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Epilepsy and Hysteria

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Charcot's term 'hysteroepilepsy' implies brain mechanisms in common between epilepsy and hysteria. No such direct link between these two conditions exists. Nevertheless, there are a number of

indirect associations between epilepsy and hysteria that can lead to confusion in the differential diagnosis and management of the patient with seizures.

Hysterical pseudoseizures are attacks of sudden unconsciousness, usually (but not invariably) associated with dramatic motor manifestations, which simulate epileptic attacks to a varying degree. The term 'pseudoseizure' is not ideal, since it conveys the impression that the event is not really a seizure. The term 'pseudoepileptic seizure' is more appropriate, since it acknowledges the occurrence of seizures and their resemblance to genuine epileptic fits, but avoids the implication of physiological mechanisms in common between epilepsy and hysteria.

The other terms used to describe hysterical fits ('simulated epilepsy' and 'psychogenic seizures') should not be used. 'Simulated epilepsy' implies malingering; this is rarely the case, since the patient with hysterical fits is not aware of the underlying psychological motivation for them. Psychological stress is important in precipitating frank epileptic attacks in some epileptic patients. Indeed, a recent survey of unselected primary care patients suffering from epilepsy reported that 72% of the sample of 300 found emotional upset to be accompanied by an exacerbation in seizure frequency (Dowds *et al*, 1983). Hence the term 'psychogenic seizure' should be avoided.

Pseudoepileptic seizures are not an infrequent manifestation of hysteria, being the second most common form found in 381 patients with hysteria (Ljungberg, 1967); 20% of this series presented with hysterical fits. More recently, Reed (1975) found a 9% prevalence among patients at the Maudsley Hospital admitted between 1949 and 1964 with a diagnosis of hysteria. Rather more surprising is the paradoxical observation that genuine epilepsy and pseudoepilepsy can coexist in the same patient. A present or past history of epilepsy is found in a significant number of patients with pseudoepileptic seizures referred to hospital for investigation, the prevalence varying from 12% to around 65% (Merskey, 1978*a*; Fenton, 1982; Lesser, 1985). Doubtless the wide variance reflects the heterogeneous nature of the series of small groups of patients reported in the literature from different centres.

It may seen strange for people with genuine fits to display pseudoepileptic attacks as an hysterical manifestation. One would think that genuine epileptic fits would provide a more than adequate locus for the illness-rewarding behaviour of the patient who responds to stress using hysterical mechanisms. However, the real epileptic attacks of the patient with pseudoepileptic seizures are often well-controlled by anticonvulsant therapy. Indeed, there is evidence that the development of classical hysterical fits can be facilitated by anticonvulsant drug toxicity (Niedermeyer *et al*, 1970), but this would only account for pseudoepileptic seizures in a minority of patients with epilepsy. Previous episodes of unconsciousness, either experienced personally or observed within the family or work environment, often provide a model for the choice of pseudoepileptic seizures as the hysterical manifestation. Roy (1980) found a personal history of epilepsy or an experience of working in a medical environment in about one-third of a series of patients with hysteria, the majority of whom had hysterical convulsions. Therefore, if the person with epilepsy is predisposed to hysterical mechanisms as a means of coping with a stressful situation, the previous experience of genuine epileptic attacks will have a major influence in determining the selection of fits as the hysterical manifestation.

Pseudoepileptic seizure patients also display many other features indicative of psychopathology: a family history of psychiatric illness, a past personal history of psychiatric disorder with suicidal attempts, a diagnosis of personality disorder, sexual maladjustment, disturbed interpersonal relationships with parents or heterosexual partner, an unsatisfactory current life situation, and concurrent affective symptoms. They also tend to have significantly higher scores on the General Health Questionnaire, the Wakefield Self Assessment Depression Inventory and the Hamilton Rating Scale for Depression (Roy, 1977; Lesser, 1985).

Epileptic and pseudoepileptic seizures: differential diagnosis

Clinical manifestations

In the differential diagnosis between epileptic and pseudoepileptic seizures, the clinician should always supplement the patient's account of the seizures by obtaining an accurate history from a reliable observer and be thoroughly familiar with the clinical features and the classification of the wide variety of epileptic seizure phenomena and their pathophysiological mechanisms (Commission on Classification, 1981).

