

# Post-ictal rage and aggression: a video-EEG study

Alexei E. Yankovsky, Martin Veilleux, François Dubeau,  
Frederick Andermann

Montreal Neurological Hospital and Institute, McGill University, Montreal, Quebec, Canada

Received November 5, 2004; Accepted March 10, 2005

**ABSTRACT** – Post-ictal rage and aggression have been mentioned in the literature but have rarely been documented by video-EEG recording. We studied a patient with dramatic, episodic, seizure-related rage and violence. This mentally retarded man had a lifelong history of seizures. He developed increasing episodic rage and aggression. Caregivers were afraid of him, although there was no record of directed violence. In one of these episodes he fractured his tibia and fibula. Interictal discharges arose from both temporal areas independently. He had focal seizures with secondary generalization. Immediately after cessation of the ictal discharge he became greatly agitated, with undirected aggression, loud screaming, kicking and fighting against the restraints. A video sequence illustrates the behavior. Imaging studies showed bilateral, periventricular, nodular heterotopia in the lateral aspect of both temporal horns and the trigones. Increasingly frequent, severe bursts of rage and aggression were proven to be post-ictal. Documented attacks occurred while he was restrained and this may have been a factor in their severity. Such attacks however, have been described while he was not restrained and these increased in severity and frequency over time. Developmental abnormalities with periventricular, nodular heterotopia in the region of the trigones and inferomesial temporal areas are considered to be causally related to his retardation and epilepsy.

*[Published with video sequences]*

**Key words:** post-ictal aggression, video-EEG, periventricular nodular heterotopia, behavioral disorders

A link between epilepsy and aggression has been disproportionately emphasized. Aggression and violence in epilepsy can be associated with ictal, post-ictal and interictal states. Post-ictal rage and aggression have been described (Delgado-Escueta *et al.* 2002, Marsh and Krauss 2000, Gerard *et al.* 1998, Borum and Appelbaum 1996) but have only rarely been documented by video-EEG recording (Delgado-Escueta *et al.* 2002, Gerard *et al.* 1998). We describe a patient with epilepsy and severe outbursts of episodic agitation, aggression and rage, which presented both diagnostic and treatment problems.

