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SYSTEMATIC REVIEW

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Recommendations on the use of EEG monitoring in critically ill patients: consensus statement from the neurointensive care section of the ESICM

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Abstract *Objectives:* Recommendations for EEG monitoring in the ICU are lacking. The Neurointensive Care Section of the ESICM assembled a multidisciplinary group to establish consensus recommendations on the use of EEG in the ICU. *Methods:* A systematic review was performed and 42 studies were included. Data were extracted using the PICO approach, including: (a) population, i.e. ICU patients with at least one of the following: traumatic brain injury, subarachnoid hemorrhage, intracerebral hemorrhage, stroke, coma after cardiac arrest, septic and metabolic encephalopathy, encephalitis, and status epilepticus; (b) intervention, i.e. EEG monitoring of at least 30 min duration; (c) control, i.e. intermittent vs. continuous EEG, as no studies compared patients with a specific clinical condition, with and without EEG monitoring; (d) outcome endpoints, i.e. seizure detection, ischemia detection, and prognostication. After selection, evidence was classified and recommendations developed using the GRADE system. *Recommendations:* The panel recommends EEG in generalized convulsive status epilepticus and to rule out nonconvulsive seizures in brain-injured patients and in comatose ICU patients without primary brain injury who have unexplained and persistent altered consciousness. We suggest EEG to detect ischemia in comatose patients with subarachnoid hemorrhage and to improve prognostication of coma after cardiac arrest. We recommend continuous over intermittent EEG for refractory status epilepticus and suggest it for patients with status epilepticus and suspected ongoing seizures and for comatose patients with unexplained and persistent altered consciousness. *Conclusions:* EEG monitoring is an important diagnostic tool for specific indications. Further data are necessary to understand its potential for ischemia assessment and coma prognostication.

Keywords EEG · Intensive care · Seizures · Cerebral ischemia · Prognosis · Recommendations

Introduction

Acute brain dysfunction is a leading cause of admission to the ICU, either due to structural diseases, for example traumatic brain injury (TBI), intracranial hemorrhage, cerebral ischemia and encephalitis, or to functional disorders, for example septic encephalopathy. Electroencephalography (EEG) provides information about brain electrical activity, even when brain function is depressed and cannot be explored otherwise, as in comatose patients. EEG is essential to detect electrical seizures and to document their duration and response to therapy. It can disclose alterations associated with the development of delayed cerebral ischemia (DCI) and improve coma prognostication. It is useful to monitor barbiturate coma for refractory intracranial hypertension [1] and is mandatory in several countries for the diagnosis of brain death [2].

Evidence, however, is sparse, and recommendations for EEG monitoring in the ICU are not well defined. The Neurointensive Care (NIC) Section of the ESICM assembled a multidisciplinary panel to establish a consensus statement on the use of EEG monitoring in adult ICU populations. The aim was to provide better guidance for EEG monitoring and to improve implementation of EEG in ICU practice. Two indications were excluded from this review: EEG for brain death diagnosis, since it is regulated by local legislation in many countries, and for barbiturate coma, since it has been reviewed in authoritative guidelines [1].

Methods

Authors and study selection

In 2010, the NIC section of the ESICM decided to develop evidence-based consensus recommendation on the indications for EEG monitoring for ICU patients. Authors were proposed during an official NIC section meeting and included neurointensivists (N.S., J.C.), medical/surgical intensivists (F.S.T., M.O.), anesthesiologists (N.S.), neurologists (J.C.), neurosurgeons (P.H.) and epileptologists (M.H.) who would review the existing literature and provide a consensus manuscript. This systematic review was reported following the PRISMA criteria [3].

Eligibility criteria

Studies were considered eligible based on the PICO approach, which includes:

(a) Population, i.e. ICU patients with at least one of the following: TBI, subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), acute ischemic stroke (AIS), coma after cardiac arrest (CA), sepsis/metabolic encephalopathy, encephalitis, and status epilepticus (SE).
(b) Intervention, i.e. EEG monitoring of >30 min duration.
(c) Controls, i.e. intermittent vs. continuous EEG, as no studies compared patient population with a specific clinical condition with and without EEG.
(d) Outcome endpoints, i.e. seizure detection, ischemia detection, prognostication.

Search strategy

Using the PubMed database, we conducted a systematic review from 1966 up to August 2012. The search strategy included the terms "EEG" or "electroencephalogram" or "electroencephalography", used with one of the following: "intensive care" or "critical care" or "ischemia" or "prognosis" or "outcome" or "traumatic brain injury" or "subarachnoid hemorrhage" or "intracerebral hemorrhage" or "stroke" or "cardiac arrest" or "sepsis" or "metabolic encephalopathy" or "encephalitis" or "meningitis" or "status epilepticus". Additional references for relevant studies were also searched from review articles. We restricted the language of the articles to English. No unpublished data or congress abstracts were considered.

