ORIGINAL ARTICLE

Frequency and Timing of Nonconvulsive Status Epilepticus in Comatose Post-Cardiac Arrest Subjects Treated with Hypothermia

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Abstract

Background Therapeutic hypothermia (TH) improves outcomes in comatose patients resuscitated from cardiac arrest. However, nonconvulsive status epilepticus (NCSE) may cause persistent coma. The frequency and timing of NCSE after cardiac arrest is unknown.

Methods Review of consecutive subjects treated with TH and receiving continuous EEG (cEEG) monitoring between 8/1/2009 and 11/16/2010. Demographic data, survival, and functional outcome were prospectively recorded. Each cEEG file was analyzed using standard definitions to define NCSE. Data were analyzed using descriptive and non-parametric statistics.

Results Mean age of the 101 subjects was 57 years (SD 15) with most subjects being male ($N = 55$, 54%) and experiencing out-of-hospital cardiac arrest ($N = 78$; 77%). Ventricular fibrillation was the initial cardiac rhythm in 39 (38%). All subjects received TH. Thirty subjects (30%) awoke at a median of 41 h (IQR 30, 61) after cardiac arrest. A total of 29/30 (97%) subjects surviving to hospital discharge were awake. Median interval from arrest to placement of cEEG was 9 h (IQR 6, 12), at which time the mean temperature was 33.9°C. NCSE occurred in 12 (12%) subjects. In 3/12 (25%) subjects, NCSE was present when the cEEG recording began. In 4 subjects, NCSE occurred within 8 h of cEEG recording. One (8%) subject with NCSE survived in a vegetative state.

Conclusions NCSE is common in comatose post-cardiac arrest subjects receiving TH. Most seizures occur within the first 8 h of cEEG recording and within the first 12 h after resuscitation from cardiac arrest. Outcomes are poor in those who experience NCSE.

Keywords Hypothermia · Cardiac arrest · Seizures · Status epilepticus · Coma · Anoxic brain injury · Outcomes

Background

Cardiac arrest is the most common cause of death in North America, resulting in approximately 350,000 deaths per year [1]. After resuscitation from cardiac arrest, the degree of brain dysfunction varies from consciousness without deficit to brain death [2]. However, the majority of patients resuscitated from cardiac arrest are comatose. Therapeutic hypothermia (TH) and standardized treatment protocols improve neurologic outcome in these patients [3–6]. Despite these interventions, a significant number of patients do not regain consciousness after resuscitation.

Seizures after cardiac arrest are associated with poor outcome [7]. Prolonged seizures may contribute to secondary brain injury after cardiac arrest. After traumatic brain injury, for example, seizures increase extracellular glutamate for several days, which may lead to cell death [8]. Seizures also increase glucose metabolism, raise intracranial pressure, increase neuronal lipid peroxidation, and disrupt cell membranes [9].

Prior literature in an undifferentiated cohort of comatose intensive care unit patients noted a frequency of

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electrographic seizures of 19% on continuous electroencephalography (cEEG) monitoring [10]. The frequency of seizures in post-cardiac arrest patients is reported between 3 and 44% [3, 5, 11–14]. All seizures in one pediatric study occurred more than 6 h after initiation of cEEG monitoring, with the majority found during rewarming [11]. However, the frequency and timing of nonconvulsive status epilepticus (NCSE) after cardiac arrest in adult patients is unknown. This study determined the frequency of NCSE in a cohort of cardiac arrest patients treated with TH.

Methods

In August 2009, our hospital added 22-channel digital continuous EEG recordings for the first 48 h after resuscitation from cardiac arrest as standard monitoring for all comatose post-cardiac arrest patients, as part of an ongoing quality improvement process [6]. A minority of subjects had video cEEG recordings obtained. For consecutive patients hospitalized after in-hospital or out-of-hospital cardiac arrest presumed because of cardiac etiology, demographic data, survival, and functional outcome were prospectively recorded in a quality improvement database. Analyses of these quality improvement data were deemed an exempt activity by the University of Pittsburgh Institutional Review Board. The Cerebral Performance Category (CPC) and Modified Rankin Score (mRS) were used as outcome measures. The five categories of the CPC are: CPC 1, conscious and alert with good cerebral performance; CPC 2, conscious and alert with moderate cerebral performance; CPC 3, conscious with severe cerebral disability; CPC 4, comatose or in persistent vegetative state; and CPC 5, brain dead, circulation preserved. The mRS was also used to evaluate disability. The mRS is a 7-point scale that ranges from 0 (no symptoms at all) to 6 (death). A good outcome was defined as a CPC of 1 or 2 or a mRS of ≤3. Both the measures are reported because they measure different aspects of the subject's outcome [15]. Additionally, time intervals from arrest to cEEG placement, from cEEG placement to seizure development, and from arrest to seizure development were recorded.

