Diagnosis and management of dissociative seizures

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Up to one in five people diagnosed with epilepsy will turn out to have dissociative seizures (DS) – psychologically mediated episodes of altered awareness and/or behaviour that may mimic any type of epilepsy[1,2]. These patients are typically treated with antiepileptic medication for a number of years before the correct diagnosis is made. During this time they are exposed to significant iatrogenic risks including drug toxicity, teratogenic risk (most patients are young women) and the risk, in approximately 10%, of receiving emergency treatment for 'status'[3,4]. By the time the correct diagnosis is made many patients and their families have already adapted their lives to chronic disability. For some, a medical 'sick role' seems preferable to a psychiatric one from the start. For the majority, however, years of inappropriate medical interventions will have reinforced the patients' view of themselves as medically disabled. The one factor consistently associated with a better prognosis in this and other functional disorders is a short duration of illness at the time of diagnosis: in other words, prompt diagnosis[5]. How to recognise and treat DS is therefore an important subject for all clinicians working in the field of epilepsy.

Definitions and terminology

A review in 1997 found no less than 15 synonyms for this disorder[6]. Some terms (pseudoseizures, hysterical fits) are clearly pejorative and have been abandoned. Others (non-epileptic seizures, non-epileptic events, non-epileptic attack disorder) define the condition by what it is not and may well be interpreted by the patient as suggesting that 'the doctor doesn't know what's wrong with me'[7]. Furthermore, some of these terms are ambiguous. Non-epileptic seizures (NES), for example, is used by some to describe conditions, both medical and psychiatric, that may be mistaken for epilepsy, while on other occasions NES is used as a form of shorthand for the psychogenic attacks alone. The debate about terminology is likely to continue, but in the meantime ICD 10[8] does in fact provide a perfectly acceptable and useful label – dissociative convulsions. In recognition of the fact that many patients with this disorder do not actually suffer a 'convulsion', the term *dissociative seizures* is probably better.

Psychiatric disorders that may be mistaken for epilepsy

A list of the medical and psychiatric disorders that may be mistaken for epilepsy is given in Table 1. The clinical features distinguishing epilepsy from paroxysmal cardiological, neurological and other medical disorders are reviewed elsewhere in this section[9,10]. Syncope is probably the most frequent missed diagnosis in non-specialist settings but by the time patients are referred to specialist epilepsy clinics DS is by far the most important differential diagnosis[1]. Indeed, the possibility of DS should be one of the first considerations in a patient with medically intractable seizures.

Apart from DS a number of psychiatric disorders may occasionally be mistaken for epilepsy and vice versa. The most important example is panic disorder which may be confused with

Table 1. The differential diagnosis of epilepsy.

A. Medical causes of paroxysmal neurological dysfunction

1. Syncope
- vasovagal
- cardiogenic

2. Neurological
- cerebrovascular
- migraine
- vertigo
- cataplexy
- parasomnias
- movement disorders
- startle-induced phenomena

3. Endocrine and metabolic
- hypoglycaemia
- hypocalcaemia
- hereditary fructose intolerance
- pheochromocytoma
- drugs and alcohol

B. Psychiatric disorders

1. Dissociative seizures

2. Psychiatric disorders that may be mistaken for epilepsy
- panic disorder
- psychosis
- attention deficit hyperactivity disorder
- depersonalisation disorder

3. Factitious disorder

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partial seizures that feature anxiety as part of the aura[11,12]. The cognitive symptoms of panic disorder (specific feared consequences of the attack, such as a fear of choking, having a heart attack, dying, losing control, etc), the presence of environmental precipitants (crowded places, queues in supermarkets, etc) and the avoidance of such situations (agoraphobia) help identify panic. The often unique subjective quality of 'ictal fear', abrupt onset without environmental triggers and the presence of other epileptic semiology are useful in recognising the epileptic origin of such symptoms in partial seizures. Very rarely, paroxysmal symptoms in psychosis (hallucinations, thought block) may raise the possibility of epilepsy, and attentional problems in a child may raise the differential diagnosis of attention deficit hyperactivity disorder and petit mal seizures. An uncomfortable sense of unreality concerning one's self (depersonalisation) or the environment (derealisation) is not uncommon in temporal lobe seizures. These symptoms may be the primary complaint in depersonalisation disorder and are a non-specific feature of affective disorder and psychosis[13]. In psychiatric disorder these phenomena are usually of relatively gradual onset, prolonged duration and accompanied by other psychiatric symptoms. Overall, the abrupt onset, brief duration and highly stereotyped nature of epileptic symptoms help distinguish them from functional psychiatric disorder.

