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Predicting outcome of status epilepticus

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ABSTRACT

Background: Status epilepticus (SE) is a frequent neurological emergency complicated by high mortality and often poor functional outcome in survivors. The aim of this study was to review available clinical scores to predict outcome.
Methods: Literature review. PubMed Search terms were "score", "outcome", and "status epilepticus" (April 9th 2015). Publications with abstracts available in English, no other language restrictions, or any restrictions concerning investigated patients were included.
Results: Two scores were identified: "Status Epilepticus Severity Score — STESS" and "Epidemiology based Mortality score in SE — EMSE". A comprehensive comparison of test parameters concerning performance, options, and limitations was performed. Epidemiology based Mortality score in SE allows detailed individualization of risk factors and is significantly superior to STESS in a retrospective explorative study. In particular, EMSE is very good at detection of good and bad outcome, whereas STESS detecting bad outcome is limited by a ceiling effect and uncertainty of correct cutoff value. Epidemiology based Mortality score in SE can be adapted to different regions in the world and to advances in medicine, as new data emerge. In addition, we designed a reporting standard for status epilepticus to enhance acquisition and communication of outcome relevant data. A data acquisition sheet used from patient admission in emergency room, from the EEG lab to intensive care unit, is provided for optimized data collection.
Conclusion: Status Epilepticus Severity Score is easy to perform and predicts bad outcome, but has a low predictive value for good outcomes. Epidemiology based Mortality score in SE is superior to STESS in predicting good or bad outcome but needs marginally more time to perform. Epidemiology based Mortality score in SE may prove very useful for risk stratification in interventional studies and is recommended for individual outcome prediction. Prospective validation in different cohorts is needed for EMSE, whereas STESS needs further validation in cohorts with a wider range of etiologies.

This article is part of a Special Issue entitled "Status Epilepticus".

1. Introduction

Status epilepticus (SE) is a neurological emergency. Mortality rates for convulsive SE vary from 7.6% to 39% in population-based studies [1]. Many risk factors for death were reported as statistically significant group effects (for comprehensive review see [2]). However, group effects do not necessarily predict individual outcomes, which are needed in clinical practice. Thus, data on group effects must be integrated into clinically useful scores to allow individual and fast risk assessment for optimal patient management. In this regard, detection of bad outcome is mandatory to avoid underdetection and undertreatment of SE. Adequate detection of good outcome is necessary to minimize risks of potentially harmful overtreatment and to assure economic use of resources, e.g., intensive care unit admission. We performed a literature search to identify all currently available scoring systems for outcome prediction in status epilepticus.

2. Methods

We searched the PubMed database on April 9th 2015 for "score", "outcome", and "status epilepticus" and their combinations for papers in any language with abstracts available in English with no restrictions concerning investigated patients.

Abbreviations: SE, status epilepticus; SEO-C, status epilepticus outcome code; SLP, survival limiting process; OS, outcome score.

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Table 1
Comparison of STESS and EMSE.

