Welcome to the tenth in a series of articles in ACNR exploring clinical dilemmas in neuropsychiatry. In this series of articles we have asked neurologists and psychiatrists working at the interface of those two specialties to write short pieces in response to everyday case-based clinical dilemmas. We have asked the authors to use evidence but were also interested in their own personal views on topics. We would welcome feedback on these articles, particularly from readers with an alternative viewpoint.

How do you identify and manage convulsive psychogenic nonepileptic seizures presenting acutely to casualty?

Case

A 35-year-old man presented to casualty, having had a prolonged seizure. He had a diagnosis of epilepsy and was taking carbamazepine. He had further seizures in casualty, each with several minutes of generalised movements, followed by a similar period of unresponsiveness. On the last occasion the motor manifestations of the seizure went on for 12 minutes, and intravenous diazepam was given, following which the movements settled, and he appeared to sleep. During all this, his oxygen saturation remained at near 100%. Staff who witnessed the attacks expressed the view that they might have been psychogenic rather than epileptic, as they felt there was a degree of responsiveness during them. What clinical clues help you identify psychogenic seizures? How do you manage the situation acutely? What do you say to patients and relatives? Where do you refer, and in what terms?

Psychogenic nonepileptic seizures (PNES, pseudoseizures) have an incidence of 44/100,000/year,\textsuperscript{1,2} up to one eighth that of epilepsy. There are a number of reasons why convulsive PNES might be more likely to present to casualty than epileptic seizures. They last longer, they are often serial, and the movements are often more impressive,\textsuperscript{3} all producing a picture that can be quite alarming to carers and relatives. Attending paramedics are likely to have protocols requiring them to treat long or recurring seizures with benzodiazepines. These drugs disinhibit, and may make progression to prolonged serial PNES or pseudostatus more likely.

It has been practice in the past to disregard the diagnostic problem and treat all such cases as epilepsy on the grounds of safety. This is in many ways understandable: the clinical risk of treating pseudoseizures as epilepsy is rightly perceived as being less than that of making the opposite error. However, neither choice is risk-free: treating pseudostatus as status epilepticus carries a significant risk of iatrogenic death.\textsuperscript{4} Treatment with intravenous benzodiazepines can depress respiration sufficiently to require respiratory support, and patients occasionally end up in ITU for this reason. These risks can be kept to a minimum if the focus is kept firmly on the patient’s life support systems. Is the patient breathing? Are his pulse and organs? Are blood gases or oxygen saturation consistent with adequate ventilation or hyperventilation? If the answer to these questions is yes, then the emergency doctor should think very carefully indeed before giving treatments that carry risk.

This patient had a diagnosis of epilepsy. In the past, it was thought that most patients with PNES also had epilepsy. Recent consensus is that only 10-15% have a dual diagnosis,\textsuperscript{5} with the exception that the rate is probably 30% in the learning disabled.\textsuperscript{6} In the the emergency situation, it is difficult to meaningfully evaluate a past diagnosis of epilepsy, and it is probably better to concentrate on the identity of the attacks that are happening there and then.

Clinically the great majority of PNES fall into two clinical patterns.\textsuperscript{7} The majority are convulsive, with unresponsiveness accompanied by generalised movements, eyes usually closed. The movements have been described in a number of ways, but are essentially tremors, either high frequency and low amplitude, or
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Low frequency and high amplitude. The latter may look quite dramatic, and in a few patients develop into actual ‘thrashing’ movements. A large majority of patients have fall-down-lie-still attacks (sometimes called ‘swoon’ attacks) during which the eyes are usually closed and the patient is unresponsive. For obvious reasons, these do not fall into the differential diagnosis of tonic clonic convolution (rather than vasovagal syncope, cardiac syncope, or decaesthesia). Nonetheless, some simple diagnostic clues are helpful, focusing initially on life support. Patients do not breathe during the tonic and clonic phases of a tonic clonic convolution, whereas hyperventilation is usual during a PNES. Therefore, if a patient has been in what appears to be a convulsive seizure for several minutes with normal oxygen levels (or saturations), particularly if accompanied by low CO₂, then it becomes difficult to sustain the diagnosis of tonic clonic convolution. The longer the duration of convulsive movements before measurement of a normal O₂ saturation, the more likely the diagnosis is to be PNES. Oxygen saturations should not drop below normal levels during PNES. In tandem with this, a ‘grey’ colour, or cyanosis indicates an epileptic seizure (though patience it does not occur in all cases, especially if the seizure is short). Impressive rubefaction usually means hyperventilation and PNES. Clear observation of breathing may not be all that easy, but should be attempted. Rhythmic grunts or guttural noises with no normal breathing pattern suggests tonic clonic seizure. Rapid but otherwise normal respiration suggests PNES.

This particular patient had a sinus tachycardia of 140, with a blood pressure of 180/100. Both sinus tachycardia and mildly raised blood pressure are usual during PNES of convulsive type. Oxygen saturations were over 97% throughout his attacks, even when the convulsive movements had been going on for more than 10 minutes. This would be difficult to reconcile with a diagnosis of tonic clonic seizure.

Other clinical features of PNES can be of some help, though all have an error rate. PNES are generally much longer than epileptic seizures. Side-to-side head movement is fairly common in PNES, not usually seen during tonic clonic convulsions. PNES do not usually have a tonic phase, and similarly tonic posturing of limbs is uncommon. Signs of emotional distress are common after PNES, uncommon after epileptic seizures. Resistance to forced eye-opening is highly suggestive of PNES. However, tolerance of painful stimuli may be surprisingly great, so this test is of limited use. Similarly, plantar responses may be difficult to induce, and the presence of a relative in the room may allow a more realistic evaluation of whether the attacks are like those previously documented. The use of labels for the attacks (e.g. ‘This patient presented today with a tonic-clonic seizure...’) is much less helpful. Clearly relevant measurements, such as oxygen saturations, should also be included.

In this case, the patient eventually underwent video-EEG monitoring, which confirmed the diagnosis of PNES. There was no evidence of epilepsy (only one attack type clinically; no history suggestive of tonic clonic seizures, no interictal epileptiform EEG abnormality over two days of video EEG monitoring). The diagnosis of PNES was explained to him, carbamazepine was withdrawn without incident, and he was referred for psychological intervention.