Review article

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Non-convulsive status epilepticus in the elderly

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ABSTRACT – Altered mental state is a very common presentation in the elderly admitted to the emergency department. It has been determined that about 16% of patients aged 60 or older with confusion of unknown origin have non-convulsive status epilepticus. The diagnosis of non-convulsive status epilepticus is difficult in the elderly because possible aetiologies of confusion may present with the same clinical picture. Non-convulsive status epilepticus in the elderly carries major morbidity and mortality, attributable primarily to aetiology, and treatment is complex, involving treatment of the aetiology and concomitant medical illnesses, whilst balancing the side effects and drug interactions of antiepileptic drugs.

Key words: non-convulsive status epilepticus, elderly, mental state, EEG, etiology

Non-convulsive status epilepticus (NCSE)

Definition and criteria for NCSE

Non-convulsive status epilepticus is a term that covers a range of disparate conditions, denoting prolonged electrographic seizure activity (set arbitrarily at 30 minutes) with resultant non-convulsive clinical symptoms (Walker *et al*., 2005).

The electrographic seizure activity is divided into clear-cut and equivocal criteria (*table 1*).

There continues to be difficulties and controversies (equivocal criteria; description number 5 in *table 1*) with regards to determining EEG abnormal periodic discharges as epiphenomena in severely injured brains or as harmful epileptiform discharges that could lead to additional brain injury. One theory is that periodicity is a surrogate marker for severe cerebral injury and does not cause further brain injury. Another hypothesis is that periodic discharges and seizures in the acutely-injured brain lead to secondary neuronal injury via excessive metabolic demand, excitotoxicity, or other mechanisms (Claassen, 2009). Chong and Hirsch (2005) proposed an ictal-interictal injury continuum (*figure 1*) to put into context the likelihood of neuronal injury for each type of discharge in a given clinical setting in order to guide management (Chong and Hirsch, 2005).

A working definition used in some studies is the Young's criteria in adults (*table 2*) (Young *et al*., 1996). Chong and Hirsch (2005) discussed two caveats of Young's criteria. Firstly, the criteria were intended to be specific to seizures and not necessarily for the purpose of accurate diagnosis. Therefore, a pattern that does not fulfil these criteria

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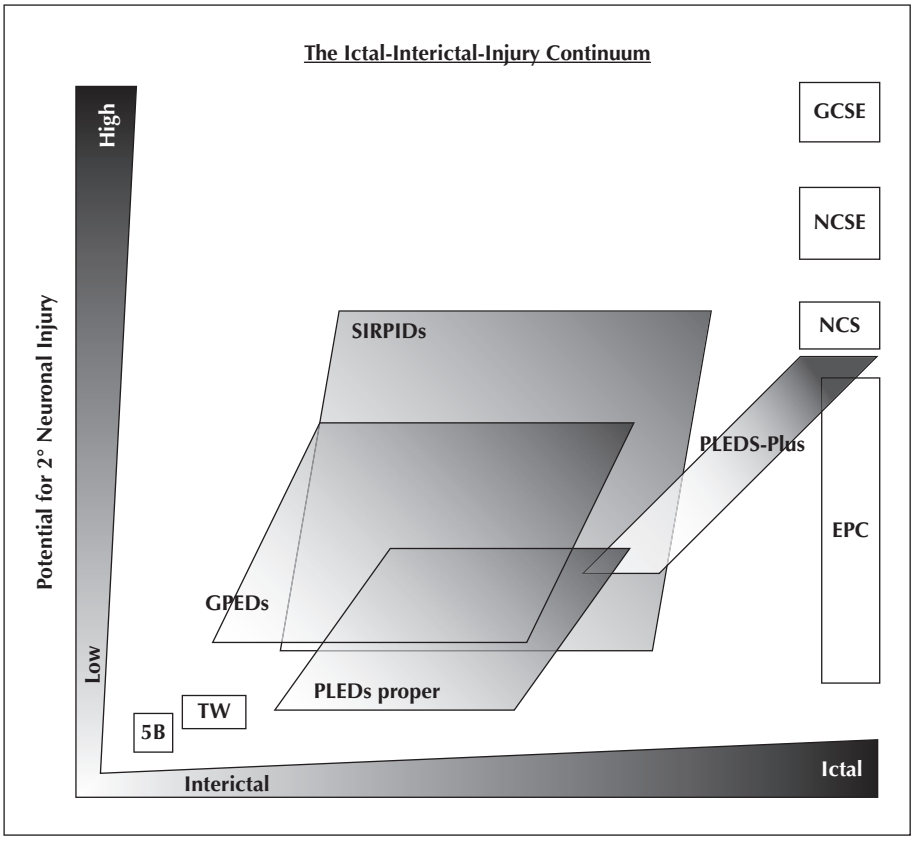