Typical of the pseudoepileptic grand mal attack is the marked involvement of truncal musculature, with some opisthotonos (a complete *arc de cercle* is now very rare) and random struggling or thrashing movements of the limbs, trunk or head. Side-to-side throwing or rolling of the head or body is common.

The pseudoconvulsive movements may increase if restraint is imposed, and the patient may struggle to free himself from the restraint and even become combative; this is in marked contrast to the stereotyped tonic-clonic convulsions of the grand mal epileptic fit.

Cyanosis, an invariable feature of grand mal convulsions, is rare in pseudo-grand mal attacks, although it may occur as a result of breath-holding. The pupillary reaction to light and the corneal reflexes are retained during a pseudoepileptic seizure, but may be difficult to elicit due to the patient actively moving about or to upward deviation of the eyes. Indeed, during the pseudoepileptic attack the patient may actively resist attempts to open his eyes. Pressure on the supraorbital notch may cause head withdrawal, indicating that the patient has some perception of pain. Responsiveness to painful stimuli is entirely lost during the tonic-clonic phase of an epileptic grand mal attack. The facial expression during a grand mal seizure is characterised by trismus in the tonic phase and symmetrical twitching during the clonic phase. In the pseudo-grand mal fit, the face reflects either repose or a variety of emotions, including ecstasy.

The level of consciousness may fluctuate during the pseudoconvulsive phase, so that the patient may be able to recall some or all of the events of the fit. However, the presence or absence of diminished awareness during the attack can be difficult to determine; moreover, some epileptic attacks of partial origin can occur in clear consciousness. Nevertheless, the demonstration of retained awareness of the environment while bilateral or whole body convulsive movements are in progress is strongly in favour of the diagnosis of a pseudoepileptic seizure. A marked emotional display either after, or (on rare occasions) during an attack, with the patient in floods of tears or shouting obscenities, is again suggestive of an hysterical fit.

Following a genuine grand mal attack, the plantar responses become extensor for a short time. However, the responses must be elicited carefully, since it has been shown that healthy adults can learn to dorsiflex their toes in response to plantar stimulation of the foot (Lesser, 1985). If the tongue is bitten at all in pseudoepileptic fits the lacerations tend to be minor, involving the tip rather than the side of the tongue, or the lips rather than cheeks. Loss of bladder control and injury by falling during pseudoepileptic attacks may occur, but are uncommon.

The epileptic aura signals the transition of the episodic interictal firing of epileptogenic tissue into the continuous discharge that recruits additional neuronal circuits and provokes the clinical manifestations of the fit as it spreads to involve large areas of brain. Unless the patient has multifocal disease, which occurs only in cases of extensive brain damage, the aura of the epileptic patients, if present at all, will be identical or very similar during different attacks. Neither the presence nor absence of an aura, nor the type of the aura, provides useful differential diagnostic information.

Subjective phenomena herald the onset in a significant minority of pseudoepileptic fits. Specific symptoms include palpitations, malaise, choking, dizziness, paraesthesiae in the face or extremities, pain, olfactory and gustatory hallucinations, and visual illusions and hallucinations (Lesser, 1985). Pain, either in the body, extremities, or head, is rare as an epileptic aura. The only exception is a focal seizure of parietal origin, in which there is usually contralateral parasthesia with spread of a seizure activity to the motor cortex so that the affected extremity will show clonic convulsive movements. On the other hand, a prodromal complaint of headache is quite common in pseudoepileptic seizures (Finlayson & Lucas, 1979). Auras that contain a great number of somatic or visual symptoms which vary on different occasions suggest pseudoepilepsy.

Epileptic attacks usually begin abruptly. Although many pseudoepileptic fits do so also, a consistently gradual onset favours the latter diagnosis. Precipitation by shock, surprise or emotional distress must be interpreted with caution, since such factors may trigger epileptic attacks in susceptible individuals. Pseudoepileptic seizures may be provoked by hyperventilation, which will on occasion precipitate a true ictal event. The seizure duration is the same for the two types of attack, though a gradual termination is more often observed in pseudoepilepsy (Lesser, 1985).