Study selection

Two authors (M.O. and F.S.T.) independently reviewed citations, abstracts and full-text articles to select eligible studies. We excluded: (a) review articles, (b) case reports, (c) experimental studies, (d) studies in pediatric ICU populations, (e) studies that were not conducted on ICU patients. Data were abstracted (F.S.T.) according to the PICO system. No attempt was made to re-analyze the data; accuracy of data extraction was controlled thereafter (M.O.). No additional process to obtain data from investigators was attempted. Considering the lack of randomized or case-control studies, no meta-analysis of extracted data was performed nor did we assess risk of bias or consistency, or perform subgroup analyses.

Grading of evidence

The quality of evidence was judged based on the grades of recommendation, assessment, development and evaluation (GRADE) system, which assesses the quality of evidence for each of the selected outcomes from the

available studies, considering the benefit/risk balance and the costs related to the study intervention [4, 5]. This system classifies *quality of evidence* as high (grade A), moderate (grade B), low (grade C), or very low (grade D) [6, 7]. Thereafter, *recommendations* are classified as strong (grade 1) or weak (grade 2). One advantage of the GRADE system is that a strong recommendation can be made despite moderate/low evidence. Accordingly, the authors made strong recommendations when they were confident that the desirable effects of adherence to a recommendation would outweigh the undesirable effects. A strong recommendation reflects the possibility that following the given recommendation about EEG will result in more beneficial effects (detection and therapy of seizures, reduced injury associated with ongoing seizures, improved outcome, less burden on staff and patients, cost savings) than harm to ICU patients (inaccurate predictive value, useless antiepileptic drugs (AED), difficult EEG implementation). A weak recommendation reflects the opinion that the benefit/risk balance could be in favor of this recommendation, but the members of the task force were not confident because of limited evidence. Three authors (M.O., F.S.T., J.C.) proposed initial recommendations and asked for approval from the other participants. In case of disagreement, changes to recommendations were proposed and discussed to obtain a unanimous vote. It is important to recognize that strong recommendations do not necessarily represent standards of care.

Results

A total of 42 studies were selected (Fig. 1). All were retrospective or prospective observational single-center studies. No controlled trial—either nonrandomized or randomized—was identified (Table 1). Strong recommendations for EEG use, when given in the absence of high-quality evidence, are justified by the potential harm of unrecognized seizures and the low risk of the procedure; however, costs may be considerable and have to be weighed against the benefit. A summary of GRADE recommendations for the indications for EEG monitoring in the ICU is given in Table 2.

Patient populations

EEG in patients with generalized convulsive SE

Seizure detection Generalized convulsive SE (GCSE) is a clinical diagnosis that does not require EEG. However, nonconvulsive seizures (NCSz) and nonconvulsive SE (NCSE) are frequent (48 % and 14 %, respectively) after GCSE [8] and differentiating ongoing seizure activity from postictal or medication-induced encephalopathy can be challenging. As clinical symptoms are often missing, EEG is necessary to diagnose ongoing NCSz [9, 10]. EEG, especially continuous EEG (cEEG), is urgently required in patients not waking up after cessation of clinical seizures to rule out NCSz [8, 11]. Guidelines for

