Making sense of non-epileptic seizures

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ABSTRACT – *Background.* Non-epileptic seizures (NES) are a chronic condition that is frequently misdiagnosed. Limited awareness of the condition may contribute to mismanagement and poor outcome. *Methods.* Medline and PsycLit review of clinical and laboratory studies. *Results.* The concept of NES is clarified, common clinical presentations are reviewed and the differential diagnoses considered. A general overview of possible mechanisms and pathological findings is presented. Finally, epidemiological, prognostic, and treatment issues are described. *Conclusions.* Clinicians increased awareness of NES, together with new diagnostic techniques will improve diagnosis and outcome of this condition. Further research is needed into the pathophysiology, etiology and maintenance mechanisms of non-epileptic seizures.

Key words: epilepsy, non-epileptic seizures, pseudoseizures, seizures, psychogenic

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Non-epileptic seizures (NES) are often a distressing and chronic condition, which may be easily misdiagnosed and inappropriately managed. In financial terms alone, the treatment costs of undiagnosed NES may approximate the lifetime costs associated with the treatment of intractable epilepsy. The personal and social costs are more difficult to quantify but may be severe. Yet significant reduction of medical costs and healthcare utilisation rates have been reported to follow after appropriate diagnosis and management of these patients (Walczak *et al.* 1995). Early diagnosis and treatment of NES could thus improve the overall outcome, preventing the condition from becoming chronic, avoiding much distress, and reducing personal and social costs. The purpose of this review is to present a summary of the relevant clinical findings and explanatory models to support the achieving of these aims.

Definition

A non-epileptic seizure (NES), also known as « pseudoseizure » or « psychogenic seizure », may be defined as a sudden, usually disruptive, change in a person's behaviour, perception, thinking, or feeling, which is usually time-limited and resembles, or is mistaken for, an epileptic seizure. A NES does not have the characteristic electrophysiological changes in the brain, detectable by EEG, that accompany true epileptic seizures (Betts 1998) and it has a presumed psychological aetiology.

Epidemiology

Estimates of the incidence and prevalence of NES are difficult to obtain, firstly because of problems with accurate diagnosis and secondly due to the variability of prevalence according to

which population is studied. More patients with NES have been diagnosed in recent years, which coincides with the introduction of prolonged ambulatory monitoring and video-telemetry for the assessment of patients with epilepsy.

Sigurdardottir and Olafsson (1998) published a study on the incidence of NES in an unselected, community-based population in Iceland. They included all patients older than 15 years of age and not known as previously suffering from NES, referred to the only neurophysiology laboratory in the country, over a period of four years. Each patient underwent long-term video-EEG monitoring. The authors concluded that the average annual incidence of NES was 1.4 per 100,000 person-years of observation (for epilepsy, equivalent figures were 35 per 100,000 person-years of observation); with an age-specific incidence that was higher between the ages of 15 and 24 years and with a majority of female patients (75%).

In selected populations of people attending specialist epilepsy clinics for management of what is considered to be refractory epilepsy, it has been estimated that up to 20% will have NES (Trimble 1986, Gates et al. 1985).

A factor that further complicates both the diagnosis of NES and calculations of prevalence is that a present or past history of epilepsy is found in a significant number of patients with NES referred to hospital for investigation. Estimates of the prevalence of comorbidity for epilepsy and NES vary between studies from 12% to 65% (Betts and Boden 1992, Fenton 1986, Lesser 1985).

Howell et al. (1989) suggested that approximately 50% of patients admitted to emergency care in status epilepticus do not have epilepsy. The inappropriate diagnosis and management of what has been called « pseudostatus » as status epilepticus exposes these patients to unnecessary risks (Gunatilake et al. 1997) including acute anticonvulsant administration and possibly assisted ventilation in a medical intensive care unit.

Diagnosis of NES

Phenomenology

There are no pathognomonic signs or symptoms of NES. The presentation of the NES can vary from collapse with apparent loss of consciousness with or without convulsive behaviour, to attacks without apparent loss of consciousness in which some kind of motor, emotional or cognitive experience occurs, such as a sudden change in emotional state, a disturbance of the sense of internal awareness or intense anger (Betts 1998, Wyllie et al. 1999).