STESS [3–5]	EMSE [6]
Number of parameters and total number of items to choose	
4 (etiology, age, semiology, level of consciousness pretreatment), 9 items	4 (etiology, age, comorbidity, EEG), 45 items
Rationale for scoring points	
A priori assumptions	Epidemiological "real world" data: mortality rates of big studies (identified in systematic reviews) were taken as scoring points. Different combinations of age, etiology, etc., were tested for best performance.
Initial study	
Retrospective evaluation (N = 107), then prospectively tested (N = 34) [3]	Exploratory, hypothesis generating study, fitting test/score to sample, N = 92
Prospective validation	
Yes [4]: N = 154,	Pending
First external validation	
Yes [5]: N = 171: suggested change of threshold, s. below	Pending
Cutoff indicating bad outcome (*nonsurvival*)	
3 points or higher [3,4] OR 4 points or higher [5]	64 points or higher
Young patients with preexisting epilepsy (*specialized epilepsy center with young patients with preexisting epilepsy*)	
No ceiling effect	No ceiling effect
Elderly patients without preexisting epilepsy (*general neurology*, *emergency room*)	
Ceiling effect, all pat. score high risk	No ceiling effect
Flexibility for global use, *e.g.*, *developing countries*	
Minimal	Yes
Adaptability as new epidemiological data emerge	
Minimal	Yes
Number of different items per parameter to individualize outcome prediction, *remarks*	
Age	
2 (below 65: 0 pt., 65 or higher: 2 pt.)	7 (for each decade: 21–30 a: 1 pt., 31–40 a: 2 pt., 41–50 a: 3 pt., 51–60 a: 5 pt., 61–70 a: 7 pt., 71–80 a: 8 pt., >80 a: 10 pt.) [7]
Etiology	
2 (previous seizures: 0 pt., no previous seizures: 1 pt.)	15 (etiologies [8,9]: CNS-anomalies: 2 pt.; drug reduction/withdrawal, poor compliance: 2 pt.; multiple sclerosis: 2 pt.; remote cerebrovasc. dis./brain injury: 7 pt.; hydrocephalus 8 pt.; alcohol abuse 10 pt.; drug overdose 11 pt.; head trauma 12 pt.; cryptogenic 12 pt.; brain tumor 16 pt.; sodium imbalance 17 pt.; metabolic disorders 22 pt.; acute cerebrovascular dis.: 26 pt.; acute CNS infection: 33 pt.; anoxia: 65 pt.)
Level of consciousness before treatment	
2 (alert, somnolent/confused: 0 pt.; stuporous or comatose 1 pt.)	4 (following points did not improve performance: awake 0 pt., somnolence: 5 pt., stupor: 14 pt., coma: 23 pt.) [10]
Semiology	
3 (simple partial, complex partial, absence, myoclonic-complicating idiopathic generalized epilepsy: 0 pt.; generalized convulsive 1 pt.; NCSE in coma: 2 pt.)	Not scored, but investigated regarding predictability: SE with only convulsions: correctly classified 95.5%, Seizures including/evolving to NCSE: correctly classified 81.4%
Duration	
Not included	2 (following points did not improve performance: <1 h: 3 pt., >1 h: 33 pt.) [11]
EEG	
Not included	5 (burst suppression (spontaneous): 60 pt.; ASIDs 40 pt.; LPDs 40 pt.; GPDs 40 pt.; no LPDs, GPDs or ASIDs: 0 pt.) [12,13]
Comorbidity	
Not included	18 (myocardial infarction, congestive heart failure, peripheral vascular dis., cerebrovascular dis., dementia, chronic pulmonary dis., connective tissue dis., ulcer disease, mild liver dis., diabetes: 10 pt.; hemiplegia, moderate or severe renal dis., diabetes with end organ damage, any tumor including leukemia/lymphoma: 20 pt.; moderate or severe liver dis.: 30 pt.; metastatic solid tumor, AIDS: 60 pt.) [14]

LPDs: lateralized periodic discharges, GPDs: generalized per. discharges, ASIDs: after SE discharges, dis.: disease, NCSE: nonconvulsive SE.

3. Results

We identified two scores, i.e., "Status Epilepticus Severity Score — STESS" [3–5] and "Epidemiology based Mortality score in SE — EMSE" [6] (Table 1).

There is only one study in which STESS with cutoff levels 3 and 4 (STESS-3: 3 or more points indicate bad outcome, i.e., death; STESS-4) and EMSE (cutoff 64) were applied to one population. In this retrospective, explorative study, EMSE was significantly superior to STESS: negative predictive value (NPV) of EMSE 100%, STESS-3 82.8% ($p = 0.0022$),

and STESS-4 81.8% ($p = 0.00034$); positive predictive value (PPV) of EMSE 68.8%, STESS-3 27.0% ($p = 0.000012$), and STESS-4 32.4% ($p < 0.00001$); correctly classified (CC) of EMSE 89.1%, STESS-3 44.6% ($p = 0.000029$), and STESS-4 62.0% ($p < 0.00001$); level of significance corrected for multiple testing: $p \leq 0.0044$ (Supplementary Fig. 1) [6].

4. Discussion

Status Epilepticus Severity Score and Epidemiology based Mortality score in SE are the only two available scores for outcome prediction in SE. Both are easy to apply. Status Epilepticus Severity Score is good at predicting bad outcome but has a ceiling effect especially in patients older than 65 years without preexisting epilepsy. In contrast, EMSE is good at detection of both bad and good outcomes and therefore, is highly qualified for individual risk assessment as well as risk stratification in interventional studies. However, EMSE needs prospective evaluation, because it was developed in a retrospective explorative study, i.e., where test results were known. This implies the danger of overfitting the test to the sample from a statistical point of view. However, these data can be used for fast testing of different hypotheses.

4.1. Concept of "survival limiting process"

Survival is a hard endpoint of outcome. While interpreting results, it is important to ask whether one particular factor, such as duration of SE, or the combination of many factors (i.e., EMSE-score, [6]) is or is not the survival limiting process (SLP). In other words, it depends on the investigated study population whether a particular parameter is responsible for death, or just contributes to nonsurvival among other factors, or does not play any life-limiting role at all [2].

4.2. Duration and its inherent problems

The correct measurement of duration is hampered by the fact that the beginning of SE is frequently not witnessed. Furthermore, the end of SE is also not clear-cut, as diagnosing the end of SE can either be done by EEG documentation (continuous or intermittent EEG) or by progressive clinical improvement. With EEG, it is to be defined whether pathognomonic graphoelements need to be totally eradicated (for how long?) or just "significantly reduced", which is itself not yet specified. Alternatively, the progressive improvement in consciousness indicates the end of SE. However, no uniform strategy of testing has been accepted. We use calling the patient with his first name and asking him to open his eyes, look at the investigator, count until three, and raise his arms. If the patient is not reactive, the same procedure is repeated while applying strong tactile stimuli on each side of the body.

We addressed these issues and developed a data acquisition sheet, which assures rapid documentation in busy emergency rooms and availability of data at any time during patient management. It requires physicians to enter information relevant for calculation of duration as well as many other important parameters such as level of consciousness, history of seizure semiology, EEG findings, and applied medication (Supplementary Fig. 2).

An alternative to duration is time to treatment. There is a major difference between acute treatment of ischemic stroke and SE. In stroke, the medication (usually recombinant tissue plasminogen activator, rt-PA) is just adapted to body weight; there is no repetition in case of failure. Hence, time to treatment seems adequate, as there are no modifications after initiation. However, in SE, many different medications are applied in an escalating procedure, which calls for communication as to when all of these measures were finally successful, instead of when they were started. According to these inherent problems, there are just a few studies reporting the duration instead of time to treatment. The significant time points for bad outcome show great variation depending on the investigated population ranging from 0.5 vs 1 h [15], < 1 h vs > 1 h [11], <2 vs >24 h [16], 2.4 vs 11.2 h [17], < 10 h vs > 10 h [18], 24 vs 48 h [19], to 88.9 vs 120 h [20]. Interestingly, in a recent retrospective study [20] on 111 patients, duration was markedly prolonged in nonsurvivors (without reaching statistical significance) with the etiology of "uncontrolled epilepsy". In this otherwise benign etiology, it seemed that duration was the only SLP. We speculated whether some factors (here, duration) "only matter, when nothing else matters?". Again, for pathophysiological understanding and tailored therapy, it is mandatory to identify the one or many SLP(s).

4.3. Prediction versus predictability

Semiology was not included as score parameter in EMSE, but results were investigated concerning semiology. In those patients with "only convulsive" semiology, i.e., focal motor (FM-SE) or generalized tonic–clonic (GTC-SE) — both without evolution into nonconvulsive SE (NCSE), outcome was predictable by EMSE with cases correctly classified in about 95.6%, as opposed to 81.4% in those with NCSE as part of semiology, i.e., NCSE as only semiology and FM-SE and GTC-SE — both evolving into NCSE. It seems that the brain is not the SLP as long as major motor phenomena are present (only convulsive semiology), as mortality is predictable by other factors in EMSE. This is congruent with pathophysiological considerations, where evolution of convulsive into nonconvulsive semiology is not only associated with a worse prognosis, but also reflects the individual sign of cerebral decompensation with electroclinical dissociation [21,22].

4.4. Level of consciousness (LOC)

Significant impact of LOC on survival was first reported by Rossetti et al. [10]. Their data were implemented in EMSE but resulted in no improvement of performance [6]. Another study by Rossetti et al. did not reach significance [4]. In NCSE, significant differences were reported between "mild and severe" disturbance of "mental status" [23]. Sutter et al. found significant results for stupor/coma versus awake/somnolence, which disappeared after exclusion of patients with hypoxia [20]. In the same study, NCSE-coma was significantly associated with bad outcome, even in the latter population.