## Case report

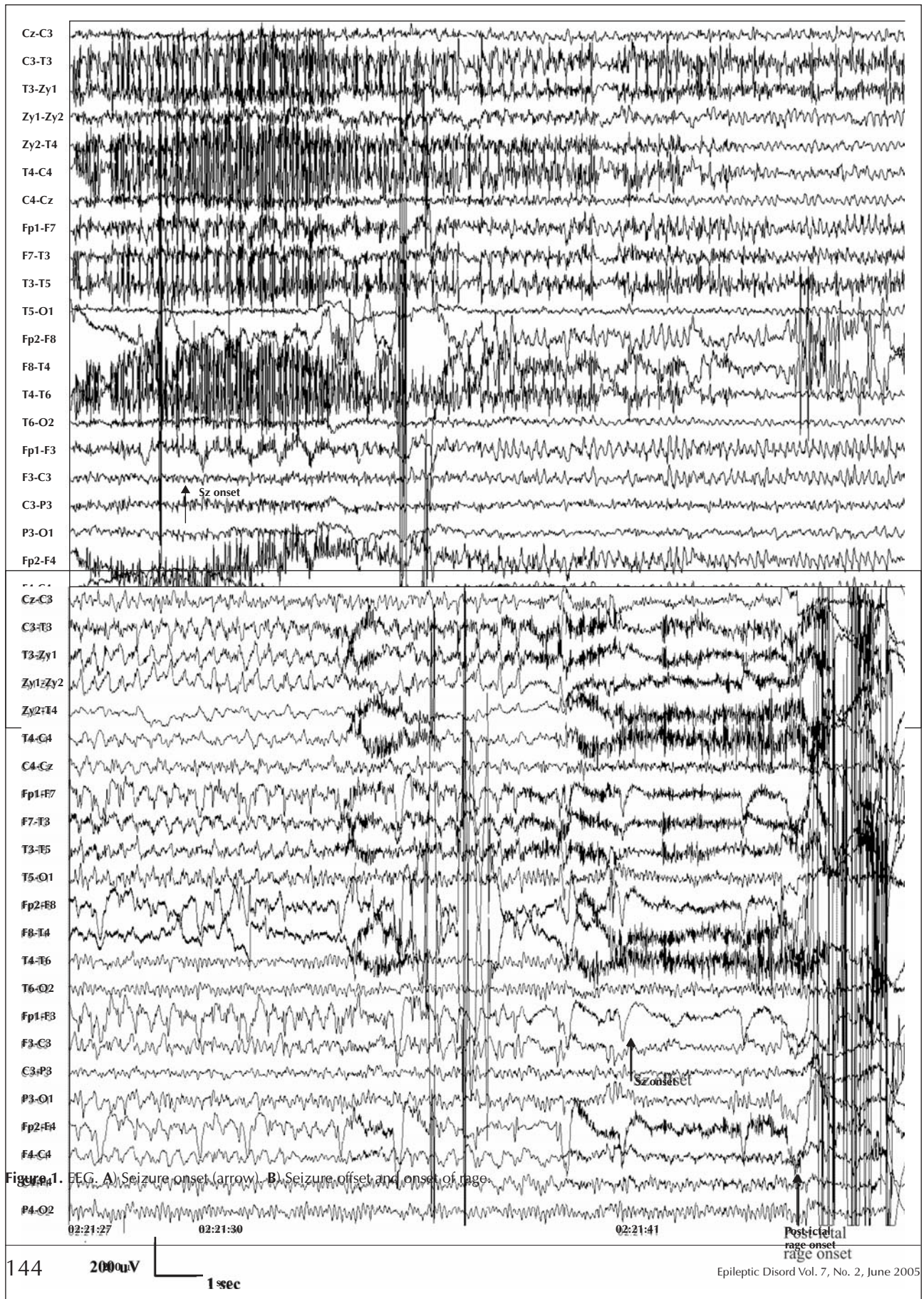
A 34 year-old male with a history of prenatal encephalopathy, severe mental retardation and lack of verbal communication was admitted to evaluate the nature of episodic agitation, aggression and rage by video-EEG recording and to document the relationship of these attacks to his seizures.

These episodes were unprovoked and occurred after periods of unresponsiveness and seizures lasting several minutes. During the attacks, he would suddenly hit furniture, objects or any person close to him, and scream and kick uncontrollably. His caregivers



### Correspondence:

F. Andermann,  
Montreal Neurological Hospital  
and Institute,  
McGill University,  
3801 University Street,  
H3A 2B4,  
Montreal,  
Quebec,  
Tel: (514) 398 1976  
Fax: (514) 398 1276  
<frederick.andermann@mcgill.ca>



were afraid of him. These outbursts lasted several minutes, abating gradually. Interictally, he was calm, with elements of obsessive compulsive behavior: he kept walking along a straight line and had to touch a wall in a repetitive fashion before continuing to walk.