Factitious disorder and dissociative seizures: the concept of unconscious symptom generation

Factitious disorder (Munchausen's syndrome) refers to the situation in which a patient is discovered to be (or admits) deliberately feigning symptoms. The most important feature, however, and this is critical for the diagnosis, is that in factitious disorder the patient's motivation is held to be psychological (understandable in terms of the patient's psychological background, personality, dependency needs, etc). By contrast *malingering* (not a medical diagnosis) involves fraudulently imitating illness to achieve some obvious practical advantage (e.g. compensation, to avoid a criminal conviction, to obtain social security benefits).

By definition, DS are regarded as being involuntary or unconscious. By consensus, the majority of patients with such seizures are believed to meet this criterion. For some, however, the fact that experienced clinicians judge this to be the case is not persuasive. For sceptics, there are three objective features of DS that are worth considering: 1) the majority of patients are compliant with their antiepileptic drugs (AEDs), often for many years and to the point of toxicity[4,14]; 2) when patients are admitted for telemetry the majority have a seizure in a setting which they must surely recognise involves intensive monitoring; 3) the seizure is usually a poor imitation of epilepsy. None of these points is by any means conclusive but if deception is involved, it is of a kind that is difficult to understand.

While psychiatric classification systems assume a dichotomy between conscious and unconscious symptom generation (implying factitious or dissociative seizures respectively) the two are best regarded as opposite ends of a continuum. The concept of *self deception,* something which at a trivial level most people can relate to, lies somewhere in the middle and provides a useful paradigm for understanding how subjective experience, and even complex behaviour, is prone to influences that are not always fully conscious, even in healthy individuals.

Clinical features of dissociative seizures

Prevalence

From prevalence figures for epilepsy and estimates of the proportion of patients referred to tertiary clinics who have DS, Benbadis and Allen[2] calculated the prevalence of DS to be between two and 33 per 100,000. However, the true prevalence may be far greater. These authors based their calculation on the assumption that most patients with DS would find their way to specialist clinics because their seizures would persist despite AED treatment in primary care. However, it remains entirely possible that some patients with DS have a (placebo) response to their first AED prescription and are never referred on for specialist advice. This possibility is borne out by a recent population-based study that found DS in a fifth of patients with new-onset seizures, the same proportion of DS reported in specialist services[15].

Demographic characteristics

Some 75% of patients are female[3,14,16,17]. Seizures typically begin in the late teens or early 20s, although there is a wide range[3,14,16]. A UK study found a median delay between seizure onset and diagnosis of three years[3], but even longer delays have been reported by others[18,19]. Patients with lower educational achievement and of lower socioeconomic groups are probably overrepresented, although not in comparison with epilepsy.

Clinical assessment

No single semiological feature distinguishes DS from epileptic seizures or vice versa. The most helpful features, as well as some important pitfalls – symptoms that are commonly

mistaken as evidence for epilepsy – are listed in Table 2. Epileptic seizures are brief, highly stereotyped, paroxysmal alterations in neurological function that conform to a number of now well-described syndromes. Broadly speaking, it is any variation from this clinical picture – an *atypical sequence of events* – that will raise the suspicion of epilepsy. Despite 30 years of videotelemetry there is no reliable shortcut to making the diagnosis: to recognise DS the clinician must have experience with epilepsy. Some features worth highlighting are the long duration of DS, their tendency to begin gradually, and to show a waxing and waning of motor activity followed by an abrupt recovery, asynchronous movements (including side-to-side head or body movements), eye closure, ictal crying and preserved recall after a period of unresponsiveness[20]. An episode of motionless unresponsiveness[77] lasting over five minutes is unlikely to have an organic cause[3]. Patients with DS commonly report injuries. Friction burns may be characteristic of DS. Bite injuries are reported in DS, especially to the tip of the tongue and lip[21], but severe scarring is extremely rare. Seizures during sleep are reported just as frequently in DS (around 50%) as in epilepsy[65].

