SHORT REPORT

Painful generalised clonic and tonic-clonic seizures with retained consciousness

William L Bell, Thaddeus S Walczak, Cheolsu Shin, Rodney A Radtke

Abstract

Two patients in whom consciousness and memory were retained during bilateral clonic or tonic-clonic seizures are reported on, and three patients reported on previously are reviewed. Ictal semiology differed from myoclonic and supplementary motor seizures, which are other seizure types characterised by bilateral motor movements and retained awareness. In the two new patients ictal pain was a prominent feature. It is proposed that propagation of seizure activity may be confined to the sensorimotor areas bilaterally while sparing the neural structures involved in maintaining consciousness and in processing language and memory. This unusual type of seizure may be misdiagnosed as a pseudoseizure. Detailed description of the ictal events and further laboratory evaluation including video-EEG monitoring may be necessary to make the distinction.

(J Neurol Neurosurg Psychiatry 1997;63:792–795)

Keywords: consciousness; pain; epilepsy; electroencephalography

It is widely accepted that total amnesia and loss of consciousness occur during generalised tonic-clonic seizures.1,2 Indeed, retention of memory, responsiveness, and the ability to speak during generalised seizures suggest a diagnosis of pseudoseizures.3–8 We report on two patients with ictal pain and retained consciousness during bilateral motor seizures. In one of these, both the electrographic and clinical events recorded during the seizure were otherwise typical of those seen during generalised tonic-clonic seizures. These patients, with the three reported previously, showed certain similarities that suggested a mechanism for this unusual type of seizure.

Case reports

CASE 1

A 44 year old right handed man had a 12 year history of episodes that began with an aura consisting of a total body “flash”, followed by left arm jerking and then jerking of all limbs. The patient claimed to be conscious throughout the episode, often praying aloud for the seizure to pass. He complained bitterly of severe “aching” pain during these episodes, but was vague as to the location, timing, and character of the pain. He did not have incontinence or tongue biting. The spells became more frequent despite therapeutic doses of carbamazepine and valproate. Because of the apparent preservation of consciousness during bilateral clonic activity, the episodes were initially diagnosed as pseudoseizures.

Neurological examination was normal except for a mild left upper limb tremor and pronator drift. A sleep deprived EEG showed mild diffuse slowing and occasional sharp transients over the right frontotemporal region, but no definite epileptiform discharges. Five seizures were recorded during continuous video-EEG monitoring with the typical aura beginning 20 to 30 seconds before the motor seizure. Two seizures started with forced version of the head to the left along with clonic left arm jerking, two seizures started with left leg jerking, and in one seizure the onset was unclear. Except for the head version, there were few tonic components to the seizures. In four seizures, clonic jerks involved the remainder of the left side before involving the entire right side and the head. In one seizure, clonic jerks remained confined to both lower limbs. The jerking was asynchronous and usually asymmetric as well, having the appearance of the clonic phase of a generalised tonic-clonic seizure rather than a myoclonic seizure. The patient seemed to breathe at his usual rate during the bilateral clonic movements. In two seizures, he prayed aloud during the bilateral clonic jerks (fig 1); in one seizure, he conversed with an observer during bilateral clonic activity complaining of the pain he was experiencing. At various times during and sometimes after the seizures his speech became dysarthric. Postictally, he was immediately oriented, but had a transient left hemiparesis. In one seizure, the patient could recall the commands given during the bilateral clonic jerks. The seizures lasted from 50 to 85 seconds. Two seconds after the onset of the aura, scalp EEG showed rhythmic right parieto-occipital delta that spread over both hemispheres as bilateral clonic activity developed (fig 1).
Brain CT showed a right superior frontal lobe hypodensity. Stereotaxic biopsy disclosed an anaplastic astrocytoma. Despite radiation and chemotherapy, the tumour progressed and the patient died two years after the initial surgery.