Pseudoepileptic attacks usually occur when other people are present, especially those of significance in the patient's life, but there are exceptions. Unlike true epileptic seizures, pseudoepileptic seizures never occur during sleep. Careful enquiry about the person's state of awareness at the time of the onset of the seizure is necessary, since patients may develop pseudoepileptic attacks immediately on waking from sleep. Pseudoepileptic seizures are sometimes frequent, attacks recurring many times per day; this is unusual in real epilepsy, with the exceptions of status epilepticus, petit mal absences and some patients with minor complex partial seizures. Indeed, the recurrence of persistent and frequent major fits despite adequate anticonvulsant drug dosage, evidence of satisfactory compliance, and serum concentrations within the optimal range should raise the suspicion of pseudoepileptic seizures.

Classification of pseudoepileptic seizure manifestations

Analysis of videotaped pseudoepileptic fits has lead to attempts to classify the range of clinical phenomena. In a study of 27 patients (Gulick *et al*, 1982), motor manifestations were found to be bilateral in 15 and unilateral in 3, while 8 displayed multiple behavioural phenomena and 3 decreased responsiveness only. Luther *et al* (1982) observed generalised convulsive-type attacks in 15 out of 37 attacks in 30 patients; 21 resembled elementary partial seizures with secondary generalisation, and one a complex partial seizure.

Lesser (1985) has proposed the following main categories of manifestation: sustained or repetitive muscular contractions, either unilateral or bilateral; muscle inactivity or loss of tone; unresponsiveness alone; and unresponsiveness in association with quasi-purposeful or automatic behaviours. Episodes may occur either with or without an aura and, in any one patient, more than one of these patterns may coexist.

Some unusual presentations of pseudoepilepsy

Repeated pseudoconvulsive seizures without recovery of consciousness between attacks, with incontinence of urine or tongue biting during some of the fits, may simulate status epilepticus, leading to transfer to an intensive care unit and exposure to the potential hazards of apnoea and hypotension due to the vigorous pharmacological treatment of the apparent status. Toone & Roberts (1979) described three such patients, and made the point that some patients with pseudoepileptic seizures have attacks that bear a striking resemblance to genuine epilepsy. This is particularly true of patients with some experience or knowledge of epilepsy, a problem among nurses with pseudoepileptic seizures; two of the patients described by Toone & Roberts were nurses. I vividly recall a trained psychiatric nurse who had a series of hospital admissions for control of status epilepticus. It was only when the hemiconvulsions suddenly and inexplicably changed from one side of the body to the other after the intravenous cannula and drip set delivering the medication and nutritional fluids had been inserted in the opposite arm that the suspicion of pseudoepilepsy was aroused! This diagnosis was confirmed during a prolonged period of in-patient observation, including gradual withdrawal of anticonvulsants and replacements by a placebo. In organic status epilepticus the patient does not regain consciousness in the postictal period, and has further seizures separated by intervals of not more than 15–30 min. As the status progresses the seizures become shorter and less vigorous, but there are never any thrashing or throwing movements. The diagnosis should be verified by electroencephalography (EEG); there will always be seizure discharges in association with the attacks in status epilepticus. In pseudostatus epilepticus the EEG tracing shows only muscle movement and electrode artefacts during the pseudoconvulsive episodes.

On rare occasions focal neurological signs can be manifest during pseudoepileptic fits. One patient has been described who produced pupillary dilation and an extensor plantar response by pressing his thumb into the left side of his neck (Hammond, 1948). Another rare but potentially confusing manifestation of pseudoepilepsy can be recurrent injury. I recall a young student nurse with pseudoconvulsive seizures; after each attack she invariably had bruising over the face, limbs and trunk. Such extensive bruising is rarely seen after a grand mal convulsion. Hence, if the nature and extent of the injuries received during a fit are inappropriate for the circumstances of the seizure, the epileptic nature of the attacks should be open to critical re-examination.

Although the most common type of pseudoepileptic seizure is the pseudoconvulsive type, less dramatic forms do occur. Some patients may present with transient episodes of unconsciousness associated with pallor and slumping to the ground without convulsive movements resembling syncope. However, in the latter condition the patient is usually responsive by the time he or she falls to the ground, the face is pale, and there is no resistance to eyelid opening or to testing the corneal reflexes. In the pseudoepileptic seizure patient the period of unresponsiveness will usually be longer, and active resistance to eye opening may be manifest. The facial colour is either normal or flushed.