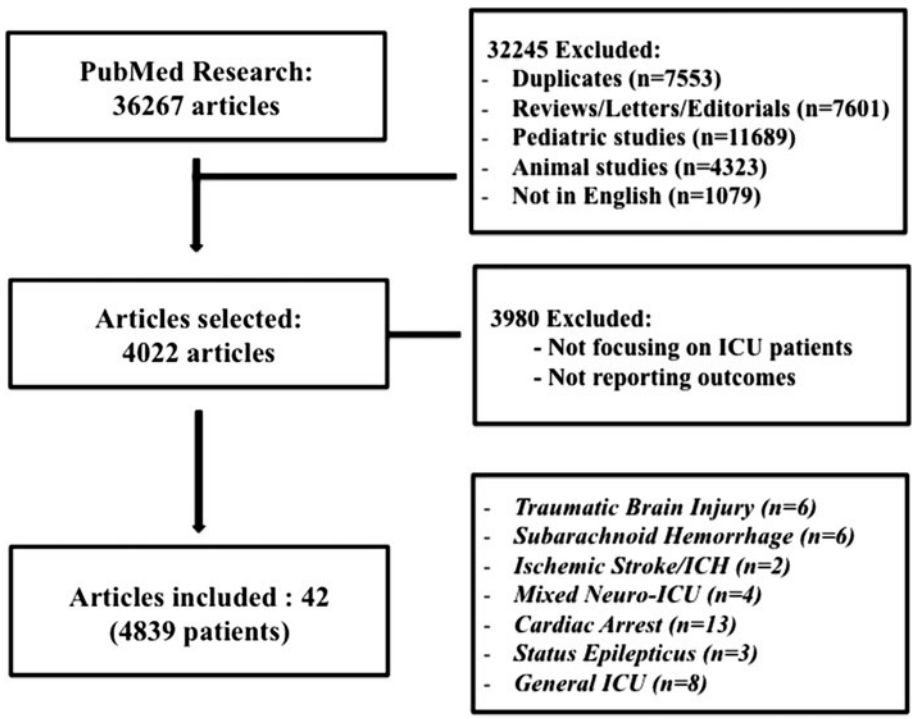

Fig. 1 Flow-chart representing the methodology for the systematic review, according to the PRISMA criteria

Table 1 Selected studies on EEG monitoring in ICU patients

Population	Reference	Study	*N*	Intervention	Risk factors	Seizures	Ischemia	Prognostication
TBI								
	Olivecrona [24]	P	47	cEEG	–	0 %	–	–
	Ronne-Engstrom [23]	R	70	cEEG	–	33 %	–	–
	Vespa [19]	P	94	cEEG	–	12 %	–	–
	Vespa [33]	P	89	cEEG	–	–	–	Reduced percentage of alpha variability was associated with worse outcome (PPV of 86 % for poor prognosis)
	Gutling [31]	P	50	EEG	–	–	–	Non-reactive EEG background predicted poor outcome
	Steudel [32]	P	50	EEG	–	–	–	EEG abnormalities predicted outcome in 80 % of patients
SAH								
	Little [36]	R	389	cEEG	Advanced age, coma, Fisher 3 and 4, hydrocephalus	3 % (NCSE)	–	NCSE had 80 % mortality
	Dennis [35]	R	233	cEEG	Advanced age, coma, brain edema, hydrocephalus	8 % (NCSE)	–	NCSE had 100 % mortality
	Claassen [37]	P	34	cEEG	–	–	>10 % decrease in ADR predictive of DCI (sensitivity 100 %, specificity 76 %)	–
	Vespa [41]	P	32	cEEG	–	–	Decrease in relative alpha variability predictive of DCI (PPV 76 %, NPV 100 %)	–
	Rathakrishnan [39]	P	12	cEEG	–	–	Decrease in mean alpha power predictive of DCI (sensitivity 67 %)	–
	Claassen [42]	R	116	cEEG	–	–	–	Epileptiform discharges, NCSE, non-reactive EEG background predicted poor recovery
ICH								
	Claassen [44]	R	102	cEEG	≥30 % increase in ICH volume	18 % (NCSz)	–	–
Ischemic stroke								
	Sheorajpanday [58]	P	110	cEEG	–	–	–	EEG derived indexes (global ADR and Brain Symmetry Index) predictive of 6-month functional recovery (PPV 60 %)
Mixed neuro-ICU								
TBI, ICH, ischemic stroke	Amantini [25]	P	68	cEEG	–	3 %	–	–
Comatose neuro-ICU	Claassen [17]	R	570	cEEG	Coma, previous seizures	9 % (NCSz), 19 % (NCSE)	–	–
Neuro-ICU GCS ≤12	Kramer [99]	P	393	EEG $n = 359$, cEEG $n = 34$	HIE, CNS infections	13 %	–	NCSz not associated with outcome

Table 1 continued

Population	Reference	Study	*N*	Intervention	Risk factors	Seizures	Ischemia	Prognostication
SAH and ICH	Bosco [43]	P	68	cEEG	–	–	–	Increase in delta power was associated with a 24 % increase in mortality
Post-CA coma								
NT patients	Bassetti [66]	P	60	EEG	–	–	–	"Malignant" EEG patterns (non-reactive background, burst-suppression, flat EEG) 100 % predictive of poor outcome
	Chen [70]	P	34	EEG	–	–	–	"Malignant" EEG patterns were found in 20/22 non-survivors—unreactive EEG was found in 23/26 non-survivors
	Rothstein [76]	P	40	EEG	–	–	–	"Malignant" EEG patterns 80 % predictive of poor outcome
	Synek [80]	P	63	EEG	–	–	–	"Malignant" EEG patterns 98 % predictive of mortality
	Yamashita [82]	P	79	EEG	–	–	–	"Malignant" EEG patterns 100 % predictive of mortality
NT + TH patients	Fugate [89]	P	192	EEG	–	–	–	"Malignant" EEG patterns 100 % predictive of poor outcome
TH patients	Kawai [90]	R	26	cEEG	–	38.5 %	–	Continuous EEG pattern predictive of good outcome
	Rittenberger [61]	R	101	cEEG	–	12 %	–	NCSE 100 % predictive of poor outcome
	Legriel [60]	P	51	cEEG	–	10 %	–	SE 100 % predictive of mortality
	Rossetti [91]	P	111	EEG	–	–	–	"Malignant" EEG patterns 100 % predictive of poor outcome
	Rossetti [92]	P	34	cEEG	–	–	–	Non-reactive cEEG background during TH 100 % predictive of mortality; all survivors had cEEG background reactivity
	Rundgren [63]	P	34	cEEG	–	–	–	Pts with good outcome had a continuous EEG pattern; pts with "malignant" EEG patterns died
	Rundgren [93]	P	111	cEEG	–	–	–	Pts with good outcome had a continuous EEG pattern; burst-suppression pattern 100 % predictive of mortality
Status epilepticus								
GCSE	DeLorenzo [8]	P	164	cEEG	–	48 % (NCSz), 14 % (NCSE)		
>65 years patients with NCSE	Litt [109]	P	25	EEG	–	–	–	NCSE was associated with poor outcome
NCSz	Young [11]	R	49	cEEG	–	–	–	NCSE was associated with poor outcome
General ICU								
Mixed ICU GCS <9	Towne [29]	R	236*	cEEG	HIE, stroke	8 %	–	–
Mixed ICU GCS <9	Varelas [111]	R	129	EEG	Age, HIE	20 %	–	–
Mixed ICU GCS <9	Young [108]	P	55	cEEG	Primary brain injury has higher incidence of NCSz	9 %	–	–
Medical ICU	Oddo [105]	R	201	cEEG	–	10 %	–	Sepsis was an independent predictor of NCSz; NCSz were an independent predictor of poor outcome

Table 1 continued

Population	Reference	Study	*N*	Intervention	Risk factors	Seizures	Ischemia	Prognostication
Medical ICU	Firosh Khan [102]	P	286	EEG	–	10 % (NCSz), 4 % (NCSE)	–	NCSz/NCSE were not associated with outcome
Mixed ICU GCS <9	Scozzafava [101]	P	169	EEG	–	2 %	–	EEG abnormalities were not associated with poor outcome
Mixed ICU GCS <9	Young [103]	R	NR	EEG	–	–	–	Burst-suppression pattern and non-reactive EEG background predicted poor outcome
Sepsis	Young [104]	R	62	EEG	–	0 %	–	Burst-suppression pattern and non-reactive EEG background predicted poor outcome

ADR alpha/delta ratio, *CA* cardiac arrest, *cEEG* continuous EEG, *CNS* central nervous system, *DCI* delayed cerebral ischemia, *GCS* Glasgow coma score, *GCSE* generalized convulsive status epilepticus, *HIE* hypoxic-ischemic encephalopathy, *ICH* intracerebral hemorrhage, *NCSE* non-convulsive status epilepticus, *NCSz* non-convulsive seizures, *NPV* negative predictive value, *NR* not reported, *NT* normothermic, *P* prospective, *PPV* positive predictive value, *R* retrospective, *SAH* subarachnoid hemorrhage, *SE* status epilepticus, *TBI* traumatic brain injury, *TH* therapeutic hypothermia

[a] Adults and children

the management of SE in the ICU have recently been published [6].

Recommendations for patients with convulsive SE

1. We recommend urgent EEG in patients with SE that do not return to functional baseline within 60 min after administration of seizure medication (strong recommendation, low quality of evidence—grade 1C).

EEG in patients with refractory SE

SE resistant to initial therapy, also known as refractory SE (RSE), is almost exclusively nonconvulsive and requires initiation of intravenous AED [12–14]. CEEG is required to guide therapy for RSE, aiming to stop ongoing electrographic seizures. One study showed that although RSE initially responded to intravenous therapy, many patients subsequently developed NCSz, detectable only with cEEG [15]. There is controversy as to the minimum duration of monitoring [16–19] (see section "Technological issues"). Video-cEEG monitoring helps with the interpretation of complex electrographic abnormalities, but its efficacy over standard EEG has not been demonstrated yet [20].

Recommendations for patients with refractory SE

1. We recommend urgent (within 60 min) EEG in patients with RSE (strong recommendation, low quality of evidence—grade 1C).

