■ Clinical commentary

Epileptic Disord 2021; 23 (2): 419-425

Epileptic Disorders

Rage and aggressive behaviour in frontal lobe epilepsy: description of a case and review of the mechanisms of aggressive behaviour in epilepsy and dementia

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Received April 01, 2020;
Accepted December 19, 2020

ABSTRACT – The study of dementia and epilepsy may provide particular insight into behavioural alterations. We describe a rare case of ictal aggressive behaviour in a patient with focal epilepsy associated with a non-dominant dorso-lateral prefrontal lesion. During focal seizures, our patient showed intense agitation and anger, for a long time misinterpreted as psychogenic attacks, which disappeared after epilepsy surgery. The defined anatomical origin of such ictal emotional behaviour is not fully understood, however, the dorso-lateral prefrontal area appears to correlate less frequently with aggressiveness compared to the antero-mesial area. We describe the electroclinical data of our patient and provide a brief review of the mechanisms underlying aggressive conduct in epilepsy and dementia. An understanding of this mechanism could help to clarify the neural basis and treatment of violence associated with these and other neurological disorders. [*Published with video sequence*].

Key words: aggressiveness; prefrontal cortex; fronto-temporal dementia; focal cortical dysplasia; psychogenic non-epileptic seizures

VIDEO ONLINE

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doi:10.1684/epd.2021.1277

Disorders of social and emotional functioning are core features of a large number of acquired and developmental diseases, ranging from traumatic brain injury and neurodegenerative disease to schizophrenia and autism [1, 2]. Violence can occur in many pathological contexts, such as the behavioural variant of fronto-temporal lobe dementia (bvFTD), which presents socially inappropriate behaviour as one of the core diagnostic criteria [3] as well as epilepsy, in which aggressiveness is a rare but well-known manifestation [4]. Moreover, violence has been reported as interictal, ictal, and post-ictal behaviour [5]. Among the different forms of abnormal human conduct, aggressive behaviour causes major health and social problems with costly effects on individuals and society. The study of epilepsy provides a unique angle on the investigation of this behavioural alteration, as during seizures, networks may be evaluated through the progressive involvement of different brain structures [6-8]. The control of aggression requires a complex system of interconnected brain structures involved in various aspects of emotion. These areas are mainly located to limbic system structures, such as the amygdala, hypothalamus, hippocampus, and

the ventro-mesial prefrontal regions such as anterior cingulate and orbito-frontal cortices [9, 10]. In addition, recent data suggest the role of the insula, which is strongly involved in the error-monitoring network [11, 12]. All of these areas are frequently involved in epilepsy. The aim of the present study was to describe a rare case of ictal aggressive behaviour in a patient with a non-dominant dorso-lateral prefrontal lesion. We report the electroclinical data and provide a brief review of mechanisms underlying aggressive behaviour.

Case study

A 32-year-old right-handed woman was seen in the neurology clinic because of suspected psychogenic non-epileptic seizures (PNES). Two months before the neurological evaluation, she had begun experiencing daily episodes of "intense feeling of anger", which made her curse, as well as suffer from violent and bizarre head and limb movements. Her parents reported these episodes as socially devastating. A witness' home video (*video sequence 1*) showed one of these events, lasting less than one minute, during which the patient had preserved awareness.

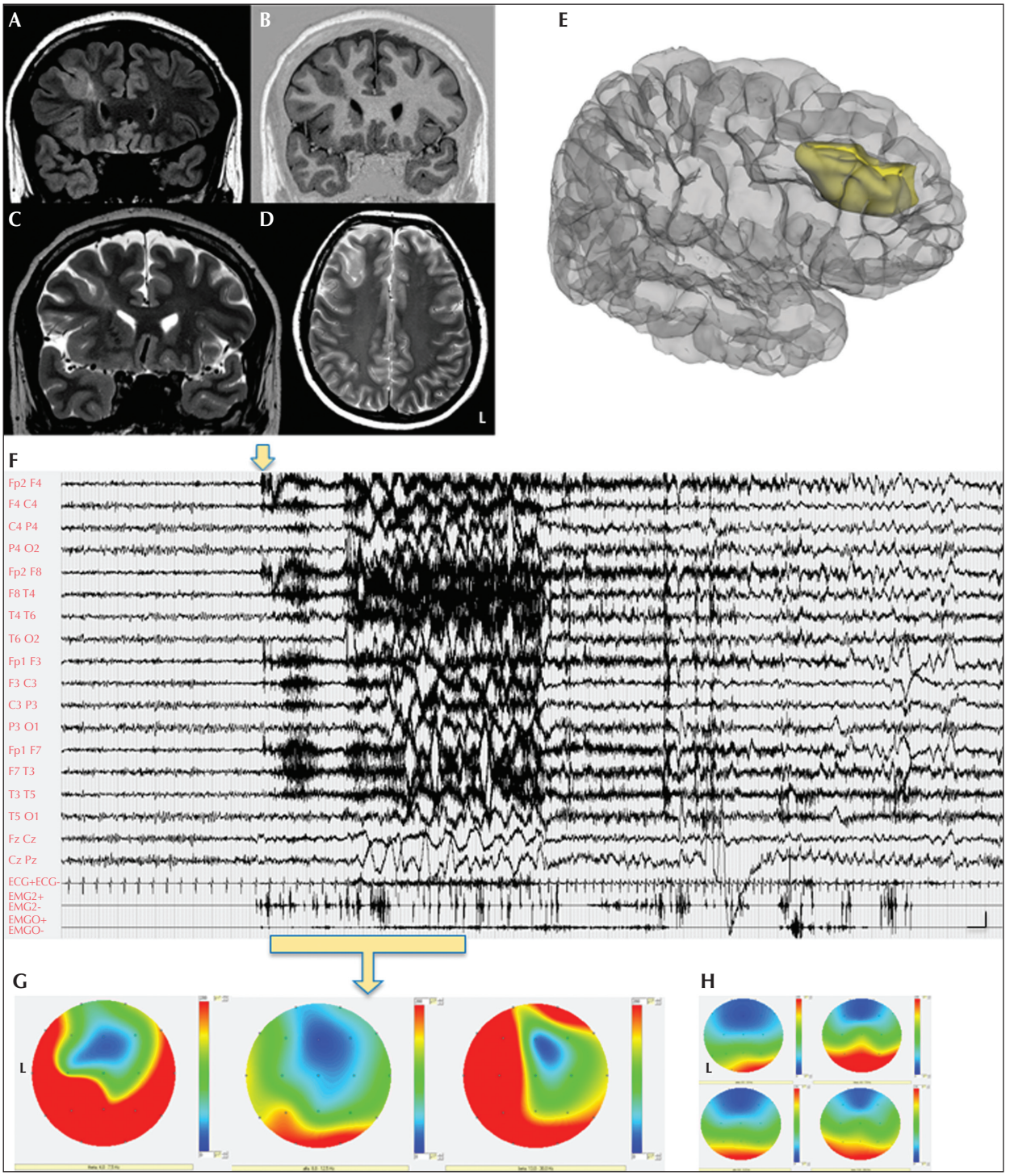
The patient was born spontaneously after an uneventful pregnancy at term, and family history for epilepsy was negative. At age six, she experienced several nocturnal focal to bilateral tonic–clonic seizures. An MRI study of the brain showed a right frontal cortical dysplasia (FCD). Psychiatric personal history was negative. The patient was started on carbamazepine and phenobarbital, after which tonic-clonic seizures were partially controlled (less than one nocturnal focal to bilateral tonic-clonic seizure per year). On admission, detailed neurological, psychiatric, and general physical examinations were normal. Long-term EEG monitoring captured several of these stereotyped self-limited events, characterized by paroxysmal rage and aggressive behaviour, all of which were reported by the patient (*video sequence 1*). MRI confirmed a focal cortical dysplasia in the right dorso-lateral prefrontal cortex, at the bottom of the sulcus between the superior and middle frontal gyri (*figure 1A-E*). A diagnosis of epileptic seizures arising from the right frontal lobe was made (*figure 1F*). Topographic EEG maps showed an asymmetrical distribution, on the frontal regions, of spectral power for distinct frequency bands during the first 10 seconds of seizure, in contrast to symmetrical distribution during the eyes-closed resting state (*figure G, H*). In combination with carbamazepine and phenobarbital, topiramate was administered, and the dosage was increased to 200 mg twice daily, resulting in a reduction in the frequency of events, from three times a day to approximately once every three days. During the following 14 months, the patient continued to have these episodes. Antiepileptic drugs, including levetiracetam, lacosamide and clonazepam, were periodically adjusted without a clinically significant effect. Surgical treatment was proposed, given medical intractability of disabling seizures. The surgically excised lesion was an FCD Type IIA. After six years of follow-up, the patient is seizure-free (Engel Class Ia).

Discussion

The neurology of violent behaviour has potential implications for clinical practice and raises social concerns. Abnormal behaviour can arise from both functional and structural brain abnormalities, that may be associated with epilepsy and dementia [13].

Ictal emotional changes are frequently observed during focal seizures that may involve the limbic system. Anxiety, fear or terror are frequently reported during temporal lobe seizures [14]; screaming, agitation, violent complex motor automatisms, but rarely hetero-aggressive behaviour such as biting, may also be observed as the possible emergence of innate motor patterns [15]. These symptoms can be misdiagnosed as paroxysmal psychiatric disturbances, resulting in therapeutic failures and persistence of disability [14]. Understanding of the neural basis of ictal abnormal affective and violent motor behaviours may help to elucidate the neural mechanisms underlying non-pathological and pathological aggressive behaviour. For example, aggressiveness during seizures or in the postictal period is often demonstrated as a reactive phenomenon that may be triggered by external stimuli in a context of high emotional arousal, anger or fear [15], frequently as a response to unpleasant external stimulation with an attempt to restrain the patient [16, 17]. It is interesting to note that, in the treatment strategy for aggressive bvFTD patients, the caregiver is required to maintain a safe distance and a position to the side of the patient, rather than standing directly in front of them, which would appear more threatening to the patient and potentially stimulate further aggressiveness [18].

Rarely have patients with epilepsy been responsible for physical assault. In this context, in 2002, an international panel conducted an analysis of vide-recorded attacks of 13 patients, as well as a review of the available literature. The panel underlined the need for assessment by a neurologist with special competence in epilepsy and video-EEG in cases in which an individual is on trial for violent crimes [19]. In terms of gender predominance, 11 out of 13 recorded patients



■ **Figure 1.** (A-D) MRI scan showing a dysplasia on the dorsolateral prefrontal cortex: (A) fluid-attenuated inversion recovery image; (B) inversion recovery-weighted coronal image; (C) T2-weighted coronal image; (D) T2-weighted axial image. (E) Localization of the dysplastic lesion is shown in yellow after manual segmentation using volumetric T2-weighted scanning. The pial surfaces were generated using the Free-surfer pipeline. (F) Ictal EEG showing a right fronto-centro-temporal theta rhythmic discharge (yellow arrow). ECG channel shows a sinus tachycardia during the seizure. Muscle artifacts partially covered the onset of the seizure. Calibration = 100mV/cm; EMG2 = right deltoid muscle; EMG3 = left deltoid muscle; ECG = electrocardiogram. (G) Topographic EEG maps of spectral power for distinct frequency bands during the first 10 seconds of a seizure. Alpha, theta and beta activities show asymmetrical distribution on the frontal regions (referential montage absolute scale, LF 1.60 - HF 70Hz, AVG, FFT tapering and detrending, interpolazione spline pl). (H) Topographic EEG maps of spectral power for distinct frequency bands during 10 seconds of the eyes-closed resting state. The oscillations remain largely symmetrical (referential montage absolute scale, LF 1.60 - HF 70Hz, AVG, FFT tapering and detrending, interpolation spline pl). L: left.

were male, suggesting that our case represents a minority [19].
With the development of different neuromodulation techniques to influence brain function (deep brain stimulation, transcranial magnetic stimulation and transcranial direct current stimulation), we may start to elucidate this phenomenon which may aid in developing new therapeutic strategies for neurologically based aggressive behaviour [20].