Convulsive seizures, including myoclonic, were defined as correlated clinical (motor) seizures and EEG seizure patterns. Myoclonic status epilepticus (MSE) was defined as a more than 30 min period of myoclonic jerks time locked with bursts in a burst-suppression pattern or associated with generalized periodic epileptiform discharges (GPEDs) [16]. Seizures were considered nonconvulsive (electrographic) if an EEG seizure pattern had no prominent motor clinical correlate on simultaneous video or on clinical examination.

Diagnostic criteria for NCSE are controversial and there are no agreed upon criteria to diagnose NCSE in obtunded or comatose patients [17].We defined an electrographic seizure as repetitive generalized or focal spikes, sharp waves, spike-and-wave or sharp-slow wave complexes at ≥3 Hz or sequential rhythmic, periodic, or quasi-periodic waves at ≥1 Hz with unequivocal evolution in frequency, morphology, or location lasting at least 10 s [18]. Electrographic status epilepticus or NCSE was defined as a state of impaired consciousness with: (1) a continuous electrographic seizure lasting 30 min or greater or recurrent electrographic seizures for over 30 min; (2) the presence of GPEDs lasting at least 30 min at a rate of ≥2.5 Hz; and (3) presence of GPEDs lasting at least 30 min at a rate of ≥1 Hz that evolved as described above. GPEDs at a rate of <2.5 Hz not satisfying criteria 3 above were classified as interictal GPEDs (Figs. 1, 2; Table 1). Our criteria represent a slight modification of several other published criteria [18–20].

EEGs were interpreted during patient care by board certified neurologists, and all files with malignant cEEG patterns were subsequently analyzed by two clinical neurophysiologists with expertise in electroencephalography (AP, RB).

Seizures were treated according to local protocol. TH (goal temperature 33°C for 24 h) is provided to the majority of comatose post-cardiac arrest patients in our facility and all subjects in this study. Induction is accomplished by rapid infusion of 30 cc/kg of 4°C saline along with surface cooling. Neuromuscular paralysis is used during induction but rarely employed during the maintenance and rewarming phases of TH. During TH, sedation is titrated for ventilator asynchrony or shivering. The rate of rewarming is 0.2–0.3°C/h. Once the temperature is >36°C, sedation is titrated for a Ramsay Sedation Scale of 3. Most subjects receiving TH treatment after cardiac arrest receive a propofol infusion titrated to suppress shivering, or midazolam infusion if hypotensive. Those experiencing NCSE or MSE are initially treated with a bolus of lorazepam followed by loading with phenytoin. Levetiracetam and valproic acid are employed next, followed by either a continuous infusion of midazolam or pentobarbital for refractory cases.

Initial neurologic examination within the first 6 h of resuscitation and without sedation or paralytic was recorded using the Full Outline of Unresponsiveness (FOUR) score by one of the physician authors (JCR, FXG, and CWC) [21]. This 16-point score is designed to evaluate the comatose patient with greater texture than the Glasgow Coma Scale, which is one subscale in the SOFA. The FOUR score is composed of a 0–4 score for Motor, Brainstem, Respiratory, and Eye responses. A lower score signifies greater impairment. Organ system dysfunction was determined using the individual organ dysfunction subscales of the Serial Organ Function Assessment (SOFA) scale [22]. The SOFA score ranges from 0 to 4 in each of