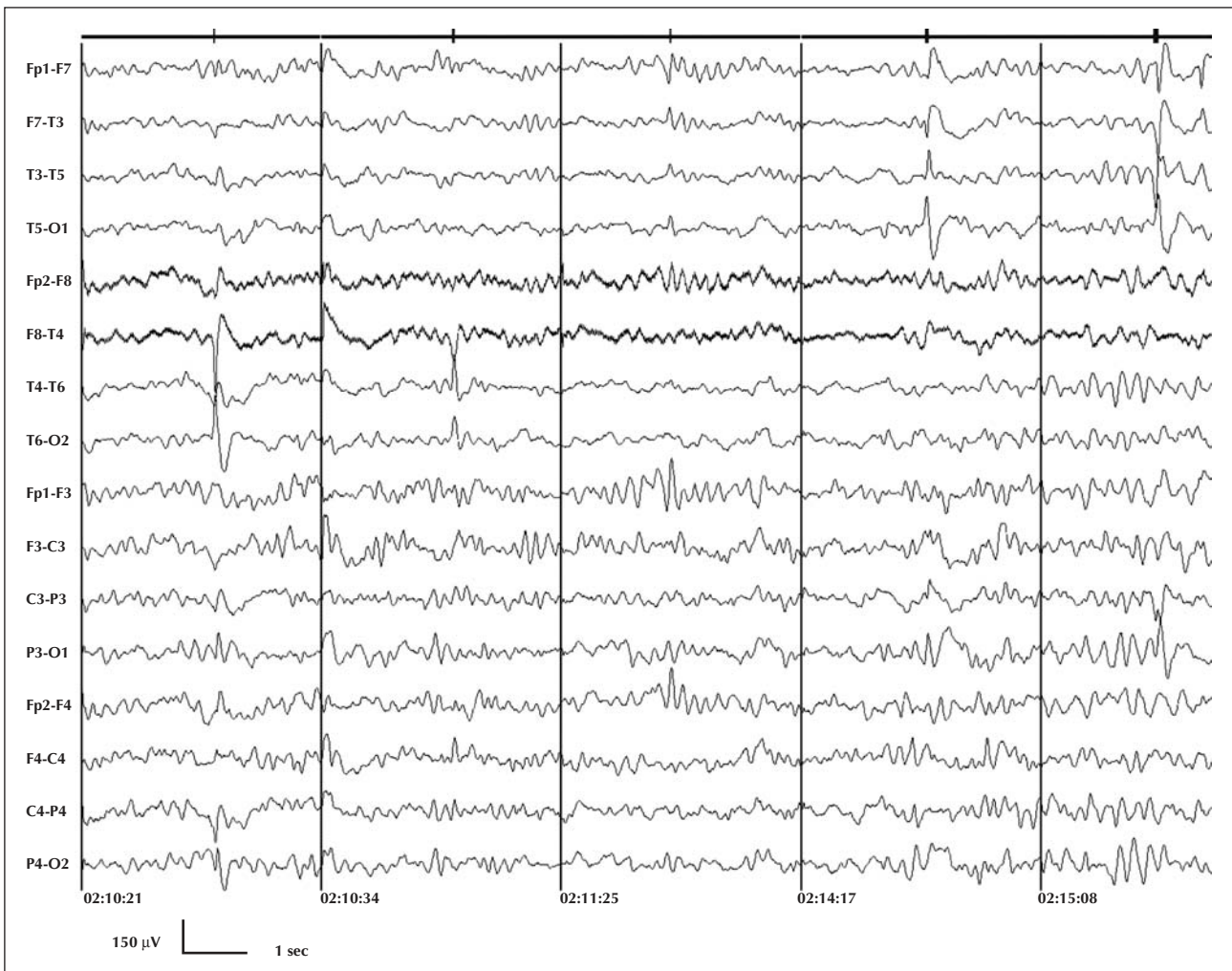
He had a history of lifelong, multiple seizure types: generalized tonic-clonic, partial complex and drop attacks. Attacks occurred up to 30 times per month, with an average of 10-15. He was treated with valproate, carbamazepine, oxcarbazepine, gabapentin and clobazam, without apparent effect on seizure frequency or severity. He also received risperidone at 2 mg a day, which initially reduced the severity of the episodic aggression, but this effect gradually disappeared. He had recently fractured his right tibia and fibula during one of his falls.

He had no family history of seizures. On physical examination he was short, with coarse facial features, low hair line, narrow forehead, thick eyebrows and lips, scoliosis