Table 2. Comparative semiology of dissociative epileptic seizures.

	Dissociative seizures	*Epileptic seizures*
Duration over two minutes	common	rare
Recall for a period of unresponsiveness	common	very rare
Motor features		
Gradual onset	common	rare
Eyes closed	common	rare
Thrashing, violent movements	common	rare
Side-to-side head movement	common	rare
Pelvic thrusting	occasional	rare
Opisthotonus, 'arc de cercle'	occasional	very rare
Fluctuating course	common	very rare
Automatisms	rare	common
Weeping	occasional	very rare
[a]Incontinence	occasional	common
[a]Injury		
Biting inside of mouth	occasional	common
Severe tongue biting	very rare	common
[a]Stereotyped attacks	common	very rare

[a]Three features that are commonly misinterpreted as evidence for epilepsy have been included. Otherwise the table lists clinical features that are useful in distinguishing DS from epileptic seizures. Figures for frequency of these features are approximate: common >30%; occasional 10–30%; rare <10%; very rare <5%. (Adapted from Mellers[20])

Studies of the semiology of DS have focused on motor phenomena and the features of epilepsy lacking in DS. Little attention has been paid to subjective symptoms that might be regarded as the psychiatric phenomenology of DS[78,79]. Patients with DS commonly report a feeling of being cut off at the onset of their seizure and describe a number of symptoms of autonomic arousal. These include tachycardia, perspiration, hyperventilation, peripheral paraesthesia, carpopedal spasm and a dry mouth. Patients may not volunteer these symptoms and sometimes a history of hyperventilation will only emerge from an eyewitness account. Such symptoms are reported by approximately 60% of DS patients, compared with around 30% of patients with partial seizures[66].

Other features on history which support (and only that) a diagnosis of DS include an absence of risk factors for epilepsy, a failed response to AEDs and the presence of risk factors for DS (see below). Here again there are pitfalls. Patients with DS commonly report a significant past neurological history[22] as well as a family history of epilepsy[23].

It used to be supposed that the majority of patients with DS also suffered from epilepsy. As studies have become more sophisticated, however, estimates of the prevalence of comorbid epilepsy have become ever smaller. Probably no more than 15 or 20% of patients with DS also have epileptic seizures[3,16,18,24]. A history of multiple (dissociative) seizure types is given by 20% of patients with DS[16,18].

Psychiatric comorbidity
Studies of psychiatric diagnoses in patients with DS have reported a broad range of prevalence figures. High rates of depression, anxiety disorder, personality disorder and post-traumatic disorder have been reported[16]. Often the presence of such a history will raise suspicion of DS, but high rates of psychiatric disorder are also seen in association with epilepsy, at least in those patients with intractable epileptic seizures, and may not help distinguish the two disorders[25-27]. A history of previous medically unexplained symptoms is very common in DS and an important pointer to the diagnosis[16].

Ictal observation/examination
An opportunity to observe a seizure may provide invaluable information. Whether the patient is responsive to verbal requests should be established. Careful note should be taken of the type and distribution of movements and whether apparent clonic movements are rhythmic and synchronous (as they usually are in epilepsy) or not (DS). Following a generalised tonic-clonic seizure the corneal reflex will usually be absent and plantar responses extensor. Pupils will be unresponsive to light in organic states of impaired consciousness. If the patient's eyes are shut the examiner should attempt to open them noting any resistance (DS). A simple test to look for avoidance of a noxious stimulus is to hold the patient's hand over their face and drop it: in DS the patient may be seen to control their arm movement so their hand falls to one side. If the eyes are open, evidence of visual fixation may be sought in two ways. The first involves rolling the patient onto their side. In patients with DS the eyes will often be deviated to the ground. If this is the case, the patient should be rolled onto the other side to see if the eyes are still directed towards the ground (the 'Henry and Woodruff sign')[31]. A second useful manoeuvre is to hold a small mirror in front of the patient and look for evidence of convergent gaze and fixation on the reflection. This procedure will often stop the seizure. Patients with factitious disorder may learn to produce the 'correct' response in all of these examination procedures.