EEG in patients with TBI

Seizure detection Patients suffering from TBI are at risk of NCSz [21, 22]. Risk factors for NCSz are depressed skull fracture, penetrating injury and large cortical contusion/hematomas [22]. Observational studies in patients with TBI monitored by EEG have shown a variable prevalence of NCSz. Vespa et al. ($n = 90$ patients, duration of cEEG 7 days) found a 22 % prevalence of seizures, of which 52 % were NCSz, despite AED prophylaxis [19]. Ronne-Engstrom and Winkler studied 70 patients (duration of cEEG 58 h, no AED prophylaxis) and found a 33 % prevalence of seizures (starting on average 74 h after TBI), the majority of which were NCSz [23]. The frequency of NCSz depends on the amount of sedatives used. Two recent studies, in which patients were given high sedative doses with intrinsic antiseizure activity, showed no [24] or a very low (3 %) [25] rate of NCSz. NCSz are associated with intracranial pressure elevations [26], increased cerebral metabolic distress [26] and long-term hippocampal atrophy [27].

Table 2 GRADE recommendations for the indications for EEG in the ICU

GRADE recommendations			Patient description		Objective
Direction	Strength	Level of evidence	Underlying etiology	Scenario	
Pro	Strong (1)	Low quality (C)	Generalized convulsive status epilepticus	No return to functional baseline after initial antiepileptic therapy	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (C)	Refractory status epilepticus	Concern for ongoing seizure activity	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (C)	Traumatic brain injury	Unexplained alteration in consciousness[a]	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (C)	Subarachnoid hemorrhage	Unexplained alteration in consciousness[a]	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (C)	Intracerebral hemorrhage	Unexplained alteration in consciousness[a]	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (C)	Cardiac arrest	Persistent coma	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (C)	Encephalitis	Unexplained alteration in consciousness[a]	Detect nonconvulsive seizures
Pro	Strong (1)	Low quality (B)	Comatose patients without primary brain injury	Unexplained alteration in consciousness[a]	Detect nonconvulsive seizures
Pro	Weak (2)	Low quality (C)	Severe traumatic brain injury	Concern for ongoing seizure activity in high-risk patients (large cortical hemorrhagic contusion/hematoma)	Detect nonconvulsive seizures
Pro	Weak (2)	Very low quality (D)	Acute ischemic stroke	Unexplained alteration in consciousness[a]	Detect nonconvulsive seizures
Pro	Weak (2)	Low quality (C)	Subarachnoid hemorrhage	Patients in whom clinical examination is unreliable	Detect ischemia
Pro	Weak (2)	Low quality (C)	Cardiac arrest	Persistent coma	Prognostication
Pro	Weak (2)	Low quality (C)	All comatose ICU patients	Unexplained alteration in consciousness[a]	Prognostication
Pro	Weak (2)	Very low quality (D)	Encephalitis	Unexplained alteration in consciousness[a]	Prognostication

[a] Unexplained alteration in consciousness: reduced consciousness state that is not attributable to metabolic disorders (sodium, calcium, glucose, ammonium, urea), organ dysfunction (hypotension, hypoxemia, sepsis, hyperthermia) or structural brain lesions on imaging (cerebral CT scan) tests

Despite variable results and lack of multicenter studies, there is a strong rationale for EEG monitoring after TBI. This is reinforced by the fact that primary AED prophylaxis is frequently unreliable in preventing or suppressing NCSz [28].

Ischemia detection No study has shown a role for EEG in detecting ischemia after TBI.

Prognostication Towne et al. [29] and Vespa et al. [19] were unable to demonstrate a difference in mortality between TBI patients with or without EEG seizures. EEG reactivity to auditory or nociceptive stimuli predicted good outcome after TBI, whereas absent EEG reactivity resulted in a poor outcome [30, 31] with a higher predictive value than GCS and somatosensory evoked potentials. In another study, EEG performed daily during the first week after admission reliably predicted outcome in 40/50 patients; however, prognosis could not be assessed in patients with alpha pattern coma or in those receiving barbiturate therapy [32]. Reduced percentage of alpha variability also predicted outcome in TBI patients with GCS $\leq$8 (positive predictive value 86 %) [33].

Recommendations for patients with TBI

1. We recommend EEG in all TBI patients with unexplained and persistent altered consciousness (strong recommendation, low quality of evidence—grade 1C).
2. We suggest EEG to rule out NCSz in patients with TBI and GCS $\leq$8, particularly in those with large cortical contusion/hematoma, depressed skull fracture or penetrating injury (weak recommendation, low quality of evidence—grade 2C).

EEG in patients with SAH

Seizure detection Acute seizures have been reported in between 3 % and 26 % of patients with comatose SAH [34–36]. Of those undergoing cEEG in the ICU, 3–19 %

have NCSz and 13 % have NCSE, which cannot be diagnosed without EEG. Risk factors for seizures include older age, poor clinical grade, large intraparenchymal hemorrhage, large amount of cisternal blood, DCI, and anterior circulation aneurysm. Seizures may be less likely in patients that have undergone coil embolization of the aneurysm [34].

Ischemia detection In SAH patients, changes in EEG trends on cEEG (performed on days 2–10) correlate with DCI [37–41]. A number of quantitative EEG (qEEG) parameters may be useful, including changes in total power, alpha/delta ratio (ADR), composite alpha index, and relative alpha variability. There is controversy over which parameter is best, but all fundamentally relate to fast to slow frequencies. QEEG can detect EEG changes associated with DCI 24–48 h prior to other diagnostic tools [39, 41]. Reported sensitivity is variable but can be as high as 90 % [37, 38], with 75 % specificity [37], and 100 % negative predictive value and 76 % positive predictive value [41].

Prognostication Epileptiform discharges or NCSE and absent EEG background reactivity was associated with poor prognosis after SAH [42]. Despite this association, there are no unequivocal human data indicating that NCSz are causally linked to poor functional outcome or that treatment improves outcome [34, 35, 42]. Progressive deterioration on the basis of EEG (increased delta pattern) was associated with an increased risk of dying by almost 24 % compared to patients whose condition did not worsen according to EEG [43].

Recommendations for patients with SAH

1. We recommend EEG to rule out NCSz in all SAH patients with unexplained and persistent altered consciousness (strong recommendation, low quality of evidence—grade 1C).
2. We suggest EEG to detect DCI in comatose SAH patients, in whom neurological examination is unreliable (weak recommendation, low quality of evidence—grade 2C).

EEG in patients with ICH

Seizure detection Seizures are seen in 3–17 % of ICH patients, occurring at 1 day (50–70 %) up to 3 days from ICH. Most seizures diagnosed in the ICU are non-convulsive (NCSz 53–76 %, NCSE 39 %) and can only be diagnosed by EEG [44–46]. Risk factors include cortical bleeding and arteriovenous malformations [44, 46].

Ischemia detection No study has provided data on ischemia detection in ICH patients.

Prognostication Seizures are associated with an increase in ICH volume and worsening midline shift [44, 46]. NCSZ worsen neurological status, but an independent association with outcome has not been demonstrated [44, 46].

Recommendations for patients with ICH

1. We recommend EEG to rule out NCSz in all ICH patients with unexplained and persistent altered consciousness (strong recommendation, low quality of evidence—grade 1C).

EEG in patients with AIS

Seizure detection One single-center study in which cEEG was performed in 177 patients with AIS showed a 7 % incidence of seizures (more than 70 % NCSz) in the acute (<24 h) phase [45]. Seizures are less frequent than in ICH, SAH or TBI patients.

Ischemia detection A decrease in cerebral perfusion pressure (CPP) may be associated with a concomitant reduction in faster EEG activity on qEEG [47], while rapid improvements in background EEG activity have been observed upon CPP/CBF increase following mannitol therapy [48] or hemodilution [49].

Prognostication Following hemicraniectomy for space-occupying middle cerebral artery infarction, the presence of faster EEG activity was associated with good recovery in patients monitored with cEEG [50]. Three studies have demonstrated that the disappearance or further slowing of delta activity in the acute phase (within 24 h) of AIS predicted a malignant course (cerebral edema) [51–53].

Preliminary studies showed a correlation between the neurological score in the acute stage of AIS and the degree of EEG abnormality [54], although this correlation was shown to be low by others [55]. CEEG improves outcome prognostication in AIS [56–59]: in particularly, the ADR and the so-called EEG brain symmetry index are significantly correlated with outcome at 6 months [56–59].

Recommendations for patients with AIS

1. We suggest EEG to rule out NCSz in all AIS patients with unexplained and/or persistently altered consciousness (weak recommendation, very low quality of evidence—grade 2D).
2. We do not recommend EEG to detect cerebral ischemia and target CPP in AIS patients (weak recommendation against, very low quality of evidence—grade 2D).

3. We do not recommend EEG to detect herniation in AIS patients (weak recommendation against, very low quality of evidence—grade 2D).

EEG in patients with coma after CA

Seizure detection Seizures occur in 10–30 % of patients with coma after CA [60–63]. EEG is required to detect seizures as most seizures after CA are nonconvulsive and to differentiate myoclonic SE from peripheral or subcortical myoclonus. When therapeutic hypothermia (TH) is applied, seizures can occur during TH and after rewarming [60, 61, 63]. "Early" seizures, occurring during TH under sedation, are an ominous sign [60–63]. "Late" seizures, occurring after TH and off sedation, carry a poor prognosis but may respond to therapy in certain cases [64]: EEG is indicated to titrate therapy [61, 64].

Ischemia detection No study has provided data on ischemia detection in comatose CA patients or used EEG to target blood pressure management.