Figure 1. The ictal- interictal-injury continuum plot demonstrating various clinico-electrographic diagnoses. The potential for secondary neuronal injury (shown on the y-axis) should be a more important indicator of whether treatment should be aggressive. If clinical correlate is present with any of the patterns, it would be considered ictal by definition, though this does not necessarily suggest an appreciable increase in the likelihood of neuronal injury (Chong and Hirsch, 2005).
EPC: epilepsia partialis continua; GCSE: generalized convulsive status epilepticus; GPEDs: generalized periodic epileptiform discharges; NCS: non-convulsive seizures; NCSE: non-convulsive status epilepticus; PLEDs: periodic lateralized epileptiform discharges; S-B: suppression burst; SIRPIDs: stimulus induced rhythmic, periodic, or ictal discharges; TW: triphasic waves.

could still be ictal, but cannot be proven based on the EEG pattern alone. Secondly, regarding number 4 of the secondary criteria ("significant improvement in clinical state or baseline EEG after antiepileptic drugs"), Chong and Hirsch (2005) cited the example of triphasic waves as non-ictal period patterns which are often eradicated with a bolus of benzodiazepines, thus qualifying as a seizure based on Young's criteria. Therefore, more stringent criteria are required in the case of the appearance of previously absent EEG patterns alone to suggest a seizure (*table 3*) (Chong and Hirsch, 2005). This appears to be a stricter set of criteria, to be used in clinical practice and research. There is a need to validate both the Young's and the modified Young's criteria. The Young's and modified Young's working criteria appear to encompass all the criteria (clear-cut and equivocal) presented in *table 1*.
With the secondary criteria (*table 3*), it is difficult to provide a consensus on the type and dose of "rapidly acting antiepileptic drugs (AEDs)" used, whether patients should have an immediate or delayed response, and to what degree an improvement is considered "significant".

Epidemiology

The epidemiological data of NCSE is fraught with methodological problems. Attempts to estimate have yielded an overall population incidence of NSCE between 5.6-18.3/100,000/year (from direct epidemiological studies) and 32-85/100,000/year (extrapolation from non-epidemiologically based data) (Walker *et al*., 2005).

Classification

The classification of NCSE can be subdivided by age, and further subdivided according to the forms of NCSE observed in epileptic encephalopathies, cerebral development, aetiology and syndrome (Sutter and

Table 1. Criteria for electrographic seizure activity (Walker *et al*., 2005).

Criteria	Descriptions
Clear-cut criteria	1. Frequent or continuous focal electrographic seizures, with ictal patterns that wax and wane with change in amplitude, frequency, and/or spatial distribution.
	2. Frequent or continuous generalized spike wave discharges in patients without a prior history of epileptic encephalopathy or epilepsy syndrome.
	3. Frequent or continuous generalized spike-wave discharges, which show significant changes in intensity or frequency (usually a faster frequency) when compared to baseline EEG in patients with an epileptic encephalopathy or epilepsy syndrome.
	4. Periodic lateralised epileptiform discharges (PLEDs) or bilateral periodic epileptiform discharges (BiPEDs) occurring in patients in coma in the aftermath of a generalized tonic-clonic status epilepticus (subtle status epilepticus).
Equivocal	5. Frequent or continuous EEG abnormalities (spikes, sharp waves, rhythmic slow activity, PLEDs, BiPEDs, generalized periodic epileptiform discharges [GPEDs], triphasic waves) in patients whose EEG showed no previous similar abnormalities, in the context of acute cerebral damage (*e.g*. anoxic brain damage, infection, trauma).
	6. Frequent or continuous generalized EEG abnormalities in patients with epileptic encephalopathies in whom similar interictal EEG patterns are observed, but in whom clinical symptoms are suggestive of NCSE.