Investigations

EEG
Unfortunately the EEG still contributes to diagnostic errors in this group of patients. Non-specific EEG abnormalities are found in up to 15% of healthy individuals and all too often

interpreted as supporting a diagnosis of epilepsy. Narrowly defined epileptiform abnormalities are much less common, but still encountered in up to 1% of the healthy population[29,30]. The risk of a 'false positive' EEG is compounded in patients with DS by the fact that both non-specific and epileptiform EEG abnormalities may be more common in patients with DS than in healthy individuals, including those who do *not* have comorbid epilepsy[31]. This is almost certainly because a variety of neurological insults associated with learning difficulties are common in patients with DS and may be associated with EEG abnormalities in the absence of epilepsy. Interestingly, patients with borderline personality disorder (also common in DS) have also been reported to have a high prevalence of non-specific EEG abnormalities[32].

VideoEEG telemetry

VideoEEG (vEEG) telemetry is the gold standard investigation. A good quality video which captures the onset and evolution of the seizure will on its own often allow a confident diagnosis. The diagnostic electrographic findings are: for epilepsy, 1) ictal epileptiform discharges; 2) post-ictal slowing; and in DS, 3) an intact alpha rhythm when the patient is demonstrably unresponsive[3]. Again there are some traps: in particular, movement artefact may obscure or even be mistaken for epileptiform discharges. There are documented cases of patients having their first ever, and possibly only, DS during telemetry, sometimes as an elaboration of a simple partial seizure[33]. This underlines the importance wherever possible of showing the video to an informant to establish that the seizure is representative of the patient's *habitual* attacks. In addition to the cost of vEEG and its restricted availability there are a number of important clinical limitations. The technique is of limited use in a patient who has infrequent seizures. Care must be taken in patients who have multiple seizure types to ensure that an example of each seizure is seen.

Special mention should be made of simple partial seizures and frontal lobe seizures which are often not accompanied by any electrographic changes on the ictal scalp EEG[34,35]. Frontal lobe seizures in particular may have bizarre behavioural features which are now well known to specialists but may easily mistaken for DS[36]. The highly stereotyped nature and very brief duration of the seizures are helpful features on video. If seizures occur in sleep, as they often do in frontal lobe epilepsy, the EEG will be helpful, demonstrating seizure onset during electrographically documented sleep. In DS by contrast (around 50% of patients with DS report seizures arising from sleep)[65] the EEG will reveal that the patient wakes and then has their seizure[37].

A number of studies have demonstrated that placebo methods such as intravenous injection of saline can be used to provoke a seizure in up to 90% of patients with DS[38]. Clearly these studies raise ethical concerns related to the use of placebo. Most recently, however, McGonigal and colleagues have combined simple suggestion with routine photic and hyperventilation stimuli, fully disclosing the aims of the procedure to patients[39]. A total of 60% of patients had a DS provoked in this way compared with 33% in a control group who received identical activation procedures but without suggestion. These authors estimate they were able to reduce the need for prolonged telemetry admission in 47% of patients. Provocation may be of particular value in patients who have infrequent seizures and would otherwise be unsuitable for telemetry. There is a small risk of false positive results with this technique (provoking a DS in a patient with epilepsy) and it is therefore critical that an informant who has witnessed the patient's seizures is available to confirm that the provoked seizure resembles their habitual seizures.

Ambulatory EEG monitoring and video recordings obtained by patients' carers may be very helpful with the accepted and obvious limitations of lacking video correlation in the first and usually failing to capture seizure onset in the second[40].

Serum prolactin
Serum prolactin rises after tonic-clonic epileptic seizures, peaking between 20 and 30 minutes following the seizure[41]. The post-ictal prolactin level should be compared with a baseline measure taken at approximately the same time of day. A prolactin rise is less reliable following complex partial seizures, may be absent following serial epileptic seizures or in status epilepticus, and is not seen following simple partial seizures. False positive rises are now known to occur following syncope[42] and, more significantly, DS[43] and the test is falling out of favour. However, a negative finding after an apparent tonic-clonic seizure may still be very helpful. A recent study has reported higher creatine kinase levels in patients with tonic-clonic status compared with DS status but again there were false positives and negatives[67].

Psychiatric formulation: an aetiological model of DS

By analogy with epileptic seizures, which are a symptom of paroxysmal neurophysiological abnormality that may have many causes, a useful model of DS would attempt to account for the mechanisms underlying individual seizures as well as for background predisposing factors. As yet there is no widely agreed model. However, many putative risk factors for DS have now been reported and studies seeking to clarify the psychiatric phenomenology and the neurophysiology of dissociative states are ongoing.

Dissociation
For practical purposes, dissociation may be defined as a psychologically mediated alteration of awareness and/or control of neurological function. Some have argued for more specific uses of the term[44], but defined in this way dissociation encompasses a spectrum of mental processes including normal phenomena, such as focused or divided attention (e.g. 'domestic deafness', mental absorption), and pathological states involving perceptual, cognitive and motor function. The advantage of such a definition is that, by explicitly assuming (and it is an assumption) that dissociative disorders lie on a continuum with normal experience, it facilitates an empathic understanding of what might otherwise seem unintelligible, if not frankly unbelievable, behaviour. This is equally important for professionals, patients and carers.

The psychophysiological basis for dissociative states is not understood. Many patients with DS describe becoming gradually cut off or distant from their environment and experience symptoms of autonomic arousal during their seizures. This suggests that for some patients, DS may represent a dissociative response to paroxysmal physiological arousal triggered by intense emotion. Some patients may even be aware of 'giving in' to a trance-like state to escape from distressing emotions[80]. Most patients, however, deny emotional symptoms in their attack (DS may be likened to 'panic attacks without panic')[66], the hypothesis being that the triggering emotion is concealed by the dissociative state (for Freud this was the primary gain of hysterical symptoms). However, clinical experience suggests that a proportion of patients who initially deny triggers for their attacks are eventually able to recognise highly specific and emotive cues (for example related to traumatic past experiences). Clearly, this model of dissociative mechanisms gives rise to a number of testable hypotheses which require further research.

Predisposing, precipitating and maintaining factors
Studies of psychosocial correlates of DS have revealed a number of potential predisposing, precipitating and maintaining factors which are summarised in Table 3. Adverse or traumatic experiences, particularly in childhood, are a common underlying theme. Sexual, physical and emotional abuse are well replicated associations[45-47,68] but other traumatic experiences or situations that foster enduring low self-esteem, for example being bullied at school or unrecognised learning difficulties, may be over-represented[48]. The high prevalence of

Table 3. Predisposing, precipitating and maintaining factors in dissociative seizures.

	Psychological	*Social*
Predisposing	Perception of childhood experience as adverse	Adverse (abusive) experiences in childhood
	Somatising trait	Poor family functioning
	Dissociative trait	Traumatic experiences in adulthood
	Avoidant coping style	
	Personality disorder	Modelling of attacks on others with epilepsy
	Mood disorder	
Precipitating	Perception of life events as negative/unexpected	Adverse life events
	Acute panic attack/syncope	
Maintaining	Perception of symptoms as being outwith personal control/due to disease	Angry/confused/anxious reaction of carers
	Agoraphobia: avoidant and safety behaviour	Fear of responsibilities of being well/benefits of being ill
	Angry/confused/anxious reaction to diagnosis	

There is no evidence at present for biological factors which are therefore not listed in the table. However, there may be genetic influences on relevant personality attributes, coping styles and traits. (Adapted from Binzer et al[45])

abnormal personality in DS[49,50] may be an effect of adverse experiences at a stage of development when personality attributes are formed. None of these features, however, is unique to patients with DS; they are seen in patients with other psychiatric disorders, including somatoform presentations other than DS[47,51]. Why some children exposed to grossly abnormal experiences develop psychiatric disorder later in life but others do not, and what determines the form the illness takes, is not understood. Further studies of coping styles, putative dissociative and somatising traits, and how these are related to childhood traumatic experiences will help tease apart the undoubtedly complex individual/environmental interactions involved.

One study has presented evidence of adverse events in the year prior to onset of DS which might be regarded as precipitating factors for the disorder[45]. Once the disorder is established a number of maintaining factors may operate. Agoraphobic avoidance is more common in patients with DS than in epilepsy and serves to heighten anxiety about having seizures which in turn makes seizures more likely[66]. Anxiety about the seizures will also be fuelled by conflicting diagnoses and advice received from the numerous contacts patients have with doctors, paramedics, accident and emergency staff as well as friends, support groups and the internet. Finally, for some individuals at least, the benefits of the sick role may provide an

acceptable alternative to the responsibilities of healthy life[52], and carers, unwittingly or otherwise, may play an important role in perpetuating disability. The stigma attached to mental illness undoubtedly has an important role in shaping the medical presentation of somatoform disorders and contributes to the reluctance many patients have in accepting psychiatric treatment.

Management

An approach to discussing the diagnosis with patients

The way in which the diagnosis of DS is presented to the patient is possibly the single most important factor determining outcome (Table 4). A clear explanation of the reasons for concluding the patient does not have epilepsy should cover both clinical features and investigation findings. It is important that patients are not left with the impression that investigations alone hold the key to diagnosis; a quest for further tests might otherwise ensue. Once the patient understands that epilepsy and other 'medical' causes have been excluded they will often be extremely sensitive about being accused of putting on their attacks. The clinician should put aside any prejudices they may have in this respect, suspend disbelief if necessary, and reassure the patient that their attacks are real, disabling and involuntary.

Next, an intelligible explanation of what the patient does have is required. The concept of dissociation can be explained as involuntary episodes of 'switching off' or going into a 'trance'. Examples of selective attention (mental absorption – not hearing one's name called when reading) and divided attention (travelling home from work and remembering nothing of the journey) can be used to illustrate the involuntary, unconscious nature of dissociative phenomena and how we can all be unaware, or have no memory of, sensory experience or complex activities despite perfectly normal neurological function.

Table 4. Presenting the diagnosis of dissociative seizures.

The discussion should cover:

1. *Explanation* of the diagnosis
    - Reasons for concluding they don't have epilepsy
    - What they do have (describe dissociation)

2. *Reassurance*
    - They are *not* suspected of 'putting on' the attacks
    - The disorder is very common

3. *Causes* of the disorder
    - Triggering 'stresses' may not be immediately apparent
    - Relevance of aetiological factors in their case
    - Maintaining factors

4. *Treatment*
    - DS may improve simply following correct diagnosis
    - Caution that AED withdrawal should be gradual
    - Describe psychological treatment

Patients often express a fear that they are 'mad' and are reassured to hear how common the problem is and that it is treatable. In trying to answer the question 'what causes the seizures?' a helpful approach is to describe the known demographic and aetiological factors as they apply to that individual, together with a speculative model of how this might be related to dissociation. For example, one might explain: 'We don't fully understand what causes this disorder but two-thirds of people with it have suffered the sort of traumatic experiences you have described. We can't explain the link for certain, but it may be that when people are exposed to repeated frightening incidents as a child they learn to switch off. Initially this is a helpful thing for them to do, it protects them emotionally at the time. But it may come back later in life as these seizures.' It is important not to suggest abuse as a possible aetiological factor if this history has not emerged spontaneously for fear of encouraging 'false memories'.

A description of maintaining factors is especially useful when other aetiological factors are not apparent. Patients will often recognise that their confusion about the nature of the seizures, avoidance of situations in which they fear having one, and the protective reactions of carers together create a 'vicious circle' whereby fear of having attacks may eventually become the most important 'cause' of them. A few patients clearly identify stress as a trigger for individual attacks but most do not. This can be a very difficult issue. It may be helpful to explain that many patients are initially unable to identify triggers for their attacks but that these often become apparent with treatment. Further, that when triggers are found they often turn out to be fleeting stressful or unpleasant thoughts that the patient was barely aware of (or could not easily remember) that have little to do with their immediate circumstances. It may be useful to explain that we all think at many different levels at any one time and some of what we are thinking about is instantly forgotten. By way of illustration, asked to introspect for a moment, most patients will acknowledge that they have been thinking of other things while listening to the doctor's explanations. Examples of the link between physical symptoms and emotional state, and of the complex automatic behavioural accompaniments to emotions (as seen with grief or with rage) may help illustrate some of the physical attributes of seizures.

Finally, in describing treatment and prognosis it is worthwhile emphasising that simply understanding the nature of the problem and withdrawing AEDs is all that is required for some patients[53]. For those who have DS alone the news that they may come off antiepileptic medication is usually very welcome. It is important, however, to caution against abrupt withdrawal. Guidelines for AED withdrawal have been published by Oto and colleagues[70].

Information about DS is available online through two comprehensive and carefully devised websites written by neurologists for patients.

The first of these, www.neurosymptoms.org also covers functional neurological symptoms in general. The second, www.nonepilepticattacks.info includes specific self-help guidance for people with DS. Both are extremely useful resources.

Patients who have comorbid epilepsy often pose the most difficult management problems. Where both types of seizures are ongoing the main challenge will be to clearly identify, with the patient and carers, the different seizure types: residual uncertainty may undermine psychological treatment and lead to over-medication in order to 'play safe'. Showing patients and carers videos of seizures captured in telemetry is useful but the semiology frequently changes and the issue often requires regular review. In this situation home videos of seizures may help to avoid repeating vEEG telemetry.

Treatment

Pharmacotherapy is clearly appropriate for the relatively small proportion of patients with significant psychiatric comorbidity. Even in those patients without a comorbid psychiatric

disorder that might be expected to respond to anxiolytic or antidepressant treatment, some authorities advocate using such treatments[54]. However, a small randomised controlled trial of sertraline recently failed to show significant benefit[71].

For the majority of patients some form of psychological treatment is usually recommended[79]. There is relatively little evidence on which to base a decision about what form of therapy is best, although it is widely supposed that the nature of any associated psychiatric comorbidity (if any) is an important consideration. In patients with learning difficulties operant behavioural programmes using simple reward systems are often helpful[55,56]. The early literature includes a number of compelling descriptions of insight-oriented, dynamic psychotherapeutic approaches in patients with a history of DS and sexual abuse[57,58]. Rusch and colleagues reported treatment outcome in 33 patients[59]. Treatment, which included psychodynamic and cognitive behavioural approaches (mostly in combination), was tailored to reflect aetiology and comorbid psychiatric diagnoses. In a larger, uncontrolled series, Mayor et al[72] have recently reported outcome in 66 patients who received brief inter-personal (dynamic) therapy 'augmented' with cognitive behavioural techniques. One-quarter of patients were seizure free after six months. Other reports have described psychoeducational group therapy[60] and eye movement desensitisation[61]. Variations of therapy based on psychodynamic, insight-oriented and group-based methods are undoubtedly widely practised and believed to be effective but controlled studies of such interventions are needed.

The paroxysmal nature of DS, prominent somatic symptoms of arousal in many patients and an association with agoraphobic avoidant behaviour suggest that techniques developed in cognitive behavioural therapy (CBT) for the treatment of panic disorder might readily be adapted for DS[59,62]. A number of uncontrolled studies have now shown that CBT is associated with significant improvement[63,73,74]. Most recently, a randomised controlled trial has demonstrated a significant advantage of CBT compared with standard outpatient care[75]. Patients receiving CBT were three times more likely to become seizure free by the end of treatment. However, improvement was seen in both CBT and standard treatment groups and by six months follow-up the difference in outcome was no longer statistically significant. A second small randomised controlled trial has also suggested the effectiveness of CBT in DS[81]. A multicentre RCT is now under way in the UK comparing the effectiveness of standardised medical care with and without CBT[82]. Controlled studies of longer-term outcome following treatment are required, as are comparisons of different treatment approaches, including evaluations of brief simplified treatments which might be delivered more easily outside specialist neuropsychiatric services. Techniques developed for post-traumatic stress disorder and dysfunctional personality traits may also be helpful, but these and other techniques also require evaluation[59,64].

A significant proportion (see below) of patients continues to have seizures despite intensive treatment. A pragmatic approach in such cases is to offer long term-follow up to provide support for the patient and their family, social interventions to improve quality of life, and also to limit the cost and morbidity associated with further unnecessary investigations and medical interventions.

Outcome

A review of outcome studies[5] found that after a mean follow-up period of three years approximately two-thirds of patients continued to have DS and more than half remained dependent on social security. Psychiatric treatment has been associated with a positive outcome in some studies, but not others. A poor prognosis is predicted by a long delay in diagnosis and the presence of psychiatric comorbidity, including personality disorder. Being unemployed and in receipt of disability benefits has recently been reported to be a predictor of poor outcome[76].

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