Fig. 1 a The start of a 3 min
electrographic seizure in an
80-year-old woman in NCSE.
b The evolving discharge 40 s
later. c The discharge ends,
followed by suppression
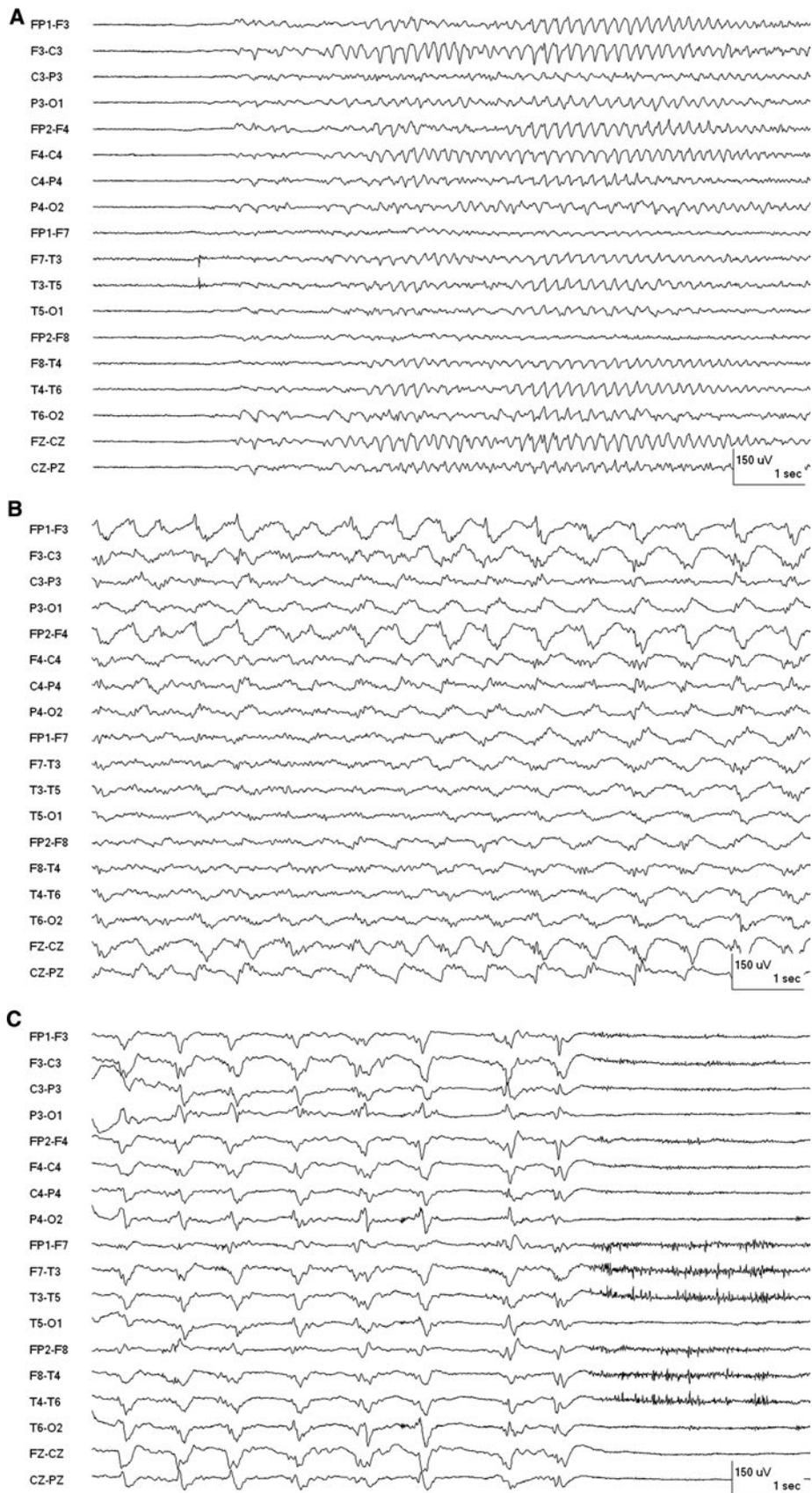
Fig. 2 Generalized periodic polyspikes in a 52-year-old woman

the following organ systems: cardiovascular, respiratory, nervous, liver, coagulation, and renal. A higher score signifies greater impairment. Four categories of post-cardiac arrest illness severity were defined by neurological examination and SOFA score on presentation [2] (Table 2). In prior work, category of illness severity is associated with survival, neurologic outcome, and development of multiple organ failure (MOF) [2]. MOF was defined as a score ≥3 on three or more subscales during the first 72 h of hospitalization.

Fisher's exact test and the Mann–Whitney test were used to explore differences in demographic and outcome data based on electrographic seizure development. Fisher's exact test was also used to determine associations between

electrographic seizure development and: (1) category of post-arrest illness severity, (2) initial rhythm of arrest, and (3) location of arrest (in-or out-of hospital cardiac arrest). These variables were chosen as they are known predictors of outcome. Data were analyzed using STATA v. 11.0 (STATA Corporation, College Station, TX).

Results

A total of 101 comatose subjects had cEEG between 8/1/2009 and 11/16/2010. None of the subjects had a prior history of epileptic disorder, use of tranquilizers, or anti-epileptic medications. Subjects had mean age of 57 years

Table 1 EEG and seizure definitions, modified from references [18–20]

Interpretation	Definition
Electrographic seizure	Repetitive generalized or focal spikes, sharp waves, spike-and-wave or sharp-slow wave complexes at ≥3 Hz or sequential rhythmic, periodic or quasi-periodic waves at ≥1 Hz with unequivocal evolution in frequency, morphology, or location lasting at least 10 s
NCSE	Impaired consciousness with:
	1. A continuous single electrographic seizure lasting 30 min or greater or recurrent electrographic seizures lasting for over 30 min
	2. Presence of GPEDs lasting at least 30 min at a rate ≥ 2.5 Hz
	3. Presence of GPEDs lasting at least 30 min at a rate of ≥ 1 Hz with unequivocal evolution in frequency, morphology, or location over time
MSE	More than 30-min period of myoclonic jerks or subtle facial movements locked in with bursts in a burst-suppression pattern or associated with GPEDs
Interictal GPEDs	GPEDs at a rate of < 2.5 Hz not satisfying criteria 3 above
EDs	Spikes, polyspikes, sharp waves, spike and wave or sharp-slow waves occurring independently, periodically (GPEDs), or during burst-suppression

MSE myoclonic status epilepticus, *GPEDs* generalized periodic epileptiform discharges, *NCSE* nonconvulsive status epilepticus, *EDs* epileptiform discharges

Table 2 Categories of initial post-arrest illness severity

Category	FOUR Motor + brainstem Score	SOFA Cardiovascular + respiratory Score	Description
I	8	Any	Awake—Follows commands
II	4–7	<4	Coma with preserved brainstem reflexes
III	4–7	≥4	Coma with preserved brainstem reflexes, and severe cardiopulmonary failure
IV	<4	Any	Coma with loss of some or all brainstem reflexes

FOUR full outline of unresponsiveness, *SOFA* serial organ function assessment

Table 3 Demographic data

|	Cohort (N = 101)	NCSE (N = 12)	MSE (N = 21)	Without NCSE or MSE (N = 68)
Age, in years (SD)	57 (15)	55 (15)	56 (16)	58 (15)
Male	55 (54%)	4 (33%)	10 (48%)	41 (60%)
Initial cardiac rhythm				
VF/VT	39 (38%)	4 (33%)	8 (38%)	27 (40%)
PEA	21 (21%)	0 (0%)	6 (28%)	15 (22%)
Asystole	28 (28%)	5 (42%)	5 (24%)	18 (26%)
Unknown	13 (13%)	3 (25%)	2 (10%)	8 (12%)
OHCA	78 (77%)	10 (83%)	19 (90%)	49 (72%)
Category of illness severity[a]				
II	39 (41%)	3 (28%)	7 (35%)	29 (45%)
III	14 (14%)	4 (36%)	1 (5%)	9 (14%)
IV	43 (45%)	4 (36%)	12 (60%)	27 (41%)
Temperature at EEG start (°C)	33.9 (1.5)	33.4 (1.0)	33.6 (1.3)	34.1 (1.5)
Followed commands	30 (30%)	1 (8%)	0 (0%)	29 (43%)
Time to following commands, in hours (IQR)	41 (30, 61)	221	–	39 (30, 60)
Survived*	30 (30%)	1 (8%)	0 (0%)	29 (43%)
Good outcome				
CPC 1–2	2 (2%)	0 (0%)	0 (0%)	2 (2%)
MRS 0–3	4 (4%)	0 (0%)	0 (0%)	4 (6%)
Length of stay, days (IQR)*	6 (4, 12)	5 (4, 6)	5 (4, 7)	9 (3, 14)
Time from arrest to cEEG recording, in hours (IQR)	9 (6, 12)	10 (4, 12)	8 (7, 11)	8 (6, 12)
cEEG record start to NCSE or MSE, in hours (IQR)	3 (0, 22)	7 (1, 24)	0 (0, 8)	–
Time from arrest to recognition of NCSE or MSE, in hours (IQR)	16 (12, 36)	15 (11, 37)	15 (10, 22)	–
Time from arrest to NCSE cessation, in hours (IQR)	38 (19, 60)	38 (19, 60)	–	–
Duration of NCSE, in hours (IQR)	5 (2, 19)	5 (2, 19)	–	–