Table 2. Young's criteria for non-convulsive seizures. NCSE is defined as any pattern lasting for at least 10 seconds, with at least one primary criteria 1-3 and one or more secondary criteria.

Criteria	EEG features
Primary criteria	Repetitive generalized or focal spikes, sharp waves, spike-wave or sharp-and-slow wave complexes at >3/sec._Repetitive generalized or focal spikes, sharp waves, spike-and-wave or sharp-and-slow wave complexes at <3/sec and secondary criterion 4._Sequential rhythmic waves and secondary criteria 1, 2 and 3, with or without 4.
Secondary criteria	Incrementing onset: increase in voltage and/or increase or slowing of frequency._Decrementing offset: decrease in voltage or frequency._Post-discharge slowing or voltage attenuation._Significant improvement in clinical state or baseline EEG after antiepileptic drugs.

Table 3. Modified Young's criteria. NCSE is defined as any pattern satisfying any of the primary criteria and lasting ≥10 seconds (for non-convulsive seizures) or ≥30 minutes (for NCSE) (Chong and Hirsch, 2005).

Criteria	EEG features
Primary criteria	Repetitive generalized or focal spikes, sharp waves, spike-and-wave complexes at ≥3/sec._Repetitive generalized or focal spikes, sharp waves, spike-and-wave or sharp-and-slow wave complexes at <3/sec and secondary criterion._Sequential rhythmic, periodic, or quasi-periodic waves at ≥1/sec and unequivocal evolution in frequency (gradually increasing or decreasing by at least 1/sec, *e.g*. 2 to 3/sec), morphology, or location (gradual spread into or out of a region involving at least two electrodes). Evolution in amplitude alone is not sufficient.
Secondary criteria	Significant improvement in clinical state or appearance of previously absent normal EEG patterns (such as posterior-dominant "alpha" rhythm), temporally coupled to acute administration of a rapidly acting antiepileptic drug. Resolution of the "epileptiform" discharges leaving diffuse slowing without clinical improvement and without appearance of previously absent normal EEG patterns would not satisfy the secondary criterion.

Kaplan, 2012). Boundary syndromes are a separate subset in which it is unclear to what extent the clinical symptoms are due to NCSE or to underlying cerebral damage/dysfunction. A compendium of EEG patterns observed in NCSE has started to help clarify this classification (Sutter and Kaplan, 2012).

Neuroimaging and biomarkers

Clinical neuroimaging in NCSE is still limited to computed tomography (CT) or magnetic resonance imaging (MRI) to detect structural abnormalities. Other imaging techniques including positron emission tomography (PET), single-photon emission computed tomography (SPECT), functional MRI, and perfusion and diffusion imaging are time-dependent, impractical, and have limited sensitivity. Ideally, neuroimaging should detect neuronal changes associated with NCSE and should be comparable, if not superior, to EEG in the diagnosis of NCSE.

Biomarkers of neuronal injury, such as neuron-specific enolase, glutamate, glycerol, and lactate-pyruvate ratio may increase after electrographic seizures. Their use is limited, as these analyses are performed on cerebral microdialysis samples. Imaging findings of the interictal-ictal continuum are not well studied, and at best, interpretation is extrapolated from findings observed in convulsive status epilepticus or in the setting of acute brain injury. At this point in time, ancillary testing, such as other neuroimaging techniques and the use of biomarkers mentioned above, could be used in situations where it is equivocal whether the periodic discharges are contributing to additional neuronal injury, to guide aggressive management (Claassen, 2009).

Clinico-electrographic features and management

Management guidelines are difficult to establish because of the varied definitions used for NCSE, the heterogeneity of presentations, and outcome measures. Management should include treatment of epileptic discharges, treatment of aetiology, treatment of associated encephalopathy, and treatment of behavioural and cognitive difficulties.

I will divide NCSE into three broad scenarios for ease of discussion of management summary guidelines (*table 4*) (Walker *et al*., 2005).

There are also many unusual alterations in consciousness, behaviour, and neurological function that can be manifestations of NCSE (Drislane, 2000; Kaplan, 2005). Drislane (2000) suggested clues to suspect NCSE: occurrence following generalized convulsions or generalized convulsive status epilepticus; subtle signs such as twitching, blinking, and nystagmus in a stuporous patient; otherwise unexplained stupor or confusion, especially in the elderly; altered mental status in the elderly, particularly after benzodiazepine withdrawal; a history of seizures and new medical or surgical stress; and "stroke plus" in patients who appear to fare worse or recover less rapidly than those typically recovering from a stroke.

Prognosis

The prognosis of NCSE is hard to ascertain due to the wide variation in patient presentation and premorbid function, as well as definitions and classifications used, the difficulty in determining duration of NCSE and determining what outcomes are important in prognosis (cognitive, behavioural, morbidity or mortality), the different patient groups studied (age groups, critically ill patients, various co-morbidities, and aetiology of cerebral injury), and the difficulty in differentiating between the morbid effects of treatment and the morbidity of the disease itself (Kaplan, 2005; Walker *et al*., 2005).

Mortality in NCSE is probably largely a function of NCSE aetiology and relates to a level of consciousness and the development of complications, rather than to a particular type or pattern of EEG. Shneker and Fountain (2003) found that patients presenting with NCSE were associated with high mortality (18%) and morbidity (39%). Mortality due to epilepsy alone was 3%, whereas in those presenting with concomitant brain or systemic injury was 27%. When grouped according to level of consciousness, patients presenting with minimal obtundation had a morbidity and mortality of 7%, whereas those presenting with deep lethargy or coma had morbidity and mortality of 39% (Shneker and Fountain, 2003). These indicators of mortality were found to be exerting an independent effect. Young *et al*. (1996) found that mortality is linked strongly to duration and delay to diagnosis of NCSE (Young *et al*., 1996).

The decision on how rapidly and aggressively to treat NCSE is fundamentally complex. A decision model has been proposed to investigate the interplay of factors influencing NCSE management decisions. Ferguson *et al*. (2013) have incorporated five variables: baseline mortality rate for specific aetiologies of NCSE, efficacy of non-aggressive treatment, impact of aetiology on outcome, excess disability attributable to delayed seizure control, and mortality risk of aggressive treatment. They then applied the model to four specific aetiologies of NCSE: absence epilepsy, discontinuation/non-compliance of AEDs, intra-parenchymal haemorrhage, and hypoxic ischaemic encephalopathy. The conclusion based on this model is that non-aggressive treatment is favoured for aetiologies with low morbidity and mortality, such

Table 4. Three broad scenarios of NCSE, clinico-electrographic features, and management guidelines.

Types	Clinico-electrographic features	Management summary
Subtle generalized convulsive status epilepticus	The presence of NSCE EEG pattern after the control of clinical convulsive status epilepticus is a predictor of mortality and poor outcome independent of aetiology and age.	EEG should be used in the evaluation of comatose patients and treatment institution. Treatment guidelines at the moment is as per convulsive status epilepticus, including intubation and induction of pharmacological coma.
"Generic" NCSE comprising of absence and complex partial NCSE (Walker *et al*., 2005)	Absence status epilepticus (ASE): Typical: prolonged state of altered consciousness, associated with generalized 3-Hz spike-wave EEG activity, often with a past history of typical absences or generalized tonic-clonic seizures. Atypical: occur in genetic generalized epilepsies, are usually associated with learning disability, other neurological abnormalities or multiple seizure types. *De novo* late-onset ASE is a distinct condition with diverse pathological conditions and warrants different management, often precipitated by toxic or metabolic factors with no past history of epilepsy and often following benzodiazepines or medication withdrawal. Complex partial status epilepticus (CPSE): presents with confusional state (sometimes punctuated by periods of less responsiveness and stereotyped automatisms), an ictal EEG with focal discharges such as those of isolated complex partial seizures on an abnormal background, prompt response of the behaviour and EEG to intravenous AEDs, and an interictal epileptiform focus in one or more temporal or frontal lobes.	The main goals of treatment are to clear EEG of epileptic discharges and to identify provoking agents and events. Benzodiazepines, phenytoin and valproate are used as first-line AEDs. There is uncertainty with regards to how aggressive the treatment should be, as there are significant morbidities resulting from the treatment. The general consensus is to use the least sedating AED in the smallest doses to achieve resolution of epileptic discharges.
Epileptic Encephalopathies	This concept reflects the clinical impression that seizures actually alter the course of normal brain development, and is defined based on the following: "the epileptic activity itself may contribute to severe cognitive and behavioural impairments above and beyond what might be expected from the underlying pathology alone and that these can worsen over time". 2. This term is also used to refer to a group of epilepsy syndromes (chiefly West, Lennox-Gastaut, Dravet, and Landau-Kleffner syndromes, and others) in which the occurrence of seizures and disordered electrographic activity is thought to contribute to the developmental disability commonly observed in these syndromes (Berg and Millichap, 2013).	Treatment is based on the hypothesis that epileptic discharges are causally related to the encephalopathy and therefore treatment of discharges may improve outcome. Pharmacological therapy is used together with behavioural and educational interventions.

