 43535892. Retrieved 2007-08-25.
4. Grossman, D.C. (Spring 2004). "Vital Signs: Remembering Dr. William Mosenthal: A simple idea from a special surgeon". *Dartmouth Medicine* (Dartmouth College, Geisel School of Medicine) **28** (3). Retrieved 2007-04-10.
5. Claassen J, Mayer SA, Kowalski RG, Emerson RG, Hirsch LJ. Detection of electrographic seizures with continuous EEG monitoring in critically ill patients. *Neurology*. 2004;62:1743-8.
6. Abend NS, Dlugos DJ, Hahn CD, Hirsch LJ, Herman ST. Use of EEG monitoring and management of non-convulsive seizures in critically ill patients: a survey of neurologists. *Neurocrit Care* 2010 Jun;12 (3):382-9.
7. Paquier P.F, Van Dongen H.R., Loonen C.B, The Landau-Kleffner syndrome or 'acquired aphasia with convulsive disorder' Long-term follow-up of six children and a review of the recent literature. *Arc. Neurol*, 1992, 49: 354-359
8. Roulet Perez E., Davidoff V., Despland P.A., Deonna T. Mental and behavioural deterioration of children with epilepsy and CSWS: Acquired epileptic frontal syndrome. *Dev. Med Child Neurol* 1993, 35: 661-674
9. Hirsch LJ, Continuous EEG monitoring in the intensive care unit, an over view. *Journal of C. Neurophysiology*, 2004; 21(5): 332-39)
10. Pandian JD, Cascino GD, So EL, Manno E, Fulgham JR. Digital video-electroencephalographic monitoring in the neurological-neurosurgical intensive care unit: clinical features and outcome. *Arch Neurol*. 2004;61:1090-4.
11. Saengpatrachai M, Sharma R, Hunjan A, Shroff M, Ochi A, Otsubo H, Cortez M, Snead Carter. Nonconvulsive seizures in the pediatric intensive care unit: etiology, EEG, and brain imaging findings. *Epilepsia*. 2006;47:1510-18.
12. Towne AR, Waterhouse EJ, Boggs JG, et al. Prevalence of nonconvulsive status epilepticus in comatose patients. *Neurology*. 2000;54:340-5.
13. Kull LL, Emerson RG. Continuous EEG monitoring in the intensive care unit: technical and staffing considerations. *J Clin Neurophysiol* 2005;22:107-18
14. Mirsattari SM, Lee DH, Jones D, Bihari F, Ives JR. MRI compatible EEG electrode system for routine use in the epilepsy monitoring unit and intensive care unit. *Clin Neurophysiol* 2004;115:2175-80
15. Young GB, Campbell VC. EEG monitoring in the intensive care unit: pitfalls and caveats. *J Clin Neurophysiol* 1999;16:40-5
16. Report on em EEGs on 20 ICU patients. Reported at the national conference BCNEPS, November 2013.
17. Jordan KG. Neurophysiologic monitoring in the neuroscience intensive care unit. *Neurol Clin*. 1995;13:579-626.]
18. Jette N, Claassen J, Emerson RG, Hirsch LJ. Frequency and predictors of nonconvulsive seizures during continuous electroencephalographic monitoring in critically ill children. *Arch Neurol*. 2006;63:1750-5.
19. Claassen J, Jette N, Chum F, et al. Electrographic seizures and periodic discharges after intracerebral hemorrhage. *Neurology*. 2007;69:1356-65.
20. Vespa PM, Nuwer MR, Nenov V, Ronne-Engstrom E, Hovda DA, Bergsneider M, Kelly DE, Martin NA, Beker DP. Increasing incidence of and impact of Nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic. *J. Neurosurg*. 1999;91:750-60.
21. DeLorenzo RJ, Waterhouse EJ, Towne AR, et al. Persistent nonconvulsive status epilepticus after the control of convulsive status epilepticus. *Epilepsia*. 1998;39:833-40.
22. Claassen J, Hirsch LJ, Frontera JA, Fernandez A, Schmidt M, Kapinos G, Wittman J, Connolly ES, Emerson RG, Mayer SA. Prognostic significance of continuous EEG monitoring in patients with poor-grade subarachnoid hemorrhage. *Neurocrit Care* 2006;4:103-12
23. Abend NS, Topjian A, Herman ST, et al. Electroencephalographic monitoring during hypothermia after pediatric cardiac arrest. *Neurology*. 2009;72:1931-40.
24. Jirsch J, Hirsch LJ, Nonconvulsive seizures: developing a rational approach to the diagnosis and management in the critically ill population. *Clin Neurophysiol* 2007; 118: 1660-70
25. Lowenstein DH, Aminoff MJ, Clinical and EEG feature of status epilepticus in comatose patients, *Neurology* 1992; 42: 100-4
26. Kaplan PW, Behavioral manifestation of nonconvulsive status epilepticus. *Epilepsy Behav* 2002;3: 122-39
27. Hussain AM, Horn GJ, Jacobson MP, Non-convulsive status epilepticus: usefulness of clinical features in selecting patients for urgent EEG. *J Nurol Neurosurg Psychiatry* 2003; 74:189-91
28. Banu SH, Hussain M, Khan NZ. Non-convulsive Status Epilepticus Presenting as Deterioration in School Performance - A Case Report. *Bangladesh J Child Health*. 1998;22(3/4):75 - 77.