Prognostication Previous to TH, a number of studies showed that adding EEG—performed at 72 h from CA—to standard neurological examination improved outcome prognostication after CA [65–84]. EEG findings associated with a poor prognosis included spontaneous burst suppression or generalized periodic discharges. Synek analyzed EEG background activity (continuous vs. discontinuous pattern) and EEG background reactivity to auditory and painful stimulation, subsequently dichotomized as "reactive" vs. "non-reactive" [30, 80, 85]: the presence of a continuous and reactive EEG background (i.e. a change in EEG frequency and amplitude following stimulation) was associated with good prognosis. At this time TH is considered the standard of care after CA. Hypothermia and sedation used during cooling alter motor response and decrease the prognostic accuracy of neurological examination. Several studies performed in patients treated with TH demonstrated that EEG improves prognostic prediction of coma after CA [63, 86–95]. The presence of discontinuous and burst-suppression patterns, and of nonreactive EEG background, were strongly correlated (false-positive rates for poor prognosis <10 %) with a poor prognosis, whilst a continuous reactive background was associated with good recovery. Importantly, in some studies, coma prognostication could be achieved during TH [63, 92, 93].

Recommendations for comatose patients after CA

1. We recommend EEG during TH and within 24 h after rewarming to rule out NCSz in all comatose patients after CA (strong recommendation, low quality of evidence—grade 1C).
2. We suggest EEG to assist with prognostication of coma after CA, particularly in patients treated with TH (weak recommendation, low quality of evidence—grade 2C).

EEG in patients with infectious and non-infectious encephalitis

Seizure detection Central nervous system (CNS) infections, mainly acute meningitis/encephalitis, are a risk factor for seizures, ranging from 6–12 % in some studies [96], and seizures are associated with higher mortality rates [97]. In a small retrospective study, Carrera et al., found seizures in one-third of 42 patients with primary CNS infections, and the majority of these were NCSz [98]. In the large cohort of patients undergoing cEEG monitoring reported by the Columbia University group, CNS infections and metabolic encephalopathy accounted for 13 % of all patients and there was 23 % and 12 % frequency of NCSE and NCSz, respectively. Comatose patients needed more than 24 h of cEEG monitoring to detect NCSz [17]. In another large cohort of neurocritical care patients ($n = 393$) with admission GCS $\leq$12 and at least one EEG (cEEG, $n = 34$), the prevalence of NCSz was 13 % and was highest among those with CNS infection, together with anoxic encephalopathy [99]. NCSz are very frequent in noninfectious encephalitis (up to 78 % of cases) and are mostly nonconvulsive [100].

Ischemia detection No study has provided data on ischemia detection in patients with encephalitis.

Prognostication No study has analyzed the prognostic accuracy of EEG in patients with encephalitis but particular patterns such as "delta brush" may be associated with a more prolonged illness [100].

Recommendations for patients with infectious and non-infectious encephalitis

1. We recommend EEG in patients with encephalitis that are comatose or have unexplained neurological deficits to rule out NCSz (strong recommendation, low quality of evidence—grade 1C).
2. We suggest EEG in patients with encephalitis to assist with prognosis (weak recommendation, very low quality of evidence—grade 2D).

EEG in comatose ICU patients without acute primary brain injury

Seizure detection In a retrospective cohort of 238 general ICU comatose patients in whom EEG was performed, Towne et al. found a prevalence of NCSz of 8 % [29].

Postanoxic encephalopathy (42 %) was the most common etiology, followed by AIS (22 %), CNS infection, TBI, metabolic encephalopathy, alcohol or AED withdrawal (5 %), and brain tumor (all 5 %). Using standard 20-min EEG, Scozzafava found NCSz only in 2 of 169 patients with GCS <8 [101]. In 286 patients, of whom 22 % had encephalitis and 24 % metabolic encephalopathy, Firosh Khan et al. found that 4 % had NCSE and 10 % NCSz [102]. Patients with primary brain injury had a higher incidence of NCSz than those with metabolic encephalopathy (32 % vs. 4 %) [103]. Only two studies specifically focused on patients admitted to the ICU without a primary acute brain condition, in whom cEEG was performed because of altered consciousness. Young et al. found no NCSz among 62 patients with sepsis [104]. In a retrospective cohort of 201 medical ICU patients monitored with cEEG, Oddo et al. found a 10 % frequency of seizures, of which 69 % were purely NCSz [105]. Sepsis was the most common etiology and was the only independent risk factor for seizures. These findings confirm those of previous studies showing that septic encephalopathy and metabolic dysfunction (mainly renal and hepatic failure) are risk factors for NCSz [11, 106, 107].

Ischemia detection No study has provided data on ischemia detection in medical/surgical ICU populations.