Betts and Boden (1992) classified some of the most common attacks observed in patients with NES as « swoons », « tantrums » and « abreactive » types. In « swoon » attacks, the patient falls to the ground in a relaxed way, followed by lying still without convulsion, with eyes closed and apparently unconscious, usually followed by

rapid recovery. In the « tantrum » type, the patient emits a cry, falls, thrashes about, kicks, and may bites, and is often noisy, shouting, roaring or crying. The « abreactive » attack usually occurs at night, but can also occur during the daytime, in which case there is falling associated with the attack. There is stiffening of the body followed by thrashing, with breath-holding, gasping, moaning, back-arching and pelvic thrusting. As discussed below, NES that present with pelvic thrusting movements, reminiscent of movements made during sexual intercourse, have to be carefully distinguished from the frontal lobe seizures and Geyer et al. (2000), using EEG video-telemetry, noted that in 24% of those with frontal lobe epilepsy, pelvic thrusting was observed.

Meierkord et al. (1991) divided their patients with NES into two broad categories, attacks of collapse with limpness and attacks with prominent motor activity including pseudoconvulsions and thrashing (two thirds of the patients). The authors emphasized that differentiation of the type of seizure is more difficult when there is excessive motor activity.

Groppel et al. (2000) analysed the clinical semiology of psychogenic NES by cluster analysis. They identified three symptom clusters. Cluster 1, « psychogenic motor seizures », was characterized by clonic and hypermotor movements of the upper and/or lower extremities, pelvic thrusting, head movements and tonic posturing of the head; cluster 2, « psychogenic minor motor or trembling seizures », comprised trembling of the upper and/or lower extremities; and cluster 3, « psychogenic atonic seizures », consisted of falling as the only symptom.

Diagnostic delay

In general, the diagnosis of NES has been one of exclusion. This approach tends to lead to long delays between the initial development of symptoms and final diagnosis. Where possible, an active diagnostic process, based on recognition of particular clinical and historical features, is preferable.

Reuber et al. (2002) found that NES were diagnosed with a mean delay of 7.2 years. The authors suggest that in order to shorten the delay, any patient with atypical seizures should be investigated at an early stage, using different methods including home video and seizure provocation under video-EEG monitoring. This, together with a more critical approach to reviewing patients with a diagnosis of epilepsy who fail to respond to anticonvulsants or who apparently develop status epilepticus would lead to the avoidance of trials with unnecessary anticonvulsants.

Differential diagnosis

The main clinical entities to consider when making a differential diagnosis are epilepsy and, less commonly, other organic conditions such as hypoglycaemia, hyperthyroidism and the pheocromocytomas, and functional

conditions such as panic attacks, intermittent explosive disorder and malingering. This review will focus on distinguishing NES from epilepsy, which is the most common diagnostic dilemma in this area.

Despite numerous studies, the differentiation of epileptic seizures from NES remains difficult. Every patient who presents with seizures needs a careful history. Patients may not be able to give much information about their seizures, hence collateral accounts should also be obtained, aimed at establishing the phenomenology of the seizure and the post-ictal period, as well as whether there are any specific precipitating factors for individual seizures. Trimble (1986) emphasises the importance of obtaining a detailed account of the very first seizure, including the settings and timing of it.

There may be positive features in the personal history, recognised to predispose to the development of psychopathology, which, if present, can point towards a diagnosis of NES. Such features include a childhood history of emotional trauma and abuse (Reilly *et al.* 1999) and specifically, childhood sexual abuse (Betts and Boden 1992). In a group of 34 children (mean age 14) with NES, 24% had some form of anxiety disorder and 32% had been sexually abused (Wyllie *et al.* 1999). It should be noted however, that many more people suffer an abusive childhood than go on to develop NES and moreover, some patients with epilepsy have also suffered abuse. A good psychiatric history is therefore important. Within the patients' background, Roy (1979) noted that family psychiatric history, past history of psychiatric disorders, attempted suicide, sexual maladjustment, and current affective syndrome helped differentiate NES from epileptic seizures.

The age and sex distribution of NES in comparison with epilepsy, shows an older female predominance with a female-male ratio observed from 2; 1 (Liske *et al.* 1964, Gower *et al.* 1981, Gates *et al.* 1985) to 15:1 (Scott 1982) and an average female age of 37 years (Gates *et al.* 1985, Liske *et al.* 1964).