29. Banu SH, Mostafa Mahbub, AZM Moshikul Azam, Shipra Rani, Naila Z Khan (2009), Non-convulsive status epilepticus in children, electro-clinical profile and response to a specific treatment protocol, *Bangladesh J. Child Health*, 2009;33(3): 90- 99.
30. 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.
31. Abou Khaled KJ, Hirsch LJ, Advances in management of seizures and status epilepticus in critically ill patients. *Crit Care Clin*, 2006;22:637-59
32. Bauer G, Aichner F, Mayr U, Nonconvulsive status epilepticus following generalized tonic clonic seizures. *Eyr Neurol*. 1982;21:411-19
33. Treiman DM, Meyers PB, Walton NY, Collins JF, Colling C, Rowan AL, Handforth A, Fought 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 Eng J Med*, 1998;339:792-8
34. 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 haemorrhage: a factor in progressive mid-line shift and outcome. *Neurology*, 2003; 60:1441-6

35. Annegers JF, Grabow JD, Groover RV, Laws ER Jr, Elveback LR, Kurland LT. Seizures after head trauma: a population study. *Neurology* 1980; 30:683-939.
36. Temkin NR, Dikmen SS, Wilensky AJ, Keihm J, Chabal S, Winn HR. A randomized, double-blind study of phenytoin for the prevention of post-traumatic seizures. *N Engl J Med* 1990;323:497-502
37. Temkin NR, Anderson GD, Winn HR, Ellenbogen RG, Britz GW, Schuster J, Lucas T, Newell DW, Mansfield PN, Machamer JE, Barber J, Dikmen SS. Magnesium sulfate for neuroprotection after traumatic brain injury: a randomised controlled trial. *Lancet Neurol* 2007; 6:29-38
38. Vespa PM, Continuous EEG monitoring for the detection of seizure in traumatic brain injury, infection, and intracerebral hemorrhage; "to detect and protect". *J Clin Neurophysiol*, 2005; 22: 99-105
39. Wang HC, Chang WN, Chang HW, Ho JT, Yang TM, Lin WC, Chuang YC, Lu CH. Factors predictive of outcome in post traumatic seizures. *J Trauma*, 2008; 64:883-8
40. Chiareth A, Piastra M, Pulitano S, Pietrini D, De Rosa G, Barbaro R, Di Rocco C. Prognostic factors and outcome of children with severe head injury, an 8 year experience, *Child Nerv Syst*, 2002; 18:129-36
41. Foy PM, Copeland GP, Shaw MD. The incidence of postoperative seizures. *Acta Neurochir (Wien)* 1981; 55:253-64
42. Baker CJ, Prestigiacomo CJ, Solomon RA. Short-term perioperative anticonvulsant prophylaxis for the surgical treatment of low-risk patients with intracranial aneurysms. *Neurosurgery* 1995;37:863-70; discussion 870-1
43. Kvam DA, Loftus CM, Copeland B, Quest DO. Seizures during the immediate postoperative period. *Neurosurgery* 1983; 12:14-17
44. Matthew E, Sherwin AL, Welner SA, Odusote K, Stratford JG. Seizures following intracranial surgery: incidence in the first post-operative week. *Can J Neurol Sci* 1980; 7:285-90
45. Rossetti AO, Logroscino G, Liaudet L, Ruffieux C, Ribordy V, Schaller MD, Despland PA, Oddo M. Status epilepticus: an independent outcome predictor after cerebral anoxia. *Neurology* 2007; 69:255-60
46. Hovland A, Nielsen EW, Kluver J, Salvesen R. EEG should be performed during induced hypothermia. *Resuscitation* 2006; 68:143-6
47. 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; 47:83-9
48. Oddo M, Carrera E, Claassen J, Mayer SA, Hirsch LJ. Continuous Electroencephalography in the medical intensive care unit. *Crit Care Med* 2009; 37:2051-6
49. Clancy RR, Legido A, Lewis D. Occult neonatal seizures. *Epilepsia* 1988; 29:256-61
50. Hosain SA, Solomon GE, Kobylarz EJ. Electroencephalographic patterns in unresponsive pediatric patients. *Pediatr Neurol* 2005; 32:162-5
51. Tay SK, Hirsch LJ, Leary L, Jette N, Wittman J, Akman CI. Nonconvulsive status epilepticus in children: clinical and EEG characteristics. *Epilepsia* 2006; 47:1504-9
52. Abend NS, Dlugos DJ. Nonconvulsive status epilepticus in a pediatric intensive care unit. *Pediatr Neurol* 2007; 37:165-70
53. Clancy RR, Sharif U, Ichord R, Spray TL, Nicolson S, Tabbutt S, Wernovsky G, Gaynor JW. Electrographic neonatal seizures after infant heart surgery. *Epilepsia* 2005; 46:84-90