Prognostication Patients with NCSz had the highest mortality rate in a large neuro-ICU population, although this finding was not significant after adjustment for confounding factors [99]. The same results were found in another study [101]. Firosh Khan et al. [102] found 42 % and 21 % of patients with NCSE and NCSz, respectively, had a poor outcome, but did not analyze the prognostic value of these findings. Young et al. [108] found that EEG suppression and lack of EEG reactivity were associated with a worse outcome in ICU patients; however, these data were only applicable to comatose CA patients. In a study of septic patients, the same group found that EEG abnormalities, but not NCSz, were associated with mortality (0 % in patients with normal EEG, 19 % in patients with theta rhythm, 36 % in patients with delta rhythm, 50 % in patients with triphasic waves and 67 % in patients with suppression) [104]. NCSz was associated with a poor outcome in septic patients [105] and in critically ill elderly (>65 years of age) patients [109].

Recommendations for comatose ICU patients without acute primary brain injury

1. We suggest EEG in comatose ICU patients without an acute primary brain condition and with unexplained impairment of mental status or unexplained neurological deficits to rule out NCSz, particularly in those with severe sepsis or renal/hepatic failure (weak recommendation, low quality of evidence—grade 2C).

Technological issues

Duration of monitoring: continuous vs. intermittent EEG monitoring

Seizure detection Continuous EEG allows the detection of NCSz [11, 18, 103, 110] but there is controversy as to the minimum duration of cEEG. In a single-center retrospective study, about 50 % of NCSz were detected within the first 60 min of EEG, but in comatose neuro-ICU patients at least 24 h and up to 48 h of monitoring may be required [17]. Continuous EEG is essential to titrate AED in RSE and to identify recurrent NCSz [15]. Intermittent (<30 min duration) EEG may be insufficiently sensitive to detect NCSz [101], but no studies have compared continuous to intermittent EEG. Standard EEG can provide useful information in selected clinical situations, such as epilepsy-related situations, CA and brain death examination [102, 111]. In a recent study, independent predictors of epileptiform activity included a history of convulsive seizure(s), increasing age, deeper coma, and female gender [99]. In this study, the "number needed to monitor" was seven, i.e. at least seven neuro-ICU patients should undergo intermittent EEG to diagnose one with seizures.

Ischemia detection Continuous EEG using qEEG analysis has been used to detect cerebral ischemia in comatose SAH patients and in subjects with AIS. In SAH patients at risk of DCI, monitoring is performed for several days, during maximum DCI risk [37, 39, 41], and on average for 7 days [39]. QEEG is similarly performed for several days after AIS, one study reporting an average of 83 h of monitoring [47].

Prognostication After CA and TH, EEG—intermittent or continuous—improves coma prognostication [63, 86–95, 112]. Whether cEEG has higher prognostic accuracy than intermittent EEG has not been evaluated. Early prognostication of AIS [56–59], ICH [44, 46] and SAH [42] has exclusively been assessed with cEEG.

Recommendations for continuous EEG over intermittent EEG monitoring

1. We recommend cEEG for seizure detection in patients with RSE (strong recommendation, low quality of evidence—grade 1C).
2. We suggest cEEG for seizure detection in patients with SE that do not return to functional baseline within 60 min after administration of seizure medication

(weak recommendation, low quality of evidence—grade 2C).

3. We suggest cEEG for seizure detection in comatose ICU patients (TBI, SAH, ICH, coma after CA, encephalitis, and septic and metabolic encephalopathy) with unexplained and persistent altered consciousness (weak recommendation, low quality of evidence—grade 2C).
4. We suggest cEEG for ischemia detection in comatose SAH patients in whom neurological examination is unreliable (weak recommendation, low quality of evidence—grade 2C).
5. We suggest cEEG to assist with prognostication of coma after CA (weak recommendation, low quality of evidence—grade 2C).

Montage: standard vs. simplified

Seizure detection The placement of 21 electrodes is the standard method for EEG monitoring. Compared to standard EEG, the sensitivities of simplified EEG for seizure detection were 93 % in one study using seven electrodes [113], 68 % in another study using four electrodes [114], and 40 % with single-channel EEG [115].

Ischemia detection All studies that examined the value of EEG for ischemia detection used a standard montage [37, 39, 41, 47].

Prognostication After CA and TH, EEG—intermittent or continuous—improves coma prognostication. The majority of the studies used a standard EEG montage [86, 88–92, 94, 95, 112], but others showed similar predictive values using simplified montages [63, 87, 93]. Prognostication of AIS [56–59], ICH [44, 46] and SAH [42] has exclusively been assessed with a standard montage.

Recommendations for standard vs. simplified EEG montage in ICU patients

1. We recommend a standard EEG montage (21 electrodes) for the detection of NCSz in ICU patients (weak recommendation, poor quality of evidence—grade 2C).

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Conflicts of interest None.

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