4.5. Comorbidities

Data provided by EMSE suggest an important role of comorbidities, as this is among the successful combination of 4 parameters. This was in contrast to recent investigations with elaborated models [24]. Importantly, another study reported a highly significant difference of "Logistic Organ Dysfunction Score" (LODS) between survivors and nonsurvivors [25]. Logistic Organ Dysfunction Score summarizes dysfunction of major organ systems [26]. Therefore, LODS can be interpreted as the functional consequences of comorbidities, which were scored by Charlson comorbidity index (CCI) and, therefore, also by EMSE. At the 5th London Innsbruck Colloquium in April 2015 data were presented, which showed a significant role of CCI [27]. Some comorbidity may have changed its treatability and associated prognosis, such as HIV infections, with significant improvements of survival over the past years [28]. This might also apply to specific malignancies, where several diseases have a high chance of five-year progression-free illness. These changes over time can be implemented easily in EMSE, but should be done in global interdisciplinary consensus.

4.6. Frequency of periodic discharges

We were able to increase performance of EMSE by including only lateralized periodic discharges (LPDs) occurring at least at seven LPDs per ten-second epoch (counted in worst epoch) and generalized periodic discharges (GPDs) occurring at least at 9 GPDs per 10 s [6]. Whether the rate of periodic discharge is associated with survival

Table 2
Status epilepticus outcome code (SEO-C) allows comprehensive communication of outcome relevant data.

SEMIOLOGY(S)		
GTC …	generalized tonic-clonic	Example: **S (FM → GTC→ NC–STE–ED/stp)** …
FM …	focal motor	SE starting with focal motor activity, transition to
NC …	nonconvulsive	generalized tonic-clonic convulsions, transition to non
NC–ED>2.5…	NC with epileptiform discharges more frequent than 2.5/s	convulsive SE, identified by spatiotemporal evolution of epileptiform discharges in EEG while patient was stuporous
NC–SCP…	NC with subtle clinical phenomena	
NC–STE–ED…	NC with spatiotemporal evolution of epileptiform discharges in EEG	Example extended version: **S (FM: 30 min → GTC: 60 min → NC–STE–ED/stp: 2 h)** … Same as above, but with durations of different semiologies
NC–STE–RDT…	NC with spatiotemporal evolution of Rhythmical Delta–Theta activity in EEG	
NC–ECI…	NC with EEG and clinical improvement to iv–AEDs	
NC–FED…	NC with fluctuation of epileptiform discharges	
NC–FRDT…	NC with fluctuation of Rhythmical Delta–Theta	
NC–OEI…	NC with only EEG improvement to iv AEDs	
"/" level of consciousness:	w… a**w**ake, som…**som**nolent, stp … **stupo**rous, com…**com**atose	
ETIOLOGY (E)		
RCV …	remote cerebrovascular	Example: **E (RCV) …** aetiology is remote cerebrovascular infarction
ACV …	acute cerebrovascular	
RTBI …	remote traumatic brain injury	
ATBI …	acute TBI	Example extended version: **E (AAW, HON) …** Patient suffered from acute alcohol withdrawal and hyponatremia
EDW…	epilepsy, drug withdrawal	
AAI …	acute alcohol intoxication	
AAW …	acute alcohol withdrawal	
HON …	hyponatremia; … other codes to be established	
AGE (A)		
Age	in years (yr), for pediatricians: months (mth) or days (d)	Example: **A (45 yr)** … Patient is 45 years old
THERAPY (T)		
B …	benzodiazepines	Example: **T (BA_2)** … Treatment with one benzo and 2 AEDs Example extended version: **T (BA2: LZM 6 (1h), LEV 2000 (1.1h), PHT 1000(1.2 h))…** Details of medication provided together with starting time
A …	antiepileptic drug	
N …	narcotic	
S …	measure against **s**uperrefractory SE	
EEG (E)		
LPD …	lateralised periodic discharges	Example: **E (STE–ED → DSL)** … EEG with spatiotemporal evolution of epileptiform discharges, transforming into diffuse slowing Example extended version: **E (STE: R frontal, at 2.5 h)** … STE was right frontal, EEG was done 2.5 hours from onset
GPD …	generalised periodic discharges	
ASID …	after status ictal discharges	
SIRPID …	stimulus induced rhythmic, periodic or ictal discharges	
STE …	typical spatiotemporal evolution	
DSL … …	diffuse slowing … other codes to be established	
DURATION (D)		
Start:	witnessed … stw; not witnessed… snw), **"/"**	Example: **D (stw/1 h/PCI) …** SE with witnessed beginning, lasting for one hour, end was diagnosed by progressive clinical improvement
Duration:	1h (h), day (d), **"/"**	
End diagnosed by:	EEG, progressive clinical improvement (PCI)	
OUTCOME (O)		
REST …	full restitution to premorbid level	Example: **O (PEND)** … Patient is transferred to tertiary care centre, outcome pending Example extended version: **O (PDEF: MCS)** … Patient survived in minimally conscious state
PDEF …	survived with persisting deficits	
NOS …	nonsurvivor	
PEND …	pending	
COMORBIDITIES (C)		
COPD …	chronic obstructive pulmonary disease	Example: **C (CHD, CHF)** … Patient suffers from coronary heart disease and congestive heart failure
CRF 2…	chronic renal failure stage 2	
DM–II…	diabetes mellitus type II	
CHF …	congestive heart failure	
CHD …	coronary heart disease … other codes to be established	
Example: S (FM → GTC→ NC–STE–ED/stp),**E**(RTBI), **A**(67 yr), **T**(BA_2),**E**(STE–ED →DSL), **D**(stw/2h/PCI), **O**(REST),**C**(COPD),		
SE starting with focal motor activity, transition to generalized tonic-clonic convulsions, transition to non–convulsive SE, identified by spatiotemporal evolution of ED in EEG while patient was stuporous. Etiology was remote traumatic brain injury. Age was 67 yrs. Patient was treated with one benzo and two AEDs. EEG showed spatiotemporal evolution of epileptiform dischargeswith transition to diffuse slowing. Start of SE was witnessed, SE lasted 2 hours, end of SE was diagnosed by progressive clinical improvement. Outcome was restitution to premorbid level. Comorbidity was chronic obstructive pulmonary disease.		