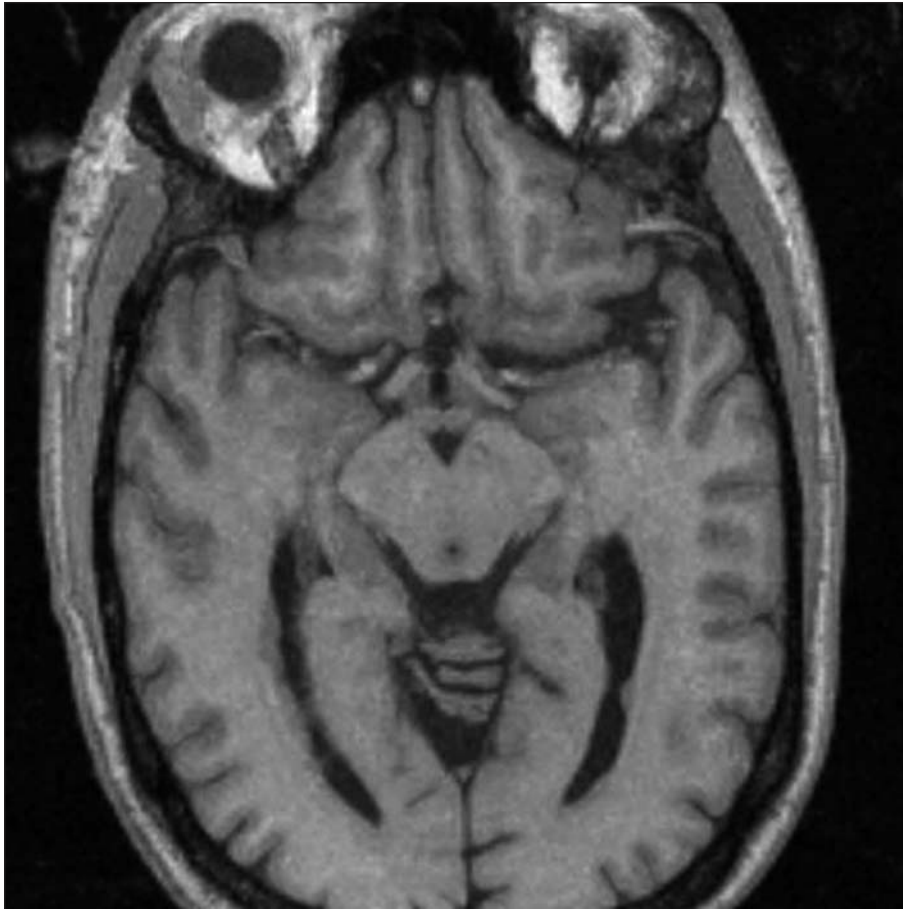
and increased muscle tone in all limbs. Chromosome studies were normal.

On video-EEG, a single seizure was recorded. The attack started with elevation and abduction of the right arm while the left arm appeared to be less mobile, slight head and eye deviation to the left side associated with staring and lip smacking (*see video sequence*). One minute and forty seconds later, the seizure generalized and stopped a minute later. Four to five seconds after cessation of his ictal discharges, he developed post-ictal rage and aggression, with screaming, kicking and fighting against the restraints. This behavior was as described by his caregivers. The localization of the ictal onset could not be clearly defined, but fast activity over the left frontal area occurred shortly after clinical onset (*figure 1a*). Seizure cessation can be clearly seen in *figure 1b*. Interictal, independent, bilateral, temporal epileptiform activity was recorded (*figure 2*).

His brain MRI showed bilateral periventricular nodular heterotopia in the lateral aspect of both temporal horns,



**Figure 2.** Inter-ictal EEG. Bitemporal independent interictal discharges.



**Figure 3.** T1 weighted MR image. Multiple noncontiguous flat and rounded periventricular nodules.

the trigone on the left, and in a series of contiguous nodules over the right trigone (*figure 3*). There was also diffuse cerebral and cerebellar atrophy. Adjustment of medication did not change the frequency or severity of the patient's attacks or his post-ictal aggressive behavior on follow-up examination 3 months later.

## Discussion

We documented severe post-ictal aggression and rage in a patient having complex partial seizures with secondary generalization. Peri-ictal aggression has been only rarely documented by video-EEG (Delgado-Escueta *et al.* 2002, Gerard *et al.* 1998).

### Ictal aggression

In a study of ictal aggression, only 13 out of 5400 patients were found to have aggressive behavior on closed-circuit television, and only 5 of them had a history of harming another person (Delgado-Escueta *et al.* 2002). Most showed spontaneous, non-directed, resistive stereotyped,

aggressive movements, with violence to property, shouting or spitting. It is highly unusual to observe well-organized purposeful and sustained aggressive acts.