54. Sundt TM Jr, Sharbrough FW, Anderson RE, Michenfelder JD. Cerebral blood flow measurements and electroencephalograms during carotid endarterectomy. *J Neurosurg* 1974;41:310-20
55. Sundt TM Jr, Sharbrough FW, Piepgras DG, Kearns TP, Messick JM Jr, O'Fallon WM. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy: with results of surgery and hemodynamics of cerebral ischemia. *Mayo Clin Proc* 1981; 56:533-43
56. Sharbrough FW, Messick JM Jr, Sundt TM Jr. Correlation of continuous electroencephalograms with cerebral blood flow measurements during carotid endarterectomy. *Stroke* 1973; 4:674-83
57. Zampella E, Morawetz RB, McDowell HA, Zeiger HE, Varner PD, McKay RD, Halsey JH Jr. The importance of cerebral ischemia during carotid endarterectomy. *Neurosurgery* 1991; 29:727-30; discussion 730-1
58. Arnold M, Sturzenegger M, Schaffler L, Seiler RW. Continuous intraoperative monitoring of middle cerebral artery blood flow velocities and electroencephalography during carotid endarterectomy. A comparison of the two methods to detect cerebral ischemia. *Stroke* 1997; 28:1345-50
59. Astrup J, Siesjo BK, Symon L. Thresholds in cerebral ischemia—the ischemic penumbra. *Stroke* 1981; 12:723-5
60. Jordan KG. Emergency EEG and continuous EEG monitoring in acute ischemic stroke. *J Clin Neurophysiol* 2004; 21:341-52
61. Dorsch NWC. Therapeutic approaches to vasospasm in subarachnoid hemorrhage. *Curr Opin Crit Care* 2002; 8:128-33
62. Wardlaw J, Berge E, del Zoppo G, Yamaguchi T. Thrombolysis for acute ischemic stroke. *Stroke* 2004; 35:2914-15
63. Labar DR, Fisch BJ, Pedley TA, Fink ME, Solomon RA. Quantitative EEG monitoring for patients with subarachnoid hemorrhage. *Electroencephalogr Clin Neurophysiol* 1991; 78:325-32
64. Vespa PM, Nuwer MR, Juha'sz C, Alexander M, Nenov V, Martin N, Becker DP. Early detection of vasospasm after acute subarachnoid hemorrhage using continuous EEG ICU monitoring. *Electroencephalogr Clin Neurophysiol* 1997; 103:607-15
65. Claassen J, Hirsch LJ, Emerson RG, Mayer SA. Treatment of refractory status epilepticus with pentobarbital, propofol, or midazolam: a systematic review. *Epilepsia* 2002; 43:146-53
66. Van Putten MJ, Tavy DL. Continuous quantitative EEG monitoring in hemispheric stroke patients using the brain symmetry index. *Stroke* 2004; 35:2489-92
67. de Vos CC, van Maarseveen SM, Brouwers PJ, van Putten MJ. Continuous EEG monitoring during thrombolysis in acute hemispheric stroke patients using the brain symmetry index. *J Clin Neurophysiol* 2008; 25:77-82
68. Abou Khaled KJ, Hirsch LJ. Updates in the management of seizures and status epilepticus in critically ill patients. *Neurol Clin* 2008; 26:385-408
69. Simmons LE, Riker RR, Prato BS, Fraser GL. Assessing sedation during intensive care unit mechanical ventilation with the Bispectral Index and the Sedation-Agitation Scale. *Crit Care Med* 1999; 27:1499-504
70. Prichep LS, Gugino LD, John ER, Chabot RJ, Howard B, Merkin H, Tom ML, Wolter S, Rausch L, Kox WJ. The Patient State Index as an indicator of the level of hypnosis under general anaesthesia. *Br J Anaesth* 2004; 92:393-9
71. Bauerle K, Greim CA, Schroth M, Geisselbrecht M, Kobler A, Roewer N. Prediction of depth of sedation and anaesthesia by the Narcotrend EEG monitor. *Br J Anaesth* 2004; 92:841-5123.
72. Wijdicks EFM (1995) Determining brain death in adults. *Neurology* 45:1003-1011.
73. Taylor RM (1997) Reexamining the definition and criteria of death. *Semin Neurol* 17:265-270.
74. Schneider S (1989) Usefulness of EEG in the evaluation of brain death in children: the cons. *Electroencephalogr Clin Neurophysiol* 73(4):276-278.
75. Wijdicks EFM (2002) Brain death worldwide: accepted fact but no global consensus in diagnostic criteria. *Neurology* 58:20-25.
76. Guidelines for Determining Brain Death. New York State Department of Health and New York State Task Force on Life and the Law, November 2011