[a] Unable to determine Category in 5 subjects due to paralysis on arrival. Times for arrest to NCSE cessation and duration of NCSE was available in 9 and 8 subjects, respectively

* $P < 0.05$ between the NCSE, MSE, and Without NCSE or MSE groups

VF/VT ventricular fibrillation/ventricular tachycardia, *PEA* pulseless electrical activity, *OHCA* out-of-hospital cardiac arrest, *IQR* interquartile range (25, 75%), *cEEG* continuous EEG

(SD 15) with a majority being male (N = 55, 54%) and experiencing out-of-hospital cardiac arrest (N = 78; 77%) (Table 3). Ventricular fibrillation was the most common rhythm of arrest and all subjects received TH. Goal temperature was achieved a median of 6 h (IQR 4, 10) after cardiac arrest. The most common post-cardiac arrest illness severity category was IV. Thirty subjects (30%) awoke at a median of 41 h (IQR 30, 61; range 15–603) after cardiac
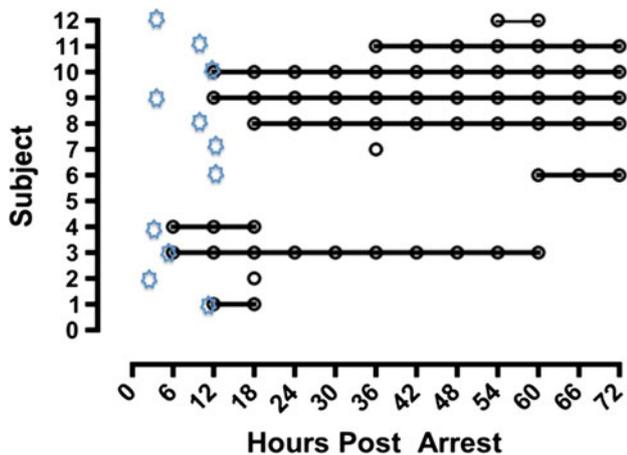
Fig. 3 Time course of NCSE. Time zero is time of arrest. *Line* represents duration of NCSE. *Open star* depicts time EEG started. Time of EEG start is unknown for Subject 5. *NCSE* nonconvulsive status epilepticus

Table 4 EEG findings in subjects without NCSE or MSE

Electroclincal findings	Incidence (%)
Interictal GPEDs	2 (2%)
One subject also had	
Burst-suppression	1 (1%)
Burst-suppression	15 (15%)
Triphasic waves	1 (1%)
Epileptiform discharges (not meeting any other criteria in Table 1)	6 (6%)
Diffuse slowing	35 (35%)

Results are based on abnormality and percentage of the total cohort in first line. *NCSE* nonconvulsive status epilepticus, *GPEDS* generalized periodic epileptiform discharges, *MSE* myoclonic status epilepticus

arrest. One subject died from MOF prior to hospital discharge. A total of 30 (30%) subjects survived to hospital discharge with 2 (2%) experiencing a CPC of 1–2 and 4 (4%) experiencing a mRS of ≤3 at hospital discharge. The majority of survivors (29/30; 97%) were awake at discharge. Subjects experiencing either NCSE or MSE were less likely to survive and had shorter hospital lengths of stay than those who did not experience NCSE or MSE ($P = 0.005$ and $P = 0.01$, respectively).

Interval from arrest to placement of cEEG was a median of 9 h (IQR 6, 12). Mean temperature was 33.9°C when cEEG was placed. NCSE occurred in 12 (12%) subjects (Table 3). Only one subject with NCSE had accompanying subtle clinical manifestations that were noted on review of the cEEG video file. NCSE was present in 3 of the 12 (25%) at the onset of the recording; an additional 4 (33%) occurred within 8 h (Fig. 3). NCSE ended in 8/12 (67%)

subjects at a median of 38 h (IQR 19, 60) following cardiac arrest. Median duration of NCSE was 5 h (IQR 2, 19). EEG patterns evolved from interictal GPEDs to NCSE in two subjects and from a prior burst-suppression pattern to NCSE in four subjects. Six subjects met criteria for NCSE based on the frequency of or evolving GPEDs; six had recurrent electrographic seizures. NCSE frequency did not differ between categories of post-arrest illness severity ($P = 0.171$), rhythm of arrest ($P = 0.113$), or location of arrest ($P = 0.727$). The most common category of post-arrest illness severity in survivors was category II ($N = 18$) and category IV in nonsurvivors ($N = 39$). Similarly, category II was more common in those with a CPC of 1–2 ($N = 1$) or mRS ≤ 3 ($N = 2$).