Some brief attacks of pseudoepilepsy may resemble simple partial seizures of the motor type, with twitching of one side of the face or jerking of limbs down one side of the body. The cortical motor representation of the face, thumb and hand occupies the largest proportion of the lateral surface of the motor cortex (Penfield & Jasper, 1954); the trunk has a small representation, and the leg areas are on the mesial surface. For this reason, epileptic attacks originating in the motor area affect mainly the face, thumb and hand. Jerking of shoulder or elbow in the absence of jerking of face and hand is therefore unlikely to be of epileptic origin. Since in most right-handed people and in 60% of left-handed people speech is dependent on the function of the left hemisphere, a right-sided motor seizure is usually associated with speech arrest; if speech is

preserved during a clinically right-sided seizure and the patient is not left-handed, an epileptic basis is therefore unlikely. The movements of the focal motor attack are either tonic or clonic in nature, in contrast with the trembling or grossly irregular shaking of the pseudoepileptic variety. Sensory manifestations may occur as part of a minor pseudoepileptic attack. However, the location and spread of the sensory phenomena do not usually fit into a consistent and orderly pattern that reflects progressive involvement of adjacent areas of the brain by the spreading seizure discharge.

Hysterical fugue states, complex partial seizures and transient global amnesia

An hysterical fugue may be misdiagnosed as a complex partial seizure with automatic behaviour. However, automatic behaviour of epileptic origin is of brief duration, lasting minutes rather than hours. A classical temporal lobe aura may precede the attack. During an epileptic automatism there is usually evidence of clouding of consciousness, poor appreciation of the environment, and purposeless, inappropriate automatic behaviour. The duration of the hysterical fugue is hours or days. The patient's behaviour is usually well structured and co-ordinated and not inappropriate to the circumstances of the environment, despite the lack of knowledge of personal identity. Indeed, the behaviour seems goal-directed, the unconscious motivation being to escape from an intolerable situation.

Transient global amnesia is another disorder that can be confused with both hysterical fugue states and epileptic automatism. It differs from the latter condition in that the patient continues with routine activities but fails to lay down any permanent memory traces throughout the episode. Knowledge of personal identity is retained during the attack. The behaviour is well-structured and consistent with what is going on in the environment at the time. Memory gaps are not restricted to matters of personal concern nor specific emotionally loaded themes, and there are no inconsistencies in memory performance.

Other episodic organic disorders that simulate hysteria

The discussion of differential diagnosis has so far emphasised the need to avoid the mistake of diagnosing true epilepsy, when the attacks are actually those of pseudoepilepsy. Less frequent, but just as important, is the risk of wrongly attributing the diagnosis of pseudoepilepsy to episodic disturbances of behaviour that have a truly epileptic basis.

Patients with paroxysmal choreo-athetosis suffer from episodes initiated abruptly by restless movements of upper and lower limbs which progress to typical choreiform activity. The movements may be unilateral with generalisation, symmetrically bilateral or strictly unilateral. The patients remain conscious, usually do not fall, and are acutely embarrassed by the occurrence of such attacks, which may last between half an hour and several hours. They tend to be precipated by emotional stress, coffee, or beer, and may be aggravated by anticonvulsant medication – especially phenytoin, which is sometimes prescribed when a seizure disorder has been mistakenly diagnosed. There is frequently a positive family history for this condition.

In startle or movement-induced seizures the patients suddenly drop to the ground with associated loss of consciousness as a result of a sudden startle stimulus or of suddenly rising from the sitting position. The attack is short-lived, and the patient returns to consciousness immediately. It usually results from a myoclonic jerk, and the EEG frequently shows spike wave discharge. Myoclonic jerks occur mainly in patients with primary generalised tonic-clonic seizures, but can be a manifestation of such progressive brain syndromes as Lafora's disease, subacute sclerosing panencephalitis and Creutzfeldt-Jakob's disease. Sometimes (in primary generalised epilepsy and Lafora's disease) the myoclonus is severe and causes gross uncoordination of movement and gait. In some patients, emotional stress greatly aggravates the myoclonus. The link between the patient's emotional state, the jerking and apparent ataxia can lead to an erroneous diagnosis of hysteria, especially as the disorder may have an episodic quality and may lead to the avoidance of situations. Myoclonus tends to be worse in the mornings. The jerks often come in rapid succession, and herald a tonic-clonic seizure. EEGs frequently reveal that they are associated with bursts of generalised spike wave activity, and photic stimulation may elicit the myoclonus and associated generalised spike wave complexes.