### Acute post-ictal aggression

Two of these 13 patients had post-ictal aggression, which started several minutes after tonic-clonic seizures, and was documented by video-EEG. Both were struggling as they were restrained, and both had generalized, arrhythmic, low-voltage 1-2 Hz slowing of background activity during the aggression. Their post-ictal behavior was similar to that of our patient, whose aggression developed several seconds after cessation of the epileptic discharges, probably during post-ictal confusion when his awareness and responsiveness were impaired. Such confusional states generally occur after complex partial or generalized tonic-clonic seizures, and in most patients are relatively brief. During the period of impaired consciousness, behavior is relatively undirected, and resistive aggression occurs often when someone attempts to restrain or assist the patient during a seizure (Marsh and Krauss 2000). Our patient had resistive aggression during the episode docu-

mented by video, but a clear history of post-ictal aggressive behavior when he was not restrained was reported by his caregivers. Spontaneous (unrestrained), post-ictal aggression is an unusual feature and may add to the full spectrum of aggression in patients with epilepsy.

### Subacute, post-ictal aggression

Subacute post-ictal aggression has been reported, beginning hours to days after an acute confusional, post-ictal period (Gerard *et al.* 1998). It tends to recur and is more likely to occur after seizure clusters in males. Two of the patients reported became aggressive during post-ictal psychosis. Post-ictal psychotic symptoms may involve delusional ideation, hallucinations, thought disorder and manic or depressive mood changes (Savard *et al.* 1991). Psychosis can evolve during a period of post-ictal confusion, but more commonly emerges after a lucid interval (So *et al.* 1990), and lasts from less than a day up to several weeks (Lancman *et al.* 1994). It is impossible to conclude whether our patient had post-ictal confusion or psychosis. Both confusion and post-ictal psychosis are self-limiting conditions.

The variability in the timing of aggression according to the particular characteristics of the seizure (e.g. reaction to ictal fear, complex partial automatisms, post-ictal confusion or psychosis, interictal aggression) suggests different physiological mechanisms for the production of this behavior.

Aggressive behavior is controlled at multiple anatomical levels within the brain, including the hypothalamus, frontal cortical areas, and limbic system. Post-ictal aggression appears to originate from cortical structures involved in the ictal event. This could permit lower-(diencephalic) mediated behaviors to occur without the usual inhibitory influences of higher cortical structures (Engel 1991). By analogy, other post-ictal phenomena such as Todd's paresis or post-ictal amnesia may have similar mechanisms but this remains speculative (Morrell 1980).

In this patient the bilateral, periventricular, nodular heterotopia suggests a temporal origin for the intractable seizures, with involvement of the limbic structures. However, the first change on a scalp EEG occurred over the left

frontal area with bilateral frontal spread following. Lack of additional recorded attacks does not allow for more definite conclusions.

### Conclusions

In this patient, severe bursts of rage and aggression were proven to be post-ictal. Attacks occurred while he was restrained, but have been described while he was unrestrained too. Bilateral, temporal heterotopia and corresponding focal, temporal, interictal discharges were detected although exact seizure origin is impossible to determine given the single seizure recorded. The periventricular, nodular heterotopia are considered to be causally related to his epilepsy and retardation. □

### References

- Borum R, Appelbaum KL. Epilepsy, aggression, and criminal responsibility. *Psychiatr Serv* 1996; 47: 762-3.
- Delgado-Escueta AV, Mattson RH, King L, *et al.* The nature of aggression during epileptic seizures. *Epilepsy Behav* 2002; 3: 550-6.
- Engel Jr. J. Neurobiological evidence for epilepsy-induced interictal disturbances. In: Smith D, Treiman D, Trimble M, eds. *Advances in neurology*. New York, NY: Raven Press, 1991: 97-111.
- Gerard ME, Spitz MC, Towbin JA, *et al.* Subacute post-ictal aggression. *Neurology* 1998; 50: 384-8.
- Lancman ME, Craven WJ, Asconape JJ, *et al.* Clinical management of recurrent post-ictal psychosis. *J Epilepsy* 1994; 7: 47-51.
- Marsh L, Krauss GL. Aggression and violence in patients with epilepsy. *Epilepsy Behav* 2000; 1: 160-8.
- Morrell F. Memory loss as a Todd's paresis. *Epilepsia* 1980; 21: 18.
- Savard G, Andermann F, Olivier A, *et al.* Post-ictal psychosis after partial complex seizures: a multiple case study. *Epilepsia* 1991; 32: 225-31.
- So NK, Savard G, Andermann F, *et al.* Acute post-ictal psychosis: a stereo EEG study. *Epilepsia* 1990; 31: 188-93.