Connectivity

Studies of functional connectivity during seizures have demonstrated that complex ictal behaviours may depend on altered dynamics within a widely distributed neural network. According to the theory of "global workspace of consciousness", seizure-related hypersynchronous activity within distant cortico-cortical and cortico-thalamic networks may disrupt the neural coherence associated with conscious processing [21].
Similar patterns of long-range changes have been found to be correlated with ictal violent action, in particular, when specific brain networks in fronto-temporal regions transiently lose synchrony [10, 22]. Bartolomei and his colleagues showed a significant desynchronization between the orbito-frontal cortex and the amygdala during ictal aggression [9]. In a different study, the same authors highlighted that, during an ictal strangulation attempt, there was a bilateral increase in connectivity between frontal and temporal regions [8]. It could be hypothesized that this pathological neural state would lead to a break-down of normal regulatory function of these cortical regions. Analogously, neuronal loss in the frontotemporal area in bvFTD could produce an imbalance in intracortical neurotransmitters, leading to cortical hyperexcitability. This results in disrupted frontolimbic connectivity and elevated local connectivity within the prefrontal cortex in bvFTD [23]. Furthermore, neurophysiological studies in bvFTD have shown interictal sub-continuous fronto-temporal theta activity in patients carrying *C9orf72*, *MAPT* and *TREM2* gene mutations [24, 25].

Neuroanatomy

One of the first indications that prefrontal cortex was involved in violent behaviour came from the case of Phineas Gage described by Damasio *et al*. [26, 27]. Patients with ventro-medial prefrontal cortex lesions may show aggressiveness, and behave in an amoral, disinhibited and impulsive manner [28]. Rolls and his colleagues demonstrated that these patients also presented with a perseverative response when the contingencies changed. Difficulty in modifying responses, especially when followed by negative consequences, may contribute to the inappropriate behaviour shown in daily life by patients with frontal lobe damage [29]. Bastin and his colleagues, recording intracerebral EEG signals during stop-signal tasks in patients with epilepsy, showed how the error-monitoring network includes the dorsomedial prefrontal cortex and anterior insula [11].In bvFTD, neuroimaging showed anterior temporal and frontal atrophy on MRI, associated with hypoperfusion or hypometabolism on SPECT or PET [3, 30]. Furthermore, in bvFTD patients, atrophy of the right dorsolateral prefrontal cortex is correlated with severity of apathy, while severity of atrophy of the right mediotemporal limbic structures showed correlation with disinhibition [31]. Reduced lateral temporal and mesial prefrontal glucose metabolism was observed on brain FDG-PET in a group of aggressive children with drug-refractory epilepsy and developmental delay [32]. Severity of aggression was inversely correlated to glucose metabolism of both the left temporal and bilateral medial prefrontal cortex. Although this observation would confirm an association between acts of aggressive impulsive behaviour and antisocial personality disorder and dysfunction of these regions [33], in frontal lobe epilepsy, we are often unable to clearly identify what might be excitatory effects of the dynamic seizure discharge, and what might be inhibitory effects. In comparison with dementia cases, in which effects are more clearly 'lesional', in epilepsy, we may only assume functional effects within the dorso-ventral axis.

Neuropsychology: theoretical frameworks

Connectivity and neuroimaging analysis have shown that antero-mesial prefrontal and anterior temporal connections are engaged in aggressiveness. Interestingly, our patient presented a dorso-lateral prefrontal lesion. This area more rarely correlated with aggressiveness compared to the antero-mesial area. If the ventromedial prefrontal cortex may emotionally control aggressive behaviour, the dorsolateral prefrontal cortex, being involved in problem-solving and cognitive control [20], may act as a "filter" in suppressing emotional reactions that lead to aggressive and violent behaviour [34]. Functional MRI studies have shown that the medial prefrontal cortex generally responds to socially salient feedback, with no significant differentiation between negative and positive feedback, while the dorso-lateral prefrontal cortex is an important modulator for individual differences in controlling aggressiveness [35]. This may imply that individuals who show strong activation in the lateral prefrontal cortex after negative social feedback may be more capable of regulating behavioural impulses, and speculatively, impulsive responses in general [35].