as absence epilepsy and discontinuation of antiepileptic drugs. The risk of aggressive treatment is only warranted for aetiologies where there is significant risk of seizure-induced neurological damage (Ferguson *et al*., 2013).

Non-convulsive status epilepticus in the elderly

NCSE is often difficult to diagnose in the elderly (aged 60 years and older). This is because of superimposed comorbid medical conditions and medications, and age-related cognitive difficulties that might be blamed when present. This diagnosis thus requires a high degree of suspicion, availability of emergent or continuous EEG, and involvement of neurologists or epileptologists in the diagnosis and management.

The causes of NCSE in the elderly can be divided into:

– Diseases of the central nervous system, especially cerebrovascular disease (whether acute or remote) and neurodegenerative diseases such as Alzheimer's disease, tumours, and trauma;

– Multifactorial, with acute metabolic or infectious precipitants superimposed on an already fragile brain

– Exacerbation of earlier epilepsy (Werhahn, 2009).

NCSE in the elderly carries major morbidity and mortality. The mortality rate was reported to be 30% in a group with NCSE compared to a control group without NCSE with a mortality rate of 5.9%. This group of elderly patients with NCSE also had longer periods of hospitalization and poor functional outcome (Bottaro *et al*., 2007). In the critically ill elderly patients with NCSE, severity of illness correlated with mortality, and aggressive treatment may not improve outcome (Litt *et al*., 1998).

Management in the elderly includes treating the aetiology of NCSE, management of the comorbid medical illnesses, and balancing out the side effects and drug interactions of introduced therapies or AEDs.

Management is outlined in *table 4* with special considerations in the elderly, including decreased tolerability of AED therapy due to age-related changes in pharmacokinetics and pharmacodynamics, the presence of polypharmacy with increased probability of drug-drug interactions or idiosyncratic reactions, and physical and psychiatric comorbidities. *Table 5* (adapted from Ruegg (2008)) gives a guideline for dose adjustments accounting for age for patients who present with NSCE fitting into the subtle generalized convulsive status epilepticus type. Most elderly patients remain on AED therapy for life as there is insufficient data to determine whether safe withdrawal of AED therapy can be attempted in the elderly after a period of seizure freedom.

Non-convulsive status epilepticus as a cause of altered mental states in the elderly

It is difficult to determine the prevalence of NCSE in patients presenting with acute altered mental states (AMS) whose cause cannot be quickly determined, as designs of the study have varied. At the moment, prevalence is reported to be between 8 and 30% from indirect sources, with two of these studies performed in the elderly population (Alroughani *et al*., 2009; Veran *et al*., 2010; Zehtabchi *et al*., 2011; Shavit *et al*., 2012).

To allow comparison between similar populations, there is a need to (Zehtabchi *et al*., 2011):

– Standardize definitions of NCSE used in the various studies. The modified Young's criteria appear more stringent than the Young's criteria.

– Prospectively enrol consecutive patients presenting with AMS; up to 53% of patients were not suspected to have NCSE by treating physicians (Seidel *et al*., 2012), therefore consecutive patients should be enrolled.

– Have larger sample sizes.

– Decide on the duration of EEG recording and monitoring and how quickly from presentation this should be performed. For example, is a routine 20-minute EEG shortly after presentation to the emergency department (within an hour) adequate? Is a continuous EEG (duration of 24-48 hours) within 72 hours of presentation the gold standard? Would this "gold standard" result in a delay in the diagnosis of NCSE by 72 hours? It is recommended that in non-comatose patients, at least 24 hours of continuous EEG should be performed, and in comatose patients, at least 48 hours is required (Claassen *et al*., 2004). Issues in delays in obtaining an EEG, especially after several hours or a weekend, also need to be addressed.

– Validate cases with particular attention to inter-rater reliability of EEG interpretation.

– Define premorbid functional status.

– Define outcome measures which are practical and clinically relevant (medium and long-term cognitive and behavioural outcomes are important outcome measures aside from morbidity and mortality).

– Analyze "pure" populations of patients with the same eventual NCSE syndrome and same underlying aetiology.

There have been attempts to determine clinical features that could predict NCSE. In an adult population (aged 15-89 years of age), remote risk factors for seizures, a depressed mental state, and ocular movement abnormalities were more common in patients in the NCSE group (Husain *et al*., 2003). In the elderly population ($\geq$60 years of age) who present with confusion of unknown origin, female gender, rapid onset (<24 hours), and a lack of response to simple

Table 5. Drug dosing for NCSE; adjusting for age in the case of subtle generalized convulsive status epilepticus.

Drug		Dosing	
A) First-line (adequate respiratory and blood pressure support must be available)			
Lorazepam	<60y	4mg i/v	Repeat once
	60-80y	2mg i/v	Repeat 1-3 times
	>80y	1mg i/v	Repeat up to 5 times
Clonazepam	<60y	1mg i/v	Repeat 1-2 times
	60-80y	0.75mg i/v	Repeat 1-3 times
	>80y	0.5mg i/v	Repeat up to 3 times
Midazolam	<60y	5mg i/v	Repeat 1-2 times
	60-80y	2mg i/v	Repeat 1-3 times
	>80y	1mg i/v	Repeat up to 5 times
B) Second-line			
Phenytoin			
Loading		15-18mg/kg body weight at 12.5-25mg/min	
Maintenance	<70kg	150mg i/v every 12 hours ; start 6 hours after loading	
	70-90kg	175mg i/v every 12 hours ; start 6 hours after loading	
	>90kg	200mg i/v every 12 hours ; start 6 hours after loading	
Valproic acid			
Loading	<60y	30-45mg/kg i/v in 30 minutes	
	60-80y	20-30mg/kg i/v in 30 minutes	
	>80y	15-25mg/kg i/v in 30 minutes	
Maintenance		Start the same dose as the loading dose concomitantly over 24 hours	
Levetiracetam			
Loading	<60y	25-30mg/kg i/v in 15 minutes	
	60-80y	15-25mg/kg i/v in 15 minutes	
	>80y	10-20mg/kg i/v in 15 minutes	
Maintenance	<60y	1000-1500mg every 12 hours	
	60-80y	750-1000mg every 12 hours	
	>80y	500-750mg every 12 hours	

commands appear to be statistically more frequent in the NCSE group compared to the non-epileptic confusion group (Veran *et al*., 2010).

An ideal EEG system

Since a high degree of suspicion and the prompt use of EEG are required to detect NCSE in elderly patients who present with AMS, emergency EEG systems have been proposed to be used in acute care settings (Abdel Baki *et al*., 2011).

An abbreviated EEG (with a duration of five minutes) using a preformed electrode placement pattern has been suggested to be incorporated into the early workup of patients who present with AMS of unknown cause, to provide useful and immediate information without interrupting the routine workup of these patients in the emergency department (Bautista *et al*., 2007). In this study, the results from the abbreviated EEG were not compared with those of routine or continuous EEG monitoring as the "gold standard". It could be ideal and cost effective to use an initial abbreviated EEG at presentation, together with clinical features, to predict patients with NSCE, or to risk stratify patients in order to then perform a routine or continuous EEG for those identified by the abbreviated EEG as being at high risk of developing NCSE.