CASE 2
A 16 year old ambidextrous man had medically intractable seizures of seven years' duration. All seizures began with dizziness followed by a sensation in his left arm described as “losing track of where my arm is in relation to the rest of my body.” Jerking beginning in the left arm would spread to all limbs. During the bilateral motor attacks, the patient experienced severe pain. He stated that he could recall material presented during events; this was confirmed by his mother.

Neurological examination was normal except for an increased left biceps reflex. A sleep deprived EEG disclosed mild background slowing and frequent spikes over the right centroparietal region. During video-EEG monitoring two seizures began with the usual aura of dizziness. Clonic jerking started in the left arm that then assumed a tonic posture with the arm forcibly flexed and abducted, with superimposed clonic movements. This was followed by similar posturing and clonic movements in the right arm and then by sustained bilaterally symmetric tonic-clonic movements of all limbs. Occasional breaths occurred independently of the tonic-clonic jerking. Both seizures lasted about 75 seconds. Verbal memory items were presented during the tonic and clonic phases of both seizures. The patient correctly recalled all information presented during the first seizure (fig 2). He correctly recalled a nursery rhyme given during the second seizure, but said that the pain during the seizure had been distracting. With the onset of clonic left arm jerking, the EEG showed generalised rhythmic theta followed by diffuse spike and wave as the generalised motor seizure continued (fig 2).
Brain MRI disclosed an area of increased T2 signal in the right centroparietal region without mass effect or oedema. Complete excision of a discrete lesion immediately posterior to the primary sensory cortex—a low grade astrocytoma—abolished the seizures over nine years of follow up.

Discussion

These cases illustrate that consciousness may be preserved during bilateral motor seizures. It could be argued that a certain degree of impaired consciousness was present during these seizures and was simply not detected. However, the detection of subtle alteration of consciousness is not a simple task. The concept of consciousness is difficult to define and previous studies have dealt with measurable aspects of consciousness such as perception, memory, affect, and voluntary movements. As a practical matter, the International Classification of Seizures suggests that an operational definition of consciousness should include preservation of perception, responsiveness, and memory. Thus patients are usually tested for verbal responsiveness and memory during seizures. Even then, responsiveness can be altered by aphasia or paralysis, both possible consequences of partial seizures without impairment of consciousness.

The table shows the features of the two patients reported on here and three other patients described previously. After reviewing serial photographs of the seizure and ictal EEG in their patient, Ajmone-Marsan and Ralston concluded that the seizure started in the right rolandic area and spread via the corpus callosum to the left rolandic area. The retention of consciousness and the ability to converse appropriately suggested that the seizure remained confined to these regions rather than propagating more diffusely to areas responsible for speech and memory. Four of the five patients in the table had seizure foci documented in the right or verbally nondominant hemisphere. Whether this may have contributed to their ability to maintain verbal responsiveness is unclear; in retained consciousness during supplementary motor seizures the side of the seizure focus does not seem to be a factor.

Seizures in patient 2 differed from those in patient 1 in that tonic posturing was followed by prolonged bilateral clonic movements giving the seizures the clinical and electrographic appearance of secondarily generalised tonic-clonic seizures. In neither of our patients did the events have the clinical or EEG appearance of myoclonic seizures often associated with generalised spike and wave or polyspike and wave discharges on EEG. Nor did these seizures have the clinical appearance of supplementary motor area seizures during which consciousness may be preserved. In the second, tonic posturing of the limbs was the most prominent component and the seizures were brief, usually lasting 10 to 40 seconds. Our patients differed in that the tonic posturing was brief, clonic movements were prominent, and the seizures were longer, lasting 50 to 90 seconds. Nevertheless, without invasive recording, we cannot rule out the possibility that these seizures originated in the supplementary motor area.

A review of the table suggests a mechanism for these unusual seizures. In all patients, the seizure focus was extratemporal. Patients 1 and 2 both had known lesions involving or close to the motor cortex and all three of the previously reported cases indicate a seizure focus in a central brain region. Therefore, a possible mechanism is that these seizures are generally limited to extratemporal regions containing the motor pathways bilaterally, sparing the regions essential for maintaining consciousness, language, and memory processing.