The psychic auras that often initiate the complex partial seizures of temporal lobe epilepsy can be a bewildering and distressing experience for some patients. Those people whose habitual way of coping with psychic pain is denial may dissociate into a fugue state in response to the distressing subjective experience of the aura. Alternatively, they may respond by manifesting disinhibited, explosive behaviour in an attempt to escape the stressful

event. In my experience such reactions are rare, but undoubtedly do occur, especially in patients of low intelligence and those predisposed to using hysterical mechanisms to deal with stress. For instance, a 30 year old man of low intelligence and limited social and verbal skills had problems in relating to the opposite sex; at a time when he was having difficulties with a girlfriend, he experienced several attacks of panic followed by disinhibited behaviour. The attacks responded to chlordiazepoxide, and were considered to be an hysterical response to stress. It was only when the same sequence of events was repeated several years later, when he was having difficulties with another girlfriend, that the attacks were shown to be of temporal lobe origin with secondary fugue states.

Aetiology of hysterical pseudoepileptic seizures

Kendell (1974) and Smith (1978) have formulated essentially similar aetiological models for hysteria, in which hysteria develops as a response to emotional stress or conflict when a series of environmental, biological and personal predisposing (vulnerability) factors are present within the individual or as part of the current life situation. These are an illness-rewarding situation (when the advantages of being sick outweigh those of health), and individual variables such as female gender, youth, inferior social status, immigrant status, low intelligence, hysterical or passive-dependent-immature personality, suggestibility, difficulty in the overt expression of feelings, a tendency to dissociate and use denial mechanisms as a means of coping with stress, and personal experience of physical illness in relatives, self, peers or acquired as a member of one of the health care professions. The symptoms and signs of the resulting hysterical syndrome mimic organic disease, and permit the patient to adopt the sick role with consequent relief from the precipitating stress or conflict.

One important element missing from this model is organic brain disease. This seems to facilitate the use of hysterical mechanisms (Slater, 1965; Whitlock, 1967; Merskey & Buhrich, 1975; Merskey, 1978*a*, *b*; Roy, 1980, 1982) and is reported in 40–63% of patients with hysteria. Although Slater made the important observation that hysteria can herald the onset of the central nervous system disease, pre-existing organic brain disease is also a common predisposing factor to the development of hysterical symptoms.

This modified model clarifies the genesis of hysterical pseudoepileptic seizures. Precipitating stress factors are frequently present (80% of those studied by Standage & Fenton (1975) – 53% psychological and 27% physical). An illness-rewarding situation is often apparent. Anxiety and depressive mood change is common, as is other evidence of psychological maladjustment (Roy, 1977, 1980, 1982). The sex distribution is predominantly female, personality disorder is not infrequent, and organic brain disease is reported in between one-quarter and one-half of patients. The brain dysfunction tends to be acquired early in life, usually before the age of 15, and is often accompanied by cognitive impairment (Standage, 1975; Standage & Fenton, 1975; Roy, 1977). Side-effects of anticonvulsant drugs can be another factor. Previous episodes of unconsciousness, including epileptic fits either experienced personally or observed within the family or work environment, often provide a model for the choice of pseudo-epileptic seizures as the hysterical manifestation. Unconscious symbolic processes are not important in symptom choice (Standage & Fenton, 1975).

Investigation

As already explained, the correct diagnosis of patients with pseudoepileptic seizures can be difficult, especially those who present with a history of both well-controlled epilepsy and frequent pseudo-epileptic seizures. Many such patients will have been treated with large doses of anticonvulsant medication in the mistaken view that uncontrolled epilepsy is the problem. Even when the differential diagnosis seems straightforward, it is often impossible on an out-patient basis to elicit any underlying psychological difficulties to account for the pathogenesis of the attacks. The use of denial mechanisms in response to conflict or stress inevitably leads to defensiveness and denial of emotional difficulties during initial diagnostic interviews. Indeed, even the mere suggestion of an emotional basis for the attacks will often be angrily rejected. Similar attitudes are usually present in the patient's close relatives. To overcome these difficulties it is best to admit the patient to hospital, where the clinical pattern of the seizures can be carefully observed and described, preferably by staff who have special experience in the observation of fits.