One subject in NCSE with recurrent electrographic seizures survived and was discharged to a long-term acute care facility without regaining consciousness. Her discharge CPC was 4 and mRS was 5. Another subject with NCSE defined by high frequency GPEDs awoke but later died from multiple organ failure in the hospital.

A burst-suppression pattern not associated with MSE was found in 30 (30%) subjects. One subject in MSE later met criteria for NCSE and was only counted as MSE. Two subjects in NCSE that later developed MSE were only counted as NCSE. Five (17%) subjects with burst-suppression pattern survived. All five were on propofol infusion for sedation and shivering suppression throughout the cEEG recording. MSE was seen in 21 subjects (21%). No subject with MSE regained consciousness or survived. EEG rhythms in subjects not experiencing NCSE or MSE are shown in Table 4.

Antiepileptic medications were frequently administered to subjects in response to cEEG findings. All subjects with NCSE were treated with antiepileptic medications. The majority of the subjects with interictal GPEDS (1/2, 50%) and MSE (19/21, 90%) were treated with antiepileptic medications. The frequency of use of other medications did not differ between subjects with NCSE, MSE, or neither (Table 5).

In two subjects, cEEG monitoring proved diagnostic for acute hemodynamic changes. The first was a 60-year-old female who became acutely tachycardiac and hypertensive. The primary team began treatment with hydralazine and noted NCSE on the cEEG. She received a bolus of midazolam and infusion of levicetaram with cessation of NCSE and improvement of hemodynamics. This subject later awoke but died from multiple organ failure later in her hospital course. The second was a 41-year old female who became acutely hypotensive while she was on a norepinephrine infusion. The norepinephrine infusion was increased and steroids were administered given the concern for possible adrenal suppression. Again, cEEG monitoring revealed NCSE, which was treated with midazolam bolus and infusion with cessation of NCSE and improvement in hemodynamics.

Table 5 Medications administered during cEEG recording

Medication	Overall (N = 101)	NCSE (N = 12)	MSE (N = 21)	Without MSE or NCSE (N = 68)
Continuous infusions				
Propofol	81 (80%)	12 (100%)	16 (76%)	53 (78%)
Midazolam	25 (25%)	7 (59%)	7 (33%)	11 (16%)
Fentanyl	51 (50%)	6 (50%)	8 (38%)	37 (54%)
Intermittent sedatives				
Diazepam	7 (7%)	0 (0%)	0 (0%)	7 (10%)
Lorazepam	37 (37%)	8 (67%)	12 (57%)	17 (25%)
Antiepileptic medications				
Pentobarbital	3 (3%)	1 (8%)	2 (10%)	0 (0%)
Valproic acid	11 (11%)	5 (42%)	6 (28%)	0 (0%)
Carbamazepine	2 (2%)	1 (8%)	0 (0%)	1 (1%)
Phenytoin	22 (22%)	6 (50%)	11 (52%)	5 (7%)
Fosphenytoin	14 (14%)	6 (50%)	6 (29%)	2 (3%)
Levetiracetam	28 (28%)	8 (67%)	16 (76%)	4 (6%)
Antibiotics				
Cefepime	5 (5%)	0 (0%)	1 (5%)	4 (6%)
Piperacillin–tazobactam	19 (19%)	1 (8%)	2 (10%)	16 (24%)
Ampicillin	1 (1%)	0 (0%)	0 (0%)	1 (1%)
Ampicillin–sulbactam	27 (27%)	5 (42%)	3 (14%)	19 (28%)
Moxifloxacin	5 (5%)	1 (8%)	1 (5%)	3 (4%)
Ciprofloxacin	3 (3%)	1 (8%)	1 (5%)	1 (1%)
Metronidazole	10 (10%)	1 (8%)	2 (10%)	7 (10%)
Vasopresors/inotropes				
Dopamine	31 (31%)	3 (25%)	9 (42%)	19 (28%)
Vasopressin	10 (10%)	0 (0%)	0 (0%)	10 (15%)
Epinephrine	8 (8%)	0 (0%)	2 (10%)	6 (9%)
Norepinephrine	49 (49%)	9 (75%)	6 (29%)	34 (50%)
Phenylephrine	7 (7%)	0 (0%)	1 (5%)	6 (9%)
Dobutamine	5 (5%)	0 (0%)	2 (10%)	3 (4%)
Milrinone	3 (3%)	0 (0%)	0 (0%)	3 (4%)

