Leading Article

Non-epileptic attack disorder

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It has long been recognized that some episodic behavioural or experiential phenomena resembling epileptic seizures have causes other than epilepsy. Only since the introduction of intensive monitoring has it been realized that non-epileptic seizures account for some 10–20% of tertiary referrals for refractory epilepsy.¹ Differentiation between epileptic and non-epileptic attacks presents both conceptual and practical problems. The distinction was acknowledged but blurred by the 19th century concept of 'hystero-epilepsy' and had little objective basis until the discovery of the electroencephalogram (EEG) provided rational grounds for defining epilepsy in terms of characteristically disordered cerebral physiology.

Non-epileptic attacks may be due to cerebral dysfunction, as syncope or Gilles de la Tourette's syndrome, or may have psychological origins ranging from conscious simulation to hysterical conversion. The terminology of non-epileptic seizures is contentious. The term 'hysterical' implies a particular pathogenesis and prejudices proper psychiatric diagnosis. 'Pseudoseizures' has been used loosely to cover various attacks which may be mistaken for epilepsy. This term can be seen as pejorative or trivializing the problem; moreover, it denies the fact that the diagnosis may be iatrogenic: it is the physician, not the patient who misdiagnoses syncope, panic attacks or abreaction as epilepsy. A further source of terminological and diagnostic confusion is that both epileptic and non-epileptic seizures may be 'psychogenic': psychological factors can precipitate epileptic seizures, non-epileptic organic attacks such as syncope, and acute psychiatric symptoms as anxiety or abreaction. In the absence of any more satisfactory and agreed terminology, the term, 'non-epileptic seizures', is to be preferred to pseudoseizures, and 'psychogenic' seizures are taken to be non-epileptic attacks caused by an action of the mind, unless specifically designated 'psychogenic epileptic seizures'.

The diagnostic approach should focus on both

the seizures and their psychosocial context. Adequate history and physical examination, possibly observation of an attack, and long-term ECG monitoring where appropriate, generally suffice to identify non-epileptic seizures of organic origin. Greater difficulties arise in the differential diagnosis of psychogenic seizures, which will be considered further here. Intensive EEG and video monitoring has helped to reveal the varied ictal symptomatology of both epileptic and psychogenic seizures. Exceptions are known to almost every typical diagnostic sign: incontinence and tongue-biting can sometimes occur in psychogenic seizures, and opisthotonus and screaming in epilepsy. Striking and largely unexplained differences exist between series; in some centres most non-epileptic seizures are convulsive and epilepsy rare, in others a majority of patients with non-epileptic attacks also have epilepsy and the ictal symptoms are more subtle² often resembling the initial events of their habitual epileptic attacks.³ Some of the more consistent signs are noted below.

Generalized seizures present least diagnostic difficulty. It is almost impossible to simulate ictal clonus convincingly: the flapping movements of a psychogenic tonic-clonic seizure lack the characteristic alternating brisk contraction and relaxation, the jerks do not usually become gradually slower over the course of the attack, and the rhythm intermittently falters. Flailing of the limbs and side to side rocking of the head are common.⁴ Physiologically improbable postures and combinations of movements may occur, for instance of one arm and the contralateral leg; when a jerking limb is restrained the pattern of movement changes. If seated, the patient often stiffens, extends the legs and slides without injury to the floor. A characteristic pattern of back arching with pelvic thrusting occurs in abreactive attacks of sexually abused females⁵ but also in some epileptic seizures of medial frontal origin. Preserved consciousness may be evident, if the patient can communicate or subsequently recalls ictal events, spontaneously or under hypnosis.⁶ However, clonus with intact consciousness can occur, albeit rarely, in epilepsy.⁷ Passive opening of the eyes of a patient apparently unconscious after a pseudoseizure is usually

resisted, and if the subject is placed on one side the eyes are directed towards the floor; if the patient is turned onto the other side the eye deviation is reversed.⁸ Extensor plantar responses and reduced corneal reflex can occur in psychogenic seizures.⁹ Injury from falling and biting of the side of the tongue or buccal mucosa are uncommon in non-epileptic seizures, but unusual injuries, biting of hands, lips or tip of the tongue, or indeed frank automutilation may occur. Damage to property and injury to bystanders are more common in non-epileptic seizures. Incontinence is not uncommon in deliberately simulated epilepsy.

Psychogenic convulsive status epilepticus is now more common in tertiary referral centres than epileptic status. In addition to features which characterize non-epileptic convulsions a clue is provided by the lack of cyanosis and usually rapid and full recovery of consciousness between attacks, unless the picture has been confused by premature administration of antiepileptic drugs.

Apparent partial seizures of psychogenic origin may show symptoms which fortuitously resemble those of epilepsy, for instance fear, pallor and tachycardia in a panic attack, and it should be noted that associated sensory and motor symptoms due to overbreathing may be unilateral.¹⁰ However, some psychogenic seizures clearly involve simulation of epileptic symptomatology, most convincingly in patients who have experienced epilepsy in themselves or others. There are, however, often clinical features which lead to a suspicion of a non-epileptic origin. Typically, the initial events ('aura') of a complex partial seizure are brief, progressive and irresistible, and recovery of consciousness gradual with confusion and malaise. By contrast, the patient may announce an imminent non-epileptic seizure and 'fight against it' over several minutes, yet recover abruptly with full consciousness and possible emotional distress. Patients' accounts of psychogenic ictal symptoms tend to be vague and inconsistent; recollection of events during automatisms suggests psychogenic attacks, but can occur in epilepsy.¹¹ Prolonged attacks of more than 5 minutes duration are more likely to be non-epileptic and may include periods of inaccessibility, akinesia and atonia.

A knowledge of the range of epileptic ictal phenomenology is essential in assessing the seizure pattern. In particular the bizarre behaviour associated with seizures of orbital or medial frontal origin should be recognized. Such seizures are typically brief with rapid recovery, and frequent; they often occur during sleep and in clusters sometimes amounting to status epilepticus. Amongst the curious activities are bimanual-bipedal movements, hand clapping, pedalling and running; aggressive or scatological utterances may occur and pelvic thrusting or other overtly sexual

behaviour is sometimes seen. Several of these features are common to frontal and psychogenic seizures. The more reliable distinguishing characteristics are the relative brevity (typically less than one minute), nocturnal occurrence and tendency for the patient to assume a prone position in frontal seizures, and tonic abduction of the upper limbs with supplementary motor involvement.^{12,13}

Of the available diagnostic aids the ictal EEG is of greatest but limited value. The presence of epileptiform discharges in the interictal EEG is of marginal relevance in diagnosis of non-epileptic seizures, as in many patients these coexist with epilepsy. Overinterpretation of normal, or irrelevant spiky EEG transients^{14,15} can lead non-epileptic attacks to be wrongly designated epileptic. Conversely, 50% of single waking EEGs in people with epilepsy are normal,¹⁶ and in 8% of subjects even repeated wake and sleep recordings fail to show discharges in the interictal state.¹⁷ However, epilepsy is unlikely if no spikes occur during an 8 hour recording which includes the first hour of nocturnal sleep.¹

A technically adequate EEG during a convulsive epileptic seizure will invariably contain epileptiform activity, and even if this is obscured by artifact an early post-ictal record (or interictal record in status) is likely to show slowing. Absences too have a consistent ictal EEG signature of generalized spike-wave activity. An experienced observer will rarely need an ictal EEG to identify psychogenic convulsive seizures, but a record should be obtained if possible for purposes of documentation and to convince others. However, during brief partial seizures involving circumscribed brain regions EEG changes may be subtle, non-epileptiform or absent. Simple partial seizures with viscerosensory or psychic symptomatology are particularly likely to occur without EEG change. Thus if a patient with experience of partial epilepsy claims to experience a rising epigastric sensation and a feeling of fear there may be no observational means, either clinical or electroencephalographic, of determining whether the symptoms are epileptic. Complex partial seizures are more likely to be associated with ictal EEG changes, but these may be unremarkable, a reduction in amplitude, or the appearance of bitemporal theta activity. EEG assessment of frontal seizures presents especial difficulty. Often the interictal record is normal and the seizure may be accompanied by frontal slow activity which is readily mistaken for artifact. The occurrence of a seizure from sleep (as is common in frontal lobe epilepsy) during EEG telemetry provides useful diagnostic information: if seizure onset precedes electrographic evidence of waking the attack may be presumed epileptic. As the significance of an apparently negative ictal EEG depends on the technical

quality of the record and the clinical phenomenology, video and EEG telemetry should be used where possible.¹⁸ Ambulatory monitoring with recorders of restricted technical specification and without accurate seizure documentation may suffice to establish that a seizure was epileptic, but not to prove the converse. Where the differential diagnosis includes cardiac dysrhythmia, combined EEG and electrocardiogram (ECG) monitoring is advisable, as the EEG will change during cardiogenic seizures and cardiac changes including asystole can occur in epilepsy.

An increase in serum prolactin levels above 500 IU/ml occurs 15–20 mins after electroconvulsive therapy and after some 80% of tonic-clonic attacks^{19–21} and many complex partial seizures with automatism, but rarely in simple partial seizures.^{22,23} Sperling *et al.*²⁴ from a study of patients with intracerebral electrodes concluded that partial seizures with limbic discharges (either simple or complex) were associated with prolactin elevation. Increased prolactin after complex partial seizures of frontal origin appears to be rare, although this is disputed.^{25,26} Reliable prolactin estimations can be made from capillary blood collected on filter paper and stored for up to a week, a technique suitable for use in outpatients.²⁷ The specificity of this test may, however, be questioned as marked elevation can sometimes occur with emotional stress.²⁸

It may appear perverse to focus on the phenomenology rather than the psychiatric context of psychogenic seizures. However, psychiatric morbidity is common among patients with suspected

non-epileptic attack disorder including those in whom the diagnosis proves incorrect. Their problems may arise in part from the psychosocial consequences of therapy-resistant seizures, and rejection when the reality of the attacks is questioned by their physicians and families. Thus the presence of psychiatric disorder and evidence that seizures are stress-related are poor guides to diagnosis. The preferred strategy is first to establish that the seizures are non-epileptic and then to address the psychiatric causes and their treatment. When patients with non-epileptic seizures are compared with those with undoubted epilepsy, clear group differences emerge. Typical features include: a current affective disorder, family and personal history of psychiatric disorder and attempted suicide, sexual maladjustment,²⁹ and high Minnesota Multiphasic Personality Inventory (MMPI) scores on hypochondriasis, hysteria and schizophrenia scales.³⁰ Tests of suggestibility, attempting to induce a seizure by intravenous saline injection, for instance,³¹ are of doubtful diagnostic value as they do not address the issue of whether the habitual seizures are psychogenic.

Often, the differential diagnosis of non-epileptic seizures presents little difficulty to the physician familiar with the phenomenology of epilepsy; sometimes the issue remains unresolved even after intensive monitoring, prolactin estimations and psychiatric assessment. However, a greater error than misdiagnosis is to regard non-epileptic seizures as unreal, and less disabling or demanding of care than epilepsy.

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