With respect to the physical nature of seizures, there is no clinical phenomenon that occurs solely and exclusively in epilepsy. It has been stated that everything that happens in epilepsy can happen in NES and *vice versa* (Wyler *et al.* 1993). It used to be considered that patients who injured themselves were less likely to suffer from NES, but several studies show that this is not the case (Gates *et al.* 1985, Betts 1998). Tongue biting and urinary incontinence are also not reliable indicators of epileptic seizures (Wilkus *et al.* 1984, Lesser 1985, Meierkord *et al.* 1991). In relation to onset and duration, NES usually comes on gradually, with fluctuating course and often lasting from 1-30 minutes. NES do not occur during sleep but have been observed during apparent sleep (Thaker *et al.* 1993, Devinsky *et al.* 1996). Complex partial seizures of frontal lobe origin, which is the type of seizure most difficult to differentiate from NES, are usually short (less than 1 minute) with a

rapid onset and cessation (Meierkord *et al.* 1991); moreover they often occur during sleep (Saygi *et al.* 1992, Groppel *et al.* 2000). In the past, frontal lobe epilepsy was at times misdiagnosed as NES, in part because these seizures may not be detected on routine scalp EEG and in part because they may be associated with apparent bizarre behaviours. Spencer *et al.* (1983) reported several cases of patients with probable frontal lobe seizures who manifested sexual automatisms including masturbatory or other sexual activity for which the patients were subsequently amnesic. Geier *et al.* (1976) also reported complex movements, dancing, mimicking fear, crying or laughing during frontal lobe seizures.

There are a small number of relatively reliable clinical observations, which may distinguish an epileptic seizure from an NES. Fenton (1986) points out that the pupillary reaction to light and the corneal reflexes are retained during an NES but not during a generalized epileptic seizure. Moreover, unlike epileptic seizures, NES do not occur during sleep, although EEG monitoring may be necessary to establish whether or not a patient had awoken prior to seizure onset. The tenor of the motor attack may be a useful pointer to its origin. The tonic-clonic sequence and the evolution of the clonic jerks from fast, small-amplitude to slower, large-amplitude movements and the rapid contraction and slow relaxation seen in tonic-clonic seizures are not usually observed with NES (Meierkord *et al.* 1991).

Regarding the EEG findings, the diagnosis of epilepsy can be difficult as only 30-40% of patients with epilepsy show epileptiform discharges on a single, interictal, awake recording (Ajmore and Zivin 1970). Moreover, EEG abnormalities have been observed in up to 2.4% of healthy individuals (Trojaborg 1992). Artifactual activity resulting from marked motor activity during the NES can obscure the EEG almost as much as a *grand mal* convulsion (Rodin 1984). Sometimes it is only by careful EEG analysis and video-telemetry that the nature of the artifactual origin of the waveforms can be clarified (Burnstine *et al.* 1991).

About half of those patients with epilepsy and an unhelpful awake recording will show definite epileptiform activity during a sleep recording (Gastaut *et al.* 1991). However, whilst ictal recordings are much more reliable for distinguishing between epilepsy and NES, they are difficult to obtain and, even in these circumstances, occasionally the standard EEG 10-20 scalp electrode placement fails to reveal epileptiform activity that remains restricted to deep temporal or frontal foci.

A diagnosis of NES is suggested by a lack of EEG changes during the seizures. The diagnosis is further supported by: (a) the presence of a waking alpha rhythm during what appears to be a clinical alteration in consciousness, (b) the non-paroxysmal or nonstereotypical nature of the seizure behavior, (c) a normal background EEG in apparently different physiological states during an entire day of continuous recording (Shen *et al.* 1990).

The use of ambulatory EEG monitoring, which can take place in the patient's home where most seizures probably occur, or the use of EEG-video-telemetry in a specialist unit, with continuous recording maintained for several days while the patient is under observation, have become more readily available and represent the gold standard in distinguishing NES from epileptic seizures.

In relation to neurohormonal changes, the initial research into the increase in prolactin levels after seizures (Trimble 1978) suggested that this could be helpful in distinguishing epileptic seizures from NES. However, more recent studies have suggested that this approach is only of limited use in clarifying the diagnosis. Alving (1998) reported not only a statistically significant rise in post-ictal prolactin levels in both epilepsy and NES groups, but also a significant difference between the groups. Furthermore, in frontal, sensorimotor and simple partial seizures, which are often the seizures that need to be distinguished most carefully from NES, prolactin levels may not be significantly elevated.