NCSE nonconvulsive status epilepticus, *MSE* myoclonic status epilepticus

Discussion

NCSE is common (12%) in comatose subjects receiving TH following cardiac arrest. In this cohort, only one subject in 12 demonstrated subtle clinical manifestations during the seizure. Given the significant risk for NCSE in post-cardiac arrest subjects, these data support cEEG monitoring for all comatose subjects receiving TH. Most NCSE developed within the first 8 h of cEEG recording. However, the median time from arrest to cEEG placement was 9 h, and some patients may experience NCSE prior to cEEG placement. The median time from arrest to NCSE development was 15 h. Increasing time to diagnosis of NCSE has been associated with increased mortality in other patient cohorts [20]. We believe that earlier EEG monitoring of comatose post-cardiac arrest patients should be a goal for the resuscitation community.

These data are relevant to recent guidelines recommending frequent or continuous EEG recording in all post-cardiac arrest patients [23]. Intermittent EEG sampling may fail to detect all seizures. In this cohort, only three subjects had NCSE when cEEG was started, while nine subjects initially were in another EEG pattern that progressed to NCSE. Moreover, two subjects demonstrated acute hemodynamic changes that improved with treatment of NCSE. Burst-suppression was the most common EEG pattern recorded, and four patients with burst-suppression progressed to NCSE. Given this dynamic progression, cEEG is superior to intermittent EEG recordings.

This series is too small to precisely estimate survival for patients with each EEG pattern. However, subjects in NCSE had worse outcomes than the overall cohort of post-cardiac arrest patients. Only one subject with NCSE awoke, but subsequently died from non-neurological

complications. The one subject in NCSE who survived remained in a vegetative state and was discharged to a long-term acute care facility. The poor outcomes for subjects with MSE in this cohort are similar to other recent series [16, 24, 25].

An important question is whether NCSE contributes to poor outcome after cardiac arrest or are simply an epiphenomenon of severe brain injury. It is unknown if prophylactic antiepileptic medications would reduce NCSE frequency or improve neurologic outcome. One recent study demonstrated an improvement in the Disability Rating Scale at 3 months and the Glasgow Outcome Scale at 6 months in subjects treated with levetiracetam after traumatic brain injury [26]. Interestingly, this was not accompanied by a reduction in seizure incidence and may instead be due to a direct neuroprotective effect [27]. Previous studies did not find any improvement in outcome after cardiac arrest with thiopental administration [28] or diazepam administration [29]. However, neither study examined seizure frequency, and both were conducted at times when intensive care differed significantly from current practice. A study of preventative antiepileptic medications after cardiac arrest that documented EEG changes could clarify the contribution of NCSE to brain injury.

Limitations to this study are the single-center design. A larger, multi-center study using common data definitions could provide more precise estimates of seizure prevalence in this population. The definition of NCSE can be debated and alternate definitions may result in different frequencies. Our definition of NCSE includes both recurrent seizures and a subset of GPEDS, but is relatively conservative and is a minor adaptation from prior NCSE definitions [18–20]. It is important to recognize that there is a continuum of EEG findings with a continuum of risk for secondary neuronal injury [18]. Our facility routinely treats comatose subjects with propofol or benzodiazepines for shivering suppression during induction, maintenance, and rewarming from TH. These sedatives may alter seizure frequency, and propofol in particular may increase the frequency of burst-suppression patterns. The use of propofol along with TH may explain the high survival rate of subjects with burst-suppression patterns in this study. Future studies should consider more frequent interruption of sedation for cEEG interpretation. Finally, the time between resuscitation from cardiac arrest and cEEG recording averaged 9 h. A significant number of subjects may have experienced NCSE prior to cEEG monitoring. Most of the subjects treated in our facility are referred in from outlying hospitals, contributing to this delay.

Conclusions

In this cohort of comatose post-cardiac arrest subjects receiving TH, NCSE is common. Most seizures occur within the first 8 h of cEEG recording and within the first 12 h after resuscitation from cardiac arrest. Many seizures develop from a burst-suppression pattern. Outcomes are poor in those who experience NCSE.

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