The ability to continue conversation and retain memory, the lack of incontinence, and persistent respirations during bilateral clonic and tonic-clonic movements suggest pseudo-seizures and, in fact, our first patient was referred with a diagnosis of probable pseudo-seizures. Other features suggest the correct diagnosis. Rather than exhibiting the lack of concern often noted in pseudoseizures, our patients complained of severe pain during the clonic movements. Because of the pain, each strongly resisted attempts to taper anticonvulsants to hasten recording of seizures.

Painful seizures have been described, but are uncommon and thought to be more often related to involvement of the somatosensory cortex than simply the conscious awareness of painful involuntary motor movements. Furthermore, pain is often part of the somatosensory aura preceding the motor manifestations. In our patients, the pain occurred during motor movements, but seemed excessive given the intensity of the movements.

Therefore, whereas the pain was timed most closely with the limb movements, dysaesthesia related to involvement of the primary sensory (SI) or secondary sensory (SII) cortex is an alternative and perhaps a more likely mechanism. Unfortunately, our patients gave only vague descriptions of their pain. An association with pain was not noted in the previously reported patients with bilateral motor movements and retained consciousness and may not be a constant feature.

In summary, we describe painful seizures in which consciousness was preserved throughout bilateral clonic or tonic-clonic motor seizures documented by simultaneous video-EEG monitoring. We think that this seizure type results from the spread of the seizure activity to

---

Table 1  Electroclinical features of bilateral clonic and tonic-clonic seizures with retained consciousness

<table>
<thead>
<tr>
<th>Patient</th>
<th>Location of lesion</th>
<th>Interictal EEG</th>
<th>Ictal EEG onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ajmone-Marsan and Ralston(^{11})</td>
<td>Right frontal, paramedian</td>
<td>Bilateral posterior slowing</td>
<td>Right frontoparietal spikes, theta</td>
</tr>
<tr>
<td>Botes et al(^{20})</td>
<td>Right rolandic, paramedian</td>
<td>&quot;focal discharges&quot;</td>
<td>Generalised rhythmic theta</td>
</tr>
<tr>
<td>Weinberger and Lusino(^{11})</td>
<td>Central parasagittal, Right frontal</td>
<td>Diffuse slowing</td>
<td>Right parietal, rhythmic delta</td>
</tr>
<tr>
<td>Case 1</td>
<td>Right centroparietal</td>
<td>Right centroparietal spikes</td>
<td>Generalised rhythmic theta</td>
</tr>
<tr>
<td>Case 2</td>
<td>Right centroparietal</td>
<td>Not reported</td>
<td>Not reported</td>
</tr>
</tbody>
</table>
both hemispheres, yet is limited to sensorimotor pathways in the central brain regions, and sparing the neural structures involved in maintaining consciousness and in processing language and memory.

This work was presented in part at the 39th Meeting of the American Academy of Neurology, Chicago, IL, USA, 15 April 1989.

We thank Dr Jay Luther for referring patient 2 and Dr J Kiffin Penry for his constructive comments. Ms Gloria Rigsbee and Ms Sara Fernet typed the manuscript.


Painful generalised clonic and tonic-clonic seizures with retained consciousness

William L Bell, Thaddeus S Walczak, Cheolsu Shin and Rodney A Radtke

*J Neurol Neurosurg Psychiatry* 1997 63: 792-795
doi: 10.1136/jnnp.63.6.792

Updated information and services can be found at:
http://jnnp.bmj.com/content/63/6/792

**References**

This article cites 12 articles, 3 of which you can access for free at:
http://jnnp.bmj.com/content/63/6/792#BIBL

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Topic Collections**

Articles on similar topics can be found in the following collections

- Epilepsy and seizures (846)
- Pain (neurology) (763)

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/