Some clinicians have employed more indirect means in order to try to differentiate patients with NES from patients with epilepsy. Methods have included hypnosis, used to recover lost memories of the ictal stage with the premise that if ictal memory can be recovered then the attack is an NES (Betts 1998). Barry et al. (2000) noted that patients with NES had a greater degree of hypnotizability than those with epilepsy and suggest that this, together with hypnotic seizure induction, may also be a relatively sensitive means of diagnosing NES. Zaidi et al. (1999) proposed head-up tilt as a safe, simple and inexpensive outpatient technique for investigating NES. Shen et al. (1990) proposed outpatient video/EEG monitoring, for use particularly when NES are suspected. This monitoring is performed for an entire day with a relative or friend present to identify the habitual « seizures ». If the usual fits do not occur, activation procedures (suggestion, hyperventilation, photic stimulation, intravenous saline injection) are used.

Non-epileptic seizures and epileptic surgery

The co-occurrence of NES with epilepsy and the observations that some patients with epilepsy may develop NES for the first time after epilepsy surgery (Krahn 1995) are both factors that indicate that the risk of current or future NES should be considered as part of the surgical management of epilepsy.

Reuber et al. (2002) emphasize the need for careful assessment of patients who suffered from epilepsy before surgery, and point out that a diagnosis of additional NES in patients who have clearly demonstrable, surgically amenable seizures, adds a further dimension to the evaluation of the patient, but should not be considered an exclusion criterion. The authors also place emphasis on the evaluation of underlying psychological disorders that may be being expressed as NES. They compared the good outcome of a group of 13 patients they followed up after surgery in comparison with another group of patients who had poor outcome with conservative treatment (Henry et al. 1997).

Abnormalities of function associated with non-epileptic seizures

Electrophysiology

The interictal findings of patients with NES are not always normal; minor sharp transients of localised (mainly temporal) or diffuse character are found (Lelliott and Fenwick 1991). Intermittent photic stimulation sometimes elicits mild paroxysmal bursts. Gastaut (1949) found a low « myoclonic threshold » to combined photic and pentylenetetrazol (metrazol) activation in these individuals. Indeed, these findings have been taken to support the idea that organic brain disease may facilitate the occurrence of NES (Fenton 1986, Lelliott and Fenwick 1991).

Drake et al. (1993) reported auditory event-related potential differences between patients with NES and with epilepsy, finding significantly longer P3 (P3b component) latencies in those with epilepsy. P3b reflects a process of memory updating by which the current model of the environment is modified as a function of incoming information (Coles and Rugg 1995). A prolonged latency suggests a delay in evaluating environmental stimuli in those with epilepsy but not in those with NES.

Pouretemad et al. (1998) looked into sensorimotor gating in a group of 21 patients with NES and 22 healthy controls. The startle response is usually inhibited when a weak to moderate stimulus is presented at a brief interval before the startle-eliciting stimulus. The degree to which the startle response is inhibited by this prepulse is called prepulse inhibition (PPI), indicating the amount of sensorimotor gating. The results of Pouretemad et al. (1998) showed a significant reduction in PPI in unmedicated patients with NES, suggesting that patients with NES, at times of stress, may become overloaded with stimuli, overwhelming their coping abilities.

Psychological tests

Although neither the Minnesota multi-phasic personality inventory (MMPI) nor its revised version (MMPI-2) can establish a diagnosis of NES, some authors have considered these questionnaires to be clinically useful adjuncts to diagnosis. Wilkus et al. (1984), using the MMPI, found that patients with NES exhibited significantly higher scores on hysteria, hypochondriasis, and schizophrenia ratings than patients with epilepsy; while Derry and Mclachlan (1996), using the MMPI-2, reported that patients with NES show a personality profile suggestive of conversion or somatoform disorder. However, it has been concluded (Kalogjera-Sackellares and Sackellares 1997) that al-

though patients with NES show a range of elevated clinical scales on the MMPI, that the nature of the psychological profiles derived is complex and not amenable to explanation using a single psychological mechanism. More recently, it has been reported that combining MMPI-2 findings with the results of a routine EEG and with the duration of the condition, generated a model with an accuracy in predicting a correct diagnosis of NES of 86% (Storzbach *et al.* 2000). It is possible that this combined approach may, in the future, find a place in specialist epilepsy centers.