needs prospective evaluation. The maximum of EMSE performance was achieved, when after SE ictal discharges (ASIDs) were not included, but numbers were too small to draw firm conclusions [6].

4.7. Adequate reporting of outcome scores in studies

Group effects are adequately reported as statistically significant associations. Outcome scores (OS) are derived from these group effects and are, therefore, intrinsically associated with the underlying condition (Supplementary Fig. 3). Hence, it is not enough to demonstrate the statistically significant association of OS with clinical condition. Rather, the benchmark for OS is the rate of correctly classified cases, negative and positive predictive values in the investigated population, and further sensitivity and specificity.

4.8. Status epilepticus outcome code (SEO-C)

We searched for a measure to enhance (1) usage of scores for individual risk assessment for individually tailored therapy, (2) entrance of patient-specific data into epidemiological studies and registries and further into public health projects, and (3) support communication in case of patient transfer or discharge, or in case of regular staff rotation between day and night shifts (Supplementary Fig. 3). We developed SEO-C to address these issues with a basic and an extended version (Table 2) and, hereby, gave answer to the call for an international reporting standard for SE first raised by R. Dieckmann in 2006 [28]. Feasibility of SEO-C is not yet published.

5. Conclusion

Epidemiology based Mortality score in SE needs prospective validation. So far, EMSE qualifies for risk stratification in interventional studies and is recommended for individual outcome prediction. A specific code for collecting SE related data in clinical practice (SEO-C) is proposed.

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Conflict of interests

Eugen Trinka has acted as a paid consultant for Eisai, Ever Neuropharma, Biogen Idec, Medtronics, Bial, and UCB and has received speakers' honoraria from Bial, Eisai, GL Pharma, GlaxoSmithKline, Boehringer, Viropharma, Actavis, and UCB Pharma in the past 3 years. Eugen Trinka has received research funding from UCB Pharma, Biogen Idec, Red Bull, Merck, the EU, FWF Österreichischer Fond zur Wissenschaftsförderung, and Bundesministerium für Wissenschaft und Forschung. Eugen Trinka is also one of the investigators planning ESETT (Established Status Epilepticus Treatment Trial).

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