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LETTERS TO THE EDITOR

Aggression and Violence in Patients with Epilepsy

To the Editor:

In their article Marsh and Krauss present a comprehensive summary of the problem of aggression in epilepsy (1). In the section on interictal violence they summarize the different findings from brain imaging studies and point to the frontal lobe, the limbic circuit, and the left hemisphere as areas of critical importance in the mediation of aggressive behavior. In a recently published study we addressed the problem of structural and, in particular, limbic brain pathology in patients with temporal lobe epilepsy (TLE) and intermittent explosive disorder. While the prevalence of mesial temporal lobe sclerosis was significantly lower in patients with TLE and aggression, we found evidence of amygdala pathology in 48% of the aggressive patients (2). There was an increased prevalence of encephalitis in patients with epilepsy and aggression, and overall IQ and particularly verbal IQ were significantly reduced. At the same time the aggressive patients displayed highly significant loss of cortical gray matter in the left dorsolateral prefrontal cortex (3). A theory of dual brain pathology as a neural substrate of hyperarousal–dyscontrol states integrates all these findings within one theoretical framework (4). Figure 1 illustrates this concept. Amygdala-associated brain pathology is thought to result in dysfunctional states of hyperarousal triggered by minor life events. In case of simultaneous frontal lobe pathology associated with dyscontrol syndromes, hyperarousal–dyscontrol states with severe aggressive behavior may result. Most of the neuropathological findings in patients with interictal aggression presented by Marsh and Krauss are in line with this concept of hyperarousal–dyscontrol states. It is noteworthy that the brain pathology described above was subtle and detectable only with quantitative MRI and not on routine MRI investigations.

Further research using quantitative neuroimaging in patients with episodic dyscontrol or intermittent explosive disorder without epilepsy could test this

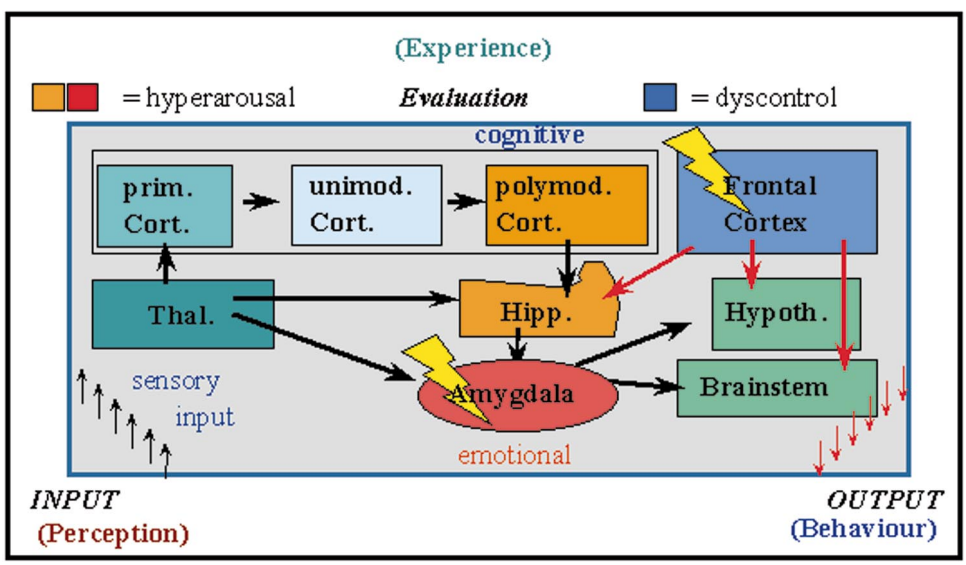

FIG. 1. Dual brain pathology as a vulnerability factor in hyperarousal–dyscontrol syndromes.

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hypothesis of a neurobiology of hyperarousal–dyscontrol syndromes.

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Reply

To the Editor:

The authors have done detailed work in this area and present an interesting scheme to explain "hyperarousal-dyscontrol syndromes." I think, however, they overstate current findings on involvement of the amygdala in aggressive states in patients with temporal lobe epilepsy (TLE) and understate the importance of bilateral or widespread unilateral brain dysfunction in patients with epilepsy and aggressive behavior. Five of 25 aggressive patients with TLE they studied (20%) had amygdalar atrophy. Most of these patients, however, had cognitive dysfunction involving both cerebral hemispheres and had histories of encephalitis [3]. The social limitations such patients face due to cognitive impairment and seizures should also be incorporated in their model. Patient 1 in our series had severe rage attacks associated with previous encephalitis and bi-temporal seizures, but did not respond to bilateral amygdalotomies, and had near resolution of aggressive outbursts following cingulotomies. The authors note that the amygdala is involved in "affective evaluation of multimodal sensory information" [2], however, it remains a hypothesis that spontaneous epileptogenic activity in human amygdala directly contributes to aggressive behavior.

TLE presents an interesting neurobiological model for studying emotional and behavioral states. It may unnecessarily stigmatize patients with epilepsy, however, to overemphasize the frequency with which aggressive outbursts are associated specifically with seizures. The authors, for example, excluded 11/39 patients from their TLE control group because the patients had what they interpreted as ictal aggression. The "hyperarousal" states they describe may in fact be a nonspecific state of post-ictal delirium in one patient, an emotional reaction due to limited coping skills in social interactions in another patient, and rarely, a direct effect of seizures on limbic and other brain structures.

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Reply

To the Editor:

I am delighted that in his response to our paper (1), Dr. Millichap has properly advised us regarding the types of patients that should be considered for antiepileptic drugs (AEDs) (2). There is likely no one in the world who has dealt with more patients with this disorder than Dr. Millichap in the course of his very long career.