Another brain region involved in controlling aggressive behaviour is the cingulate cortex, mediating between the emotional and rational components of behaviour [34, 36]. In the interplay between the emotional ventro-medial prefrontal cortex and the rational dorsolateral prefrontal cortex, the anterior cingulate cortex may act as a mediator of conflicts and contribute to the ictal hyper-motor component [37]. According to these observations, using intracranial monitoring, Bonini and colleagues described four different types of semiologic features in frontal lobe epilepsy, and the ictal stereotyped motor behaviours correlated with specific patterns of seizure localization, based on anatomical organization along the rostrocaudal axis [22]. We suggest that in our patient, these strictly linked regions are involved and their activity is disrupted during seizure discharge. The patient showed afinalistic, perseverative and hyperkinetic movements associated with violent verbal production. Some of these clinical symptoms have been interpreted as emergence of innate motor repertoires [15], in which impaired higher cortical control would allow archaic and essentially subcortically organized motor behaviours to be wrongly released. Therefore, failure of "top-down" control systems in the prefrontal cortex to modulate aggressive acts appears to play an important role in aggressive behaviour. The work of Trebuchon *et al*. [38] strongly suggests that epilepsy involving the ventromesial prefrontal network is associated with interictal antisocial personality. Their patients presented with epilepsy involving the anterior cingulate cortex, ventromedial prefrontal cortex, and the posterior part of the orbitofrontal cortex, with early propagation to contralateral prefrontal and ipsilateral medial temporal structures. Interestingly, their antisocial personality disorder improved after seizure control [38]. The absence of such interictal dysfunction in our patient might represent another argument that the dorsolateral prefrontal system has a more indirect effect in producing aggressive behaviour.

These observations are consistent with animal studies showing that lesions in these cortical areas are related to uncontrolled aggression [39].

In conclusion, hypermotor seizures and ictal behavioural alterations can exhibit bizarre motor phenomena that may be misinterpreted as psychiatric disease, particularly when awareness is preserved. Recognizing these ictal symptoms by video-EEG monitoring will assist in correct diagnosis. The understanding of this mechanism could help to clarify the neural basis and treatment of aggressiveness in epilepsy, dementia and other disorders. Our patient shows how a complex aggressive behaviour may not depend on dysfunction of a single region, but appears to be related to a large-scale anatomo-functional system, including the dorso-lateral prefrontal regions. ■

Supplementary data.
Summary didactic slides are available on the www.epilepticdisorders.com disorders.

Acknowledgments and disclosures.
The authors thank Professor Flavio Nobili for his valuable advice, Valerio Scacco for his support in English language editing, Piero Cannata for video technical assistance and the Paolo Zorzi Association for Neurosciences for its unconditional support.
None of the authors have any conflict of interest to declare.

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Legend for video sequence

Self-limited events characterized by paroxysmal rage and aggressive behaviour. In the first part, the home video reveals preserved awareness. In the second part, video-EEG monitoring underlines the patient's ability to report seizures.

Key words for video research on www.epilepticdisorders.com

Phenomenology: rage and aggressive behaviour
Localization: dorso-lateral prefrontal cortex
Syndrome: drug-resistant focal epilepsy
Aetiology: focal cortical dysplasia

TEST YOURSELF

(1) Which cortical area could act as an "intermediary" between the emotional and rational components of behaviour and be associated with a hyper-motor component of seizures?

A. Anterior cingulate cortex
B. Dorso-lateral prefrontal cortex
C. Insula

(2) Which of the following concerning the dorsolateral prefrontal cortex (DLPFC) is correct?

A. The DLPFC usually acts as a filter by suppressing emotional reactions, leading to aggressive and violent behaviours
B. Lesions in the DLPFC may be associated with focal seizures, characterized by paroxysmal rage and aggressiveness
C. The DLPFC is not involved in modulation of aggressive behavioural responses based on different social situations
D. A and B

(3) In patients with bvFTD, what symptom tends to be associated with atrophy of the prefrontal cortex?

A. Mental rigidity
B. Disinhibition
C. Apathy

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".