Electroencephalography

EEG abnormalities are not infrequently recorded, but must be interpreted with caution. The presence of EEG paroxysmal abnormalities of the type associated with epilepsy does not necessarily confirm that the observed attacks have an epileptic basis. The

former can occur in the pseudoepileptic patient for one or more of the following reasons:

(a) underlying epilepsy, usually well controlled
(b) bursts of generalised spike wave complexes or paroxysmal theta activity, due to anticonvulsant drug withdrawal. Such withdrawal effects may persist for as long as 21 days after the end of the withdrawal phase
(c) generalised fast spike and wave complexes are found in nearly 3% of apparently healthy people, presumably being a reflection of an unduly low convulsive threshold (Fenton, 1974).

Background activity abnormalities such as focal slow waves, a marked alpha or beta rhythm asymmetry or diffuse slowing of the dominant activity frequency are evidence of organic brain dysfunction. However, their presence in a patient with seizures of uncertain origin cannot be regarded as unequivocal evidence that the attacks are epileptic, since organic brain disease is an important predisposing factor in the genesis of pseudoepileptic seizures. Anticonvulsant drug intoxication produces diffuse EEG background dominant activity frequency slowing, and can also cause pseudoepilepsy to develop. Hence, the routine EEG is of limited help and may even be misleading in the investigation of pseudoepileptic attacks.

Prolonged EEG recording over hours or days, using either conventional leads, a videotape telemetry system, or ambulatory monitoring with a portable tape recorder that can be carried about by the patient, often makes it possible to record an actual seizure (Oxley *et al*, 1981; King *et al*, 1982). Muscle and movement artifacts caused by the convulsive movements may make the presence of ictal discharge during the seizure difficult to identify. However, scrutiny of the recording just prior to seizure onset and after cessation of the clinical seizure can be fruitful. An epileptic seizure of partial (focal) origin may be accompanied by an EEG seizure discharge lateralised to the hemisphere origin prior to onset of the clinical manifestations of the attack. In addition, during the postictal phase there may be an asymmetry of EEG background activity, with apparent reduction in normal rhythms and the appearance of slow waves over the hemisphere of origin of the seizure discharge. After a generalised convulsion there are always postictal EEG changes; transient electrical silence is followed by the appearance of irregular slow waves which, over the next few hours, are gradually replaced by normal activity. Hence, if the recording of an actual seizure is neither possible nor convenient, it is still useful to record an EEG within an hour of the occurrence of the seizure, so that the postictal changes (if any) can be studied. However, their absence cannot exclude epilepsy, since the changes even after a real grand mal attack may be only present for a few minutes. An attack with major convulsive movements without EEG changes is almost certainly non-epileptic in nature, but in true partial epileptic seizures there may also be an absence of any scalp EEG change.

If the patient is receiving anticonvulsant therapy on admission, the medication should be continued until there has been sufficient time to observe the nature of the attacks. If the evidence in favour of an epileptic basis is not convincing, the anticonvulsant drugs can then be gradually replaced by placebos. As well as depressing cognitive function, heavy anticonvulsant medication can suppress the EEG paroxysmal abnormalities associated with epilepsy. However, it is often difficult to obtain appropriate placebo tablets. If so, the reasons for the proposed drug withdrawal will have to be discussed with the patient and the drug withdrawal procedure can then be carried out with the patient's knowledge and consent. In the event of placebos being used, the placebo nature of the medication will have to be disclosed to the patient; this is best done at an appropriate time during the parallel programme of psychotherapy. If, following withdrawal, real epileptic attacks recur, anticonvulsant medication will have to be reintroduced. The dosage required is usually small.