Neuroimaging investigations

The vast majority of imaging studies are normal. No consistent brain abnormalities have been found in patients with NES, using either structural CT, functional SPECT blood flow imaging or MRI scans (Lelliott and Fenwick 1991, Varma *et al.* 1996, Reuber *et al.* 2002). In Lelliot and Fenwick's (1991) sample, 18% of the brain scans were abnormal, among those were two cases with NES and five with concurrent epilepsy. Varma *et al.* (1996) studied interictal blood flow single photon emission tomography (SPECT) in a group of 10 patients with NES and 10 patients with complex partial seizures and localization-related epilepsy. Seven patients with NES (70%) had normal SPECT scans and three had either clear or equivocal local hypoperfusions. In the epilepsy group however, eight (80%) had significant focal hypoperfusion and two had equivocal focal hypoperfusion. In Reuber *et al.*'s (2002) magnetic resonance imaging study, (using a 1.5-T scanner), 27% of patients with NES and 77.9% of patients with epilepsy and NES, had abnormal MRI scans. Some of the abnormalities reported in the NES group were postoperative defects (6 cases), arachnoid cyst (3), postraumatic changes (2), generalised atrophy (2) among others. The most common abnormalities found in the epilepsy with NES group included hippocampal sclerosis (28 cases), postoperative defects (13), migration disorders (9), signs of previous stroke (5) and gliosis (4).

In summary, most patients with NES alone will not show abnormalities, unlike patients with epilepsy or concurrent epilepsy and NES. Although in some cases there is evidence of physical abnormality detected in patients with NES, none of the abnormalities found are specific to NES.

Explanatory model for non-epileptic seizures

Although neuropsychological testing, EEG recording, and brain imaging studies have not clarified the basis of NES, consideration of psychiatric diagnoses that may be made in patients with this condition suggests possible underlying psychological mechanisms that may contribute to the symptoms observed. Alper *et al.* (1995), in a sample of 92 subjects with a diagnosis of NES, found that 71 patients had conversion disorder, and the remaining 21 had anxi-

ety or psychotic disorders or impaired impulse control. Moore and Baker (1997) presented psychological characteristics of 185 patients with a diagnosis of NES. They identified the following factors as being important for the development and maintenance of NES: anxiety or stress, physical and sexual abuse, significant bereavement, relationship problems and depression. An absence of relevant psychological factors was found in only 5% of patients. Other studies (Lempert and Schmidt 1990, Roy 1979) have reported a high incidence of depressive symptoms in patients with NES, and a background of personality disorder has been reported by Gummit and Gates (1986). In a series of 18 patients with pseudostatus, Rechlin *et al.* (1997) reported that five of the patients were suffering a concomitant major depressive episode and that ten of the subjects met diagnostic criteria for borderline personality disorder.

Harden (1997) has suggested that a common mechanism involving earlier, emotionally traumatic experiences may underlie both pseudoseizures and other dissociative disorders. The following explanatory models have been proposed.

Conversion disorder – a psychoanalytic model

According to psychoanalytical theories, the repression of unconscious, intrapsychic conflict and the conversion of anxiety into a physical symptom cause conversion disorders (CD). The CD symptom has a symbolic relation to the unconscious conflict. In this context it has been noted that NES are more common in people who have suffered abuse in childhood (Betts 1998, Betts and Bowden 1992, Reilly *et al.* 1999). The mechanisms by which abuse leads to NES are not clear. The victim may retain ossified behavior patterns appropriate to the early age at which the trauma occurred rather than to their current adult status.

Persistent behavioural and emotional sequelae of abuse have the characteristics of post-traumatic stress disorder, and individual NES attacks may be provoked by stimuli that lead to re-experiencing of the traumatic event or they may be an extreme response to emotional recollection of the trauma. Indeed, Rosenberg *et al.* (2000) have noted that out of their sample of 35 patients with intractable epilepsy, the presence of a diagnosis of NES rather than epilepsy was correlated with a history of PTSD and the total number of life-time traumas.

