

## EATING EPILEPSY

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**SUMMARY** — Eating epilepsy is a rare type of reflex epilepsy. A 24 years-old male with eating reflex complex partial seizures was submitted to clinical, neurological, neuroradiological and EEG studies. Neurologic and CT examinations were normal. EEG recordings including video-EEG monitoring during meals disclosed focal abnormalities related to both temporal lobes prevailing at the left side and secondary bilateral synchrony mainly in more anterior regions. Ictal findings were similar to the interictal secondary bilateral synchrony except for its longer duration. PB, VPA and DPH monotherapies were ineffective. High dose CBZ monotherapy yielded good but uncomplete seizure control. Since a big number of precipitants could be involved, no specific physiopathological basis could be established.

### **Epilepsia ao comer**

**RESUMO** — A epilepsia reflexