REVIEW

The approach to patients with ''non-epileptic seizures''

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Up to one fifth of patients who present to specialist clinics with seizures do not have epilepsy. The majority of such patients suffer from psychologically mediated episodes; dissociative seizures, often referred to as ''non-epileptic seizures''. This paper describes the diagnostic evaluation of seizure disorders, including clinical assessment and the role of special investigations. The organic and psychiatric imitators of epilepsy are outlined and findings on psychiatric assessment are reviewed. This group of patients often proves difficult to engage in appropriate treatment and an approach to explaining the diagnosis is described. As yet there are no controlled trials of treatment in this disorder but preliminary evidence suggests cognitive behavioural therapy is both a rational and promising way forward.

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U p to one in five patients with apparently intractable epilepsy referred to specialist centres are found to have no organic cause for their seizures.[1–4] It has been widely supposed that this high prevalence reflects referral bias but a recent community based study found a similar proportion among patients with recent onset seizures.[5] This group of patients suffer from psychologically mediated paroxysmal behavioural disturbances that are often dramatic, alarming for bystanders, and all too easily mistaken for epilepsy. Diagnostic errors are the rule rather than an exception. Most patients are treated for epilepsy for several years[6 7] and by the time the correct diagnosis is made they will commonly have taken more antiepileptic drugs (AEDs) at higher doses and experience more side effects than an equivalent cohort of patients with epilepsy.[8 9] One in 10 patients will present in apparent status epilepticus.[1 7 8] Astonishingly, about one quarter of referrals to a specialist neurological intensive care unit with refractory status were found to have ''pseudostatus''.[10]

What this disorder should be called has been the subject of considerable debate. Some terms (hysterical seizures, pseudoseizures) are pejorative, unacceptable to patients,[11] and have largely been abandoned. Others (non-epileptic seizures (NES), non-epileptic attack disorder) merely describe what the condition is not, rather than conveying what it is. Furthermore, these terms have been used with different meanings: the term NES, for example, is sometimes used to refer to the group of neurological, cardiological, and other medical conditions, in addition to psychiatric disorders, which constitute the differential diagnosis for epilepsy,[4] while on other occasions the term is used as a form of loose shorthand to refer to the psychological attacks alone.[12] The terms psychogenic NES and functional seizures overcome some of these objections but formal psychiatric classification systems provide clearly defined labels. Unfortunately, though, there are still inconsistencies: thus, within DSM IV[13] such attacks are classified under somatoform disorder and in ICD 10[14] the diagnostic label ''dissociative convulsions'', is classified within the group of conversion disorders. It is the latter terminology that will be adopted here.

As we have seen, dissociative convulsions or seizures (DS) are common, the diagnosis is often missed, and when it is patients not only fail to receive appropriate treatment but are subject to unnecessary, costly,[15] and potentially harmful medical interventions. In considering the management of this disorder we will therefore focus on assessment and diagnosis before considering contemporary approaches to treatment.

CLINICAL ASSESSMENT

It should be emphasised from the start that epilepsy is primarily a clinical diagnosis. Great care must be taken to establish the precise sequence of events during an attack and history taking is not complete until an eyewitness account has been obtained. The duration of each phase of symptoms, including recovery from the attack, should be determined. Any habitual pattern in the circumstances that trigger attacks should be sought. Patients and eyewitnesses should be prompted for specific symptoms because significant features may not be mentioned spontaneously (for example, psychic and cognitive symptoms, automatisms, occurrence during sleep).

Medical differential diagnosis

The box lists the medical and psychiatric differential diagnosis for epilepsy (see Cook[16] and Andermann[17] for a review). Of medical disorders mistaken for epilepsy syncope is the most common[3] and in non-specialist settings is the condition most likely to be misdiagnosed as epileptic. It is important to note that tonic or clonic movements may be seen during syncope.[18] However, characteristic prodromal symptoms (lightheadedness, clammy sweatiness, a sense of receding sound and vision, nausea), associated cardiac symptoms, and a relation in some cases to postural changes or valsalva usually make identifying cases of syncope straightforward.[19 20] In patients with cerebrovascular disease the

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Abbreviations: AED, antiepileptic drug; NES, non-epileptic seizure; DS, dissociative seizure; ES, epileptic seizure; CBT, cognitive behavioural therapy

differentiation of transient ischaemic attacks from partial seizures may sometimes be difficult. Ischaemic episodes may last for seconds to minutes but usually occur with preserved consciousness, are associated only with a loss of function, and are not followed by more typical epileptic features. A comparatively long duration of symptoms is useful in recognising migraine, migraine equivalents (the latter featuring prodromal symptoms but no headache),[21] and vertigo. Abnormal startle phenomena, including hyperekplexia, are rare but often mistaken for epilepsy and need to be distinguished from startle induced seizures.[17 22] Paroxysmal movement disorders may be mistaken for epilepsy.[23] Sudden loss of muscle tone that may produce falls in response to an emotional trigger suggest cataplexy, which is usually found in association with other features of the narcolepsy syndrome (narcolepsy, hypnopompic or hypnogogic hallucinations, and sleep paralysis). Other parasomnias giving rise to complex behavioural episodes arising from sleep may be confused with epileptic automatisms although the former lack any preceding ictus and are usually of comparatively long duration.[24] Space occupying lesions in the 3rd ventricle may produce intermittent CSF obstruction associated with visual symptoms and are a rare cause of sudden episodes of collapse with loss of consciousness. Metabolic disorders associated with loss of consciousness usually have a protracted time course and are suggested by other features in the history.

Psychiatric differential diagnosis

Once epilepsy and other organic causes of seizures have been excluded there are three categories of psychiatric diagnoses that must be considered: (1) dissociative seizures; (2) factitious disorder; (3) other psychiatric disorders that have been mistaken for epilepsy. Dealing with the last category first, paroxysmal symptoms of psychiatric disorders may sometimes raise the question of epilepsy. The most common example of this is panic disorder.[25] Patients may report depersonalisation, derealisation, and tremulousness during panic attacks while partial epileptic seizures may include both emotional and somatic symptoms of anxiety.[26] Features that are useful in distinguishing the two conditions include a longer duration, cognitive symptoms, and the presence of specific environmental triggers in panic disorder and, in partial seizures, the unique quality of the emotional symptoms (''ictal fear'') together with associated more characteristic epileptic features in partial seizures. Paroxysmal symptoms in psychosis may sometimes raise the question of epilepsy but such symptoms (for example, hallucinations) lack the highly stereotyped quality of epileptic phenomena and episodes are usually of long and variable duration. Other psychiatric disorders sometimes confused with epilepsy include depersonalisation disorder and attention deficit hyperactivity disorder in which failing school performance and poor concentration may sometimes raise the possibility of juvenile absence epilepsy.

In most cases, however, the seizures will be the principal symptom and cannot be accounted for by another psychiatric condition. The two diagnostic possibilities are dissociative seizures and factitious disorder distinguished from one another by whether the seizures are thought to arise through unconscious processes (DS) or are deliberately enacted. In factitious disorder the patient is held to be deliberately simulating epilepsy for reasons understandable in terms of their psychological background. It is distinguished from malingering (not a medical diagnosis) in which people are simulating illness for some obvious practical gain (for example, compensation, avoidance of criminal responsibility).

The differential diagnosis of epilepsy

Medical causes of transient neurological dysfunction (with or without loss of consciousness)
- Syncope
  - vasovagal
  - cardiogenic

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

- Endocrine and metabolic
  - hypoglycaemia
  - hypocalcaemia
  - hereditary fructose intolerance
  - drugs and alcohol

Psychiatric disorders
- Psychiatric disorders that may be mistaken for epilepsy
  - panic disorder
  - psychosis
  - Attention deficit hyperactivity disorder
  - Depersonalisation disorder

- Dissociative seizures
- Factitious disorder

The semiology of dissociative seizures

A careful history will usually provide sufficient grounds for suspecting DS, which is by far the commonest psychiatric imitator of epilepsy. Since the introduction of video electro-encephalographic monitoring (vEEG telemetry) 30 years ago countless studies have compared DS with epilepsy aiming to find clinical features that distinguish one condition from the other.[27] Unfortunately, no one symptom or sign allows the diagnosis to be made with absolute certainty. Some of the more useful clinical features, together with important ''red herrings'', are listed in table 1.

Some two thirds of DS involve prominent motor features. The remainder may mimic partial seizures or involve a period of unresponsiveness with little in the way of motor activity.[7 8 28 36 37 41 44] The hallmark of epileptic seizures is that they are brief, temporary changes of neurological function that follow a highly stereotyped pattern from one occasion to the next. Furthermore, epileptic seizures conform to a number of familiar syndromes that have now been clearly defined.[45] It is any variation from these well described syndromes that will alert the experienced clinician to suspect DS. In addition to recognising an ''atypical sequence of events''[7] the most helpful objective features distinguishing DS from epileptic seizures are long duration (over two minutes), a gradual onset with motor features that wax and wane throughout the seizure, and, on recovery, evidence that the patient is able to recall events for a period of unresponsiveness. An episode of motionless unresponsiveness (that is reversible) lasting over five minutes is unlikely to have an organic explanation.[7] Additional features on history that favour (and only that) a diagnosis of DS rather than epilepsy include onset of seizures after the age of 10, a failed response

Table 1 Some clinical semiological features of epileptic and dissociative seizures

	Dissociative seizures	Epileptic seizures
Duration over two minutes	common[1 7 28–30]	rare
*Stereotyped attacks	common[7 31 32]	common
Motor features		
Gradual onset	common[7 28 31 33 34]	rare
Fluctuating course	Common[7]	very rare
Thrashing, violent movements	common[28 35–37]	rare
Side to side head movement	common[29 35]	rare
Asynchronous movements	common[29 38]	very rare
Eyes closed	common[28 39]	rare
Pelvic thrusting	occasional[1 29 40]	rare
Opisthotonus, ''arc de cercle''	occasional[28 31 41]	very rare
Automatisms	rare[41]	common
Weeping	occasional[42 43]	very rare
*Incontinence	occasional[7 35 44]	common
*Injury		
Biting inside of mouth	occasional[7 35 39 41 44]	common
Severe tongue biting†	very rare[7 35 39 41 44]	common
Recall for period of unresponsiveness	common[1 7 41]	very rare

*Three features (''red herrings'') that are commonly misinterpreted as evidence for epilepsy have been included in this table for emphasis. Otherwise the table lists clinical features that are often useful in distinguishing DS from ES. Figures for frequency of these features are approximate: common >30%; occasional = 10%–30%; rare <10%; very rare <5%. †Injuries to the tongue in epilepsy usually involve the side of the tongue. Bite injuries to the tip of the tongue or lip are rare in epilepsy and suggest DS.[39]

to AEDs, and the presence of risk factors for DS (including a history of previous unexplained medical symptoms, a psychiatric history, and a history of childhood traumatic experiences—see below). An absence of risk factors for epilepsy is reassuring in making a diagnosis of DS but their presence may be misleading[32] as, for example, DS are common in patients with learning difficulties (also associated with epilepsy) and a family history of seizures is common in patients with DS.[46]

Between 10% and 30% of patients with DS also have epilepsy,[1 7 28 36 38 41 43 44 47] which may create problems in diagnosis and management. A history of multiple seizure types is not a reliable indicator of comorbid epilepsy as some 20% of patients with DS alone report more than one type of seizure.[35 36 41] Special mention should also be made of frontal lobe epilepsy, which is often mistaken for DS. Frontal lobe seizures may involve bizarre emotional and behavioural features highly suggestive of DS.[47 48] These include intense emotional reactions, asymmetrical motor posturing, kicking and thrashing, body rocking, side to side head movements, and complex behavioural paroxysms such as pelvic thrusting, undressing, masturbatory activity, and uttering obscenities. Furthermore, despite the complexity of behaviours involved patients will often claim some preservation of awareness during attacks and there is frequently an extensive past psychiatric history (not least because these patients are often initially misdiagnosed as having DS). Characteristics of frontal lobe seizures that help distinguish them from DS are short ictal duration, stereotyped patterns of movements and occurrence during sleep (sometimes associated with secondary generalisation).

An opportunity to observe a seizure first hand and to examine the patient during a seizure may provide invaluable information.[1 7 28 29 35] Careful note should be taken of the type of movements seen, their distribution, and whether apparent clonic movements are rhythmic and synchronous (as they usually are in epilepsy) or not (DS). After a generalised tonic clonic seizure the corneal reflex will usually be impaired and plantar responses extensor. If the patient's eyes are shut the examiner should attempt to open them and note any resistance to this (DS). A simple test to look for avoidance of a noxious stimulus is to hold the patients 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 entails rolling the patient onto their side. In a patient with DS the eyes will often be deviated to the ground. The patient should then be rolled onto the other side and note taken if the eyes are still directed towards the ground (the Henry and Woodruff sign).[49] A second useful manoeuvre is to place a small mirror in front of the patient and look for evidence of convergent gaze and fixation on the reflection. This procedure may also prove useful in stopping the seizure. All of these examination findings must however be interpreted with caution: the ''correct'' response to any of these tests may be learned by patients who are simulating illness. Table 2 gives a checklist of examination procedures that may help differentiate DS from ES.

After careful clinical assessment the experienced clinician may often be in a position to reach a confident diagnosis. Sometimes, however, doubt remains, even if a seizure is witnessed: in one study an experienced epileptologist viewing videotapes of seizures correctly identified only 73% of DS and 71% of ES.[38] Special investigations therefore have an important role in diagnosis but there are pitfalls and limitations that will be considered next.

Table 2 Checklist of examination procedures that may help differentiate dissociative seizures from epileptic seizures

Examination procedure	Finding in DS
Drop patient's hand over their face	hand falls to the side
If eyes closed, attempt to open them	resistance to eye opening
If eyes open	
Place mirror in front of patient	evidence of visual fixation
Roll patient from one side to the other	(Henry and Woodruff sign, see text)
Corneal reflex	intact neurological reflexes
Plantar reflex	

The findings in the first three tests suggest some preservation of intact awareness and volition. By general consensus, however, these examination findings should not be interpreted as proving the patient is fabricating their illness. As a cautionary note, some patients may learn to produce the ''correct'' neurological response with any of these examinations.

SPECIAL INVESTIGATION
EEG

According to Chadwick[50] the EEG is ''one of the most abused investigations in clinical medicine and is unquestionably responsible for great human suffering''. While a single routine EEG may be normal in some 30% of patients with epilepsy (the false negative rate falls to around 15%, or even less in patients with repeated studies or sleep recordings[51]) Chadwick was highlighting the problem of false positives. Anything up to 15% of the normal population may have a ''non-specific'' abnormality noted on EEG. There is clearly a danger both that an EEG may be ''over reported'', especially if the request form sent to the electrophysiologist expresses no doubt about the diagnosis, and also that such non-specific abnormalities might be misunderstood by inexperienced clinicians as backing a diagnosis of epilepsy when they do no such thing. This problem is compounded by the fact that such non-specific abnormalities (principally a slow background rhythm) are more common in patients with DS than in healthy volunteers[52] and in patients with borderline personality disorder,[53] which is common in patients with DS (see below). It should be noted, however, that rigorously defined specific ''epileptiform'' abnormalities (generalised spikes or polyspike and slow wave abnormalities) are very rare (about 3 in 1000) in healthy people.[54][55] The EEG is just one factor that must be weighed up in making a diagnosis of epilepsy that ultimately rests on clinical judgement.

Video EEG telemetry

The gold standard investigation for seizure disorders is long term monitoring with video EEG (vEEG) telemetry. The patient is admitted to hospital with the aim of catching a seizure (ideally more than one) on both video and EEG, allowing the semiology of the seizure to be observed and providing an ictal EEG recording. The critical EEG findings[7] include ictal epileptiform discharges (which may be obscured or even mimicked by movement artefact) and post-ictal slowing of the background rhythm. An important sign that excludes organic causes of unconsciousness is the presence of an intact alpha rhythm (a neurophysiological correlate of alertness most prominent when the patient's eyes are closed) in an unresponsive patient. Aside from practical considerations (vEEG telemetry is an expensive investigation and is not widely available) there are also some important clinical limitations. Firstly, the ictal scalp EEG is often normal in simple partial seizures (in which consciousness is preserved)[56][57] and in frontal lobe seizures.[47][48] As already discussed frontal lobe seizures are particularly problematic as they may include bizarre motor and behavioural manifestations and are often mistaken for DS. In these cases the video recording will often be extremely useful. A further helpful point is that frontal lobe seizures commonly arise from sleep and the ictal EEG will show this clearly even if there are no other electrographic signs of epilepsy. Although patients with DS often report seizures in sleep, when they are captured on telemetry they are inevitably preceded by waking,[3] which again can be recorded electrographically. A more common problem concerns patients with seizures occurring so infrequently that they are unlikely to have an episode during telemetry. Patients with more than one type of seizure also require special attention. Because DS and ES often occur in the same patient care must be taken to ensure that a representative example of each seizure type has been captured. Occasionally patients with epilepsy may have a DS for the first time in their lives on a telemetry unit, perhaps brought on by the stress of admission to hospital and ''performance anxiety'' secondary to a perceived pressure to have an attack.[58] Thus, where possible someone who has witnessed the patient's seizures should be shown the video (with the patient's consent) to verify that the recorded attack is characteristic of the patient's habitual seizures.

Ambulatory EEG[59] may be conducted as an outpatient but suffers from the disadvantage of having no video recorded semiology to correlate with the EEG. Asking a patient's carer to video seizures may also be very helpful with the accepted limitation that the first few seconds after seizure onset will usually be missed. In an attempt to reduce the need for long and costly telemetry several investigators have explored the possibility of provoking seizures through suggestion while obtaining video and EEG recordings. Using procedures such as intravenous infusion of saline up to 90% of patients will have a DS[60][61] but these methods remain controversial because of ethical concerns about the use of placebo. Most recently, however, McGonigal and colleagues[62] combined simple suggestion with activation stimuli used routinely in EEG testing (photic stimulation and hyperventilation) and fully disclosed the aims of the procedure to patients. Sixty six per cent of patients experienced a DS provoked in this way compared with 33% in a control group who received identical activation stimuli but without suggestion. In these settings, because of very occasional false positive results in patients with epilepsy,[63][64] it is particularly important to have a witness confirm the provoked seizure as habitual.

Serum prolactin

Serum prolactin[65] rises to concentrations greater than 500 IU/ml in over 90% of patients after a tonic-clonic seizure and 60% of patients after a complex partial seizure. Simple partial seizures are not followed by a rise in serum prolactin and prolactin concentrations may be normal after prolonged status epilepticus. Blood must be taken between 20 and 30 minutes after the seizure and compared with a baseline sample. The test is, however, used less and less in specialist centres as false positive results have now been found in syncope[66] and DS.[67] Thus, while a normal post-ictal prolactin may be helpful diagnostically if the seizure was tonic-clonic in semiology, an increased post-ictal prolactin is non-specific.

PSYCHIATRIC ASSESSMENT OF PATIENTS WITH DS
Conscious/unconscious symptom generation

As we have seen, after organic diagnoses have been excluded and a primary psychiatric disorder that has been mistaken for epilepsy ruled out, the diagnostic possibilities are DS and factitious disorder. The distinction made between DS and factitious disorder in psychiatric classification systems implies a dichotomy between what is conscious and what is not. In practice the distinction may be difficult to make and a definite diagnosis of factitious disorder ultimately depends on the patient confessing their intent to deceive. The notion of unconscious symptom generation remains controversial[68] and a more valid way of conceiving the problem might be as a continuum with unconscious motivation at one end, conscious simulation at the other, and a large grey area in between, with ''self deception'' lying somewhere in the middle.

Most authorities suggest that most patients are unconscious of any wilful component to their seizures and factitious disorder is held to be rare.[37][65] However, many find this hard to accept. Three points in favour of the symptoms being unconscious are worth considering, although none is by any means conclusive: (1) most patients are compliant with antiepileptic medication, often for several years and to the point of toxicity, until the correct diagnosis is made; (2) when admitted for EEG telemetry most patients have a seizure within what they must surely perceive to be an intensively monitored setting; (3) DS are generally a poor imitation of epilepsy. In fact, factitious disorder will often be suspected whenever one of these three conditions is not met.

Psychiatric formulation: epidemiological consideration, comorbidity, and risk factors for DS

Psychiatric assessment should aim to identify putative risk factors for DS that may help the patient understand why they have the disorder and may direct psychological and other approaches to treatment. What follows is an account of the demographic characteristics of DS and possible predisposing and maintaining factors identified in the literature.

About three quarters of patients are women.[7 9 36 69] Seizures usually begin in the late teens or early 20s but there is a wide range.[7 9 36] Patients in lower socioeconomic groups and with less educational achievement are probably over-represented, although not in comparison with patients with epilepsy.[69]

A history of previous medically unexplained symptoms is present in up to 80% of patients.[7 36 41 70] In some cases symptoms will have attracted a medical diagnosis although objective evidence of pathology is lacking. The recently reported association of a diagnosis of asthma in patients with DS may be an example of this.[36 71] In addition to a history suggestive of somatisation, there is a high rate of psychiatric comorbidity. Maladaptive personality features of a borderline type are common,[72] often in the form of trait accentuations rather than personality disorder in itself.[73 74] Related to this, patients with DS show less adaptive coping styles.[75 76] Comorbid anxiety disorders and depression have been widely reported[36] but prevalence rates vary between studies and have often not been strikingly greater than seen in patients with epilepsy in those studies that have used a control group.[72 73 77]

The prevalence of abnormal personality in these patients suggests adverse experiences occurring in childhood or adolescence at a time when personality attributes are developing, and the risk factor to have attracted most attention in the literature is a history of childhood abusive experiences. Although there have been negative findings,[78 79] a number of large studies in which abuse has been carefully defined have shown higher rates of reported abuse in patients with DS compared with epileptic controls and unselected psychiatric patients.[69 80 81] Traumatic, abusive experiences in adulthood have also been implicated.[81] Other traumatic experiences or situations that foster low self esteem, for example bullying at school or unrecognised learning difficulties,[82] may be over-represented but have not been studied in adults with DS.

There is evidence that adverse life events are more common in the year preceding onset of DS[72] but triggers for initial seizures are often not apparent. Adverse family interactions[83–85] may serve both as predisposing and maintaining factors for DS. A pattern of avoidant behaviour, often exacerbated by carers' overprotective reactions, is a comparatively under-recognised maintaining factor but readily apparent on history taking in the form of an agoraphobic pattern of avoidance ostensibly for fear of having a seizure in an embarrassing or vulnerable setting.[84] Finally, for some patients the psychological and social advantages inherent to a medical sick role are undoubtedly important maintaining factors.[9 68 87] Such advantages include both an avoidance of responsibility and positive benefits such as the caring response elicited in others.

It should be noted that DS share many possible aetiological factors with other somatoform disorders. The paroxysmal nature of the symptoms, however, gives this disorder a unique quality that creates special difficulties for diagnosis but also raises specific treatment approaches.

TREATMENT
Explaining the diagnosis to patients

Treatment begins with an explanation of diagnosis. This must be handled openly but sensitively: if it is not, the patient is likely to reject the diagnosis, decline treatment, and go elsewhere for more investigations; a disaster in terms of time and expense, both for the patient and medical services.[88 89]

PRESENTING THE DIAGNOSIS OF DISSOCIATIVE SEIZURES

In discussing the diagnosis with the patient the following points should be covered:

(1) Reasons for concluding they do not have epilepsy
(2) What they do have (describe dissociation)
(3) Emphasise they are not suspected of ''putting on'' the attacks
(4) They are not ''mad''
(5) Triggering ''stresses'' may not be immediately apparent.
(6) Relevance of aetiological factors in their case
(7) Maintaining factors
(8) May improve after correct diagnosis
(9) Caution that AED withdrawal should be gradual
(10) Describe psychological treatment

It is important to involve patients' carers in this explanation.

Firstly, a clear explanation must be given of the reasons for concluding that the patient does not have epilepsy. This should cover any aspects of the patient's seizure semiology that are inconsistent with epilepsy and features in their history that make epilepsy less likely (for example, a failed response to AEDs, lack of risk factors for epilepsy). A thorough explanation of investigation results should follow that, if relevant, must address any non-specific ''abnormalities'' that the patient may previously have been told about and the way in which these can be put in context.

A description of what the patient does have then follows. Many patients will react unfavourably to the news that no medical explanation has been found and great care should be taken to emphasise that the doctor understands the attacks are real, disabling, and that the patient is not suspected of putting them on. A useful approach can be to tell the patient that they have attacks in which their mind or brain ''switches off'', involuntarily, and they lose control. It is often helpful to describe the concept of dissociation, explaining that the attacks represent an extreme form of something that is part of everyday experience using examples illustrating selective and divided attention (for example, reading a book and not hearing your name called, travelling home from work and remembering nothing of the journey). It should be explained that the symptoms are stress related but that it is usual for the stresses to be difficult to identify. Patients commonly object that the seizures cannot be caused by stress as they occur at times when they are relaxed. In this situation it may be helpful to explain that attacks may be triggered by stressful or unpleasant thoughts that the patient is barely aware of (or cannot remember) and may have little to do with their immediate circumstances. The concept that thinking may occur on a number of different levels at any one time can be described. Examples of the link between physical symptoms and emotional state (for example, crying, autonomic symptoms of arousal) and complex involuntary behavioural accompaniments to emotions (for example, as seen with sudden grief or with rage) may help illustrate some of the physical attributes of seizures. If the patient experiences somatic symptoms of arousal during their seizures[90] the relation of these features to anxiety can be described and the seizures likened to a ''panic attack without the panic'' in which dissociation (''switching off'') protects the patient from unpleasant or frightening emotions.

Patients often express a fear that they are being told they are ''mad''. They should be reassured that the condition they

have is common and the profile of a typical patient should be described, emphasising the points that apply to them. A link between traumatic experiences in childhood may be made meaningful by explaining that children exposed to abuse, especially if it is repeated, learn to "switch off" as a way of coping: DS may represent a re-emergence of this once adaptive response in the face of challenges, stress, or perhaps something that reminds the patient of painful memories. Some[88] have recommended raising the subject of abuse as a causal factor even if a history of it has not emerged. However, such an approach risks encouraging "false memories" and may best be avoided.

A description of maintaining factors is important and is often welcomed by patients who are sceptical about supposed psychological origins of their symptoms. It can be explained that whatever caused the seizures in the first place may remain unknown, but that worry about seizures, including what they are attributable to, and worry about the consequences of having a seizure may actually make the seizures worse and more frequent.[91] Patients will often relate to the confusion and anxiety engendered by receiving contradictory advice from a succession of doctors and the role this may have in perpetuating attacks. The concept of how avoidant behaviour, often exacerbated by a well meaning family's protective reactions, acts to reinforce anxiety about attacks[91] may give the patient and their family a rationale for change.

Finally, the patient should be given hope that their problems are treatable. Most patients are delighted to hear they may discontinue AEDs but they should be warned that this must be done gradually for fear of triggering a withdrawal seizure. It is worth emphasising that once confusion about diagnosis is resolved a significant proportion of patients find that this explanation alone leads to a resolution of the attacks over time.[92]

Approaches to treatment

There have been no controlled trials of treatment in DS. The evidence comes from case reports and small treatment series.[80][86][91][93–101] In the small proportion of patients who have significant comorbid depression or anxiety appropriate pharmacotherapy is indicated. For most, however, some form of psychotherapy will be the mainstay of treatment. In patients with learning difficulties operant behavioural programmes using simple reward systems are often helpful.[94][95] 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.[98][99] More recent reports have described psychoeducational group therapy[100] and eye movement desensitisation[101] in similar patient groups. Variations of therapy based on psychodynamic, insight oriented, and educational principles are undoubtedly widely practised and believed to be effective but further studies of such interventions are needed.

The paroxysmal nature of the attacks, the occurrence of somatic symptoms of arousal in many patients, and the 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.[86][91] This CBT model also provides a useful rationale for treatment in patients who report no history of past traumatic experiences or who have received psychotherapy for this but continue to have seizures. A recent study involving 20 patients treated with CBT reported a significant reduction in seizures six months after treatment ended and, perhaps more importantly, found improvements in work and social outcome.[86] CBT techniques developed for personality disorder may be helpful but these and other techniques require evaluation.[102] A significant proportion (see below) of patients continue to have seizures despite intensive and varied treatment. A realistic 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 despite seizures, and also to limit the cost and morbidity associated with further unnecessary investigations and medical interventions.

Outcome

A recent review of outcome studies[27] found that after a mean follow up period of three years about two thirds of patients continued to have DS and more than half remained dependent on social security. Receiving psychiatric treatment has been associated with a positive outcome in some studies, but not in others. A poor prognosis is predicted by a long delay in diagnosis and the presence of psychiatrc comorbidity, including personality disorder.

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