Other useful investigations

Serum prolactin levels rise sharply after many, but not all, generalised tonic-clonic convulsions to 1000–2000 mUnits/litre; this may be helpful in differentiating epileptic grand mal seizures from non-epileptic pseudo-grand mal attacks (Trimble, 1978; Lesser, 1985). A major difficulty is that both false-negative and false-positive results have been reported after major convulsive attacks. Furthermore, significant elevations are less common and less consistent following complex partial seizures, and do not occur after simple partial seizures (Dana-Haeri *et al*, 1983; Lesser, 1985). Therefore, postictal serum prolactin changes must be interpreted cautiously in the light of the associated clinical and EEG manifestations. Since organic brain disease is a common finding in patients with pseudoepileptic seizures as well as epilepsy, neurological investigation is not as helpful as one might predict. However, a computerised tomography scan will often identify underlying pathology of the central nervous system, and is useful in excluding progressive brain lesions that present with hysterical manifestations.

Treatment

The psychological management of the patient should run in parallel with the programme of observation and investigation. Rapport will be gradually established during the initial interviews. As rapport and mutual trust between doctor and patient develops, the façade of denial will gradually be penetrated and a process of clarification and non-directive discussion of the patient's symptoms and current life problems and their inter-relationship can begin. As the link between the fits and their emotional state begins to be understood by the patient, the acceptance of the psychogenic origin of the attacks becomes possible without loss of face. If a videotape recording of the pseudoepileptic attack is obtained, it can be used to show the patient what he looked like during a seizure. The non-epileptic nature of the movements or behaviour can be pointed out, along with a demonstration of the normal EEG data to both patient and family. This procedure can facilitate the development of insight into the nature of the seizures (Feldman *et al*, 1982).

Providing the patient's intelligence level is appropriate, and motivation for self-examination adequate, the exploratory psychotherapy can progress to an examination of the psychological meaning of the illness and the habitual defence mechanisms used against anxiety. The patients should be encouraged to acknowledge and discuss their emotional difficulties and tensions as a healthy alternative to denying their existence. Such a psychotherapeutic approach will tend to focus on an examination of the relationship between the therapist and patient (transference) and how this reflects relationships in his or her formative years and current life situation. The number of sessions of such focal psychotherapy will depend on the amount of available time and the therapist's assessment of the patient's capacity to benefit. A minimum of ten and a maximum of thirty one-hour sessions will be required. If this psychodynamic approach is inappropriate because of low intelligence, poor motivation for self-examination, or inability to cope with the inevitable transference problems, a more supportive psychotherapeutic role is still helpful, the aim being to permit abreaction, reduce emotional tension, and provide encouragement, support and guidance in dealing with current life problems.

One of the basic principles of learning theory is that patterns of behaviour that are rewarded tend to increase in frequency. When the pseudoepileptic nature of the attacks is firmly established, the staff should be encouraged to ignore their occurrence, but to respond to the patients when they are free from seizures. The patient should be encouraged by all members of staff to express and discuss emotions when distressed. Events in the ward environment that upset the patient and are followed by attacks can be discussed and used as examples of the operation of dissociation and denial mechanisms. More healthy ways of coping with such potentially stressful events can be encouraged and rehearsed. It is important that all members of the therapeutic team adopt a consistent approach to the patient. This must be sympathetic but firm. Clear guidelines about how to handle the varied repertoire of behaviour must be formulated so that the patient is not given the opportunity to manipulate individual staff members.

As patients' awareness of their emotional difficulties grows during the psychotherapy, the fits tend to subside. However, the patients may react by becoming depressed or by developing other conversion symptoms; alternatively, they may show their distress by self-damage, damage to property, or by various types of demanding histrionic behaviour. Such behaviour should be ignored as much as possible, but if it becomes seriously disruptive firm boundaries defining the limits to be tolerated by the staff should be drawn up and communicated to the patient by the therapist. A consistent approach is facilitated when all members of staff can meet regularly to discuss management, either during regular staff meetings, ward rounds, or clinical conferences.

Work with the patient's parents or spouse is always required. Simple explanation about the nature of the attacks and their relationship to emotional conflict and tension is essential. Advice to avoid rewarding their occurrence with too much attention, sympathy or concern is necessary; again, the need for a consistent approach should be stressed. Family or marital psychotherapy may be necessary if problems exist. A further strategy of management should be based on an attempt to minimise the disadvantages of health and the advantages of sickness. This will require careful and detailed inquiry to elucidate the full implications for the patient of both states. If the stress which caused the patient to adopt the sick role is a temporary one, little is needed beyond a firm prediction of recovery and the provision of a treatment setting in which this can take place gracefully. If the advantages of the sick role are substantial and enduring, however, a carefully planned regime will be needed. This will involve the patient's relatives.

After discharge from hospital, a long-term supportive relationship with the therapist is helpful. Contact should be relatively frequent (once every few weeks) for the first few months, in order to cope with crises that may arise, followed by a gradual

lengthening of the intervals between interviews (to once every few months). The follow-up should be continued for at least two years. Contact with relatives should also be maintained after the patient leaves hospital, in order to provide support and advice about management. It is also important to inform all medical personnel concerned with the patient's care of the management strategy, especially the general practitioner. This will avoid the reintroduction of, or increase in, anticonvulsant medication and further unnecessary investigation should the pseudoseizures recur. It is best if the management is supervised by one person.

Should the patient prove unresponsive to the combined dynamic and behavioural psychotherapy approach, an alternative is to use a seemingly paradoxical procedure with the consent and co-operation of the relatives: the patient is informed that the fits are likely to continue unabated, since all attempts to understand and treat them have failed, and that their frequency may even increase. Because conventional treatment methods have been unsuccessful, strict bed rest with a minimum of sensory stimulation is recommended: a bed with cot-sides in a single darkened room with only basic amenities and no books, television or visitors. The restrictions on activities and other amenities are systematically lifted as the seizure-free intervals lengthen, but are reimposed in reverse order should relapse occur. In resistant patients this simple behavioural approach can lead to a dramatic reduction or even abolition of seizures, but must be followed by continued supportive psychotherapy and work with key relatives.

The long-term outcome for pseudoepileptic seizures is still subject to debate and disagreement. Reported improvement rates have varied from 30–80% (Lesser, 1985). The widely differing views about progress probably reflect the nature of the unit the patient is referred to and the programme of management used. Having had personal experience of the management of a substantial number of such patients, it is my view that the majority respond to a carefully structured programme of psychological management. However, some may continue to react at times of stress with occasional clusters of pseudoepileptic seizures. A few remain a chronic problem.

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Hysteria, Play-acting and Courage

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Levi-Strauss (1977) describes what happened to a child, in a primitive tribe, accused of sorcery–a crime punishable by death. The child invented more and more complex stories by way of self-justification. In the end his accusers believed his outlandish explanation because "the choice is not between this system and another but between this system and no system at all – chaos." The child's plight can represent the close affinity between magic and hysteria; his accusers might represent those contemporary psychiatrists who would prefer any system of classification of hysteria, however absurd, to no system at all (Sheehan & Sheehan, 1982*a*, *b*; DSM–III, 1980).

Commenting on similar issues, Mayou (1984) explained how lack of agreement and precise definitions cause great difficulties for psychiatry. Underlying the imprecision is a general failure to appreciate the importance of the fact that the different sicknesses to be classified are in different universes of discourse, and that any attempt to develop one system to embrace processes, things, and states of affairs is bound to fail (Taylor, 1979, 1982). Within this chaotic classification hysteria holds pride of place for ambiguity. Doctors wishing to dispose of the problem by abandoning the diagnosis, however, are mortified by the thought of how otherwise to describe their next patient with those symptoms.

This mortification will persist until the concept of 'hysteria' is replaced by a better explanation. Some people believe that this will come from the biological sciences, others that it will come from psychosocial case analysis and epidemiological techniques. Some believe that if sickness is not physical it must be psychological, or again that if sickness is not physical it is not our business. I believe that hysteria is generated as a defence mechanism and propagated as a consensual phenomenon, as is magic. Like magic, it has no real properties beyond belief. Granted that it is a belief, the physiological and psychological characteristics of the believer are probably irrelevant. But beliefs, convictions and avowals, however, are powerful phenomena.

Hysteria persistently imposes itself upon medicine by threatening the implicit contract of trust between doctors and their patients. A group of people, referred to by Bayliss (1984) as 'the deceivers', pose problems not just of diagnosis but also of management, resource allocation and ethics. Having mentioned the Munchausen syndrome, illness by proxy and factitious disorders, but having studiously avoided the word 'hysteria', Bayliss remarked that "these patients are seldom malingerers" and that