Periodic lateralised epileptiform discharges (PLEDs), generalized periodic epileptiform discharges (GPEDs), and suppression burst were frequently observed in patients with seizures on continuous EEG monitoring and could be used as EEG indicators to risk stratify patients at high risk (Claassen *et al*., 2004). In patients with seizures, there appeared to be a transition from PLEDs-proper to PLEDs-plus, to seizure, with a cycle back to PLEDs-proper. Therefore, patients with PLEDs-plus detected by EEG were more likely to have a seizure during the acute illness compared to patients with solely PLEDs-proper (Chong and Hirsch, 2005).

A survey of neurologists' use of EEG monitoring demonstrated that there is significant variation in terms of continuous EEG indications, initiation of urgent treatment, duration and frequency of review, and management of NCSE. This indicates that there is a need to clarify the optimal use of continuous EEG and the treatment of NCSE (Abend *et al*., 2010).

Continuous EEG monitoring has been shown to have a significant effect on clinical decision-making, in terms of the need for an AED and the effect of prescribed AED therapy in real time (Kilbride *et al*., 2009; Seidel *et al*., 2012). An increase in the implementation of continuous EEG could correspond to an increase in NCSE diagnosis. In the traditional setting, continuous EEG recordings generate huge amounts of data and are reviewed by an experienced encephalographer periodically. Analytical tools, such as quantitative EEG and compressed EEG pattern analysis, may be capable of alerting physicians of ongoing electrographic seizure activity (Shah *et al*., 2006; Brenner, 2009). There can be many challenges when monitoring EEG continuously in the intensive care setting (Sutter *et al*., 2013).

Future directions

At present, there are many unanswered questions. There is a need for a prospective study to determine the prevalence of NCSE, specifically consecutive elderly patients who present with AMS, in order to investigate a population ranging from patients in the community presenting with acute confusion to patients in intensive care in a coma. There probably is a need also to look at the prevalence of NCSE due to specific aetiologies in the elderly, as NCSE associated with sepsis is different from NCSE in patients with a remote history of epilepsy.

Clinical features should be identified in these elderly patients, which would predict NCSE with adequate sensitivity and specificity. Management guidelines on the duration and cost effectiveness of emergency and abbreviated EEGs in the context of investigating these elderly patients who present with AMS should also be developed.

It also remains unclear what the prognosis of NCSE is, independent of the primary aetiology. If NCSE in the elderly does not contribute to a worse outcome, then there is less need to subject an elderly patient to aggressive treatment of NCSE, which would more likely contribute to iatrogenic complications. A decision analysis tool has been proposed to weigh the benefits and risks of aggressive treatment with the morbidity and mortality of NCSE and the primary aetiology.

Conclusion

In an ideal scenario, elderly patients with altered mental states would undergo an array of investigations, performed routinely in the emergency department without delay. NCSE could be detected on the initial emergent EEG, and together with other neuroimaging and biochemical investigations, the aetiology of the NCSE might be determined and then a decision made regarding the appropriate treatment. The treatment plan could be analyzed for risk and benefits with the aid of a decision analysis tool. □

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The author has no conflict of interest to disclose.

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TEST YOURSELF

(1) In what way are the difficulties and controversies with regards to determining EEG abnormal periodic discharges harmful?

A. Periodicity is a surrogate marker for severe cerebral injury and does not cause further brain injury.
B. Periodic discharges and seizures in the acutely injured brain lead to secondary neuronal injury via excessive metabolic demand and excitotoxicity.
C. Different clinical settings are associated with a different likelihood of neuronal injury resulting from different types of periodic discharge.
D. All of the above.
E. None of the above.

(2) What can be considered as NSCE?

A. Epileptic encephalopathies.
B. Complex partial status epilepticus.
C. *De novo* late-onset absence status epilepticus.
D. All of the above.
E. None of the above.

Note: Reading the manuscript provides an answer to all questions. You can check for the correct answer by visiting the Educational Centre section of www.epilepticdisorders.com