Conversion disorder – a biological model

Increasing data implicate biological and neuropsychological factors in the development of CD symptoms. Preliminary brain-imaging studies have found hypoperfusion of the dominant hemisphere and hyperperfusion of the nondominant hemisphere and have implicated impaired hemispheric communication in the cause of CD (Yazici and Kostakoglu 1998). The suggestion that symptoms may be caused by an excessive cortical arousal that sets off

negative feedback loops between the cerebral cortex and the brainstem reticular formation is in agreement with Ludwig (1972) and Whitlock (1967), who considered CD as primarily a disorder of attention and vigilance due to a « selective depression of awareness of a bodily function » brought on by corticofugal inhibition of affected stimulation at the level of the brainstem reticular formation.

In conclusion, the models used to explain NES, conversion disorders, and PTSD are not mutually exclusive. However, the manner by which these processes are converted into seizure-like activity has not been established.

Treatment

After the diagnosis of NES has been made, the first step in its management is the presentation of the diagnosis to the patient. The main purpose of this process is to convey the non-epileptic nature of the seizures to the patient without alienating them in the process (Shen *et al.* 1990). Following this, withdrawal of anticonvulsant medication should always be slow and under expert supervision. This will prevent the occurrence of withdrawal seizures, which could complicate the diagnosis, as well as helping to decrease any resistance from patients (and their families) who may be psychologically dependent. The next step is directing the patient to psychiatric care, without totally withdrawing care by the neurologist (Shen *et al.* 1990). Psychiatric comorbidity is relatively common among patients with NES, as discussed above, but when the use of medication to manage these patients is indicated, it should be accompanied by psychological support.

Cognitive-behavioural techniques are widely used in the management of NES. The general principle is to prevent the reinforcement of non-epileptic seizure activity by reducing arousal and negative thinking in order to abort seizure progression (Betts 1998). In addition, family therapy can be very important since even if family dynamics are not the prime cause, family anxiety often reinforces seizure behaviour. Some reports suggest a reasonable response to psychotherapy in patients with recent-onset NES (Buchanan and Snars 1993), although not all studies report such a favourable outcome. For those patients who disclose previous sexual abuse, Betts and Boden (1992) have proposed a four-stage therapeutic process, which entails at least 2 years of outpatient work.

Whatever treatment approach is used, consideration should be given to psychosocial factors that may be acting to reinforce the NES activity. Similarly, unnecessary medical interventions should be avoided.

Course and outcome

Information regarding outcome in these patients is limited. A 5-year follow-up of patients with NES has revealed that the major disability in 80% was related more to the psychosocial consequences of the NES than to the attacks themselves (Krumholz and Niedermayer 1983). The value of psychotherapy is unclear. Jogsman *et al.* (1999) observed that more of those who received this treatment improved whilst Walczak *et al.* (1995) concluded that there was a lack of correlation between psychotherapy and remission of NES. They noted that persisting NES were associated with longer duration of NES before diagnosis (p < 0.02) and with the presence of additional psychiatric disorders (p < 0.01). Patients with good outcome were more likely to have experienced an acute emotional trauma preceding onset of NES. McDade and Brown (1992) reported two further factors strongly associated with poor outcome: low IQ and a history of violent behaviour.

Kanner *et al.* (1999) reported that the persistence of NES six months after disclosure of the diagnosis was associated with a history of chronic abuse (sexual and physical), personality disorders, recurrence of major depressive illness, dissociative and somatoform disorders, as well as denial of any stressors or psychosocial problems. The majority of these patients initially responded to various psychological or pharmacological interventions but at follow-up 2 years later, seizures had returned in most patients.

In children with NES, the best response is associated with early diagnosis and prompt psychiatric treatment (Wyllie *et al.* 1999).

Conclusion

NES remain a poorly understood clinical entity. Accurate diagnosis is delayed in a substantial proportion of patients and this delay may be associated with a poorer long-term outcome. Increased clinician awareness, together with prolonged ambulatory monitoring and new methods of investigation, will facilitate the correct diagnosis and improve the outcome of this condition. With respect to management, there is no single and effective treatment strategy. Appropriate and sensitive initial presentation of the diagnosis to the patient and their family is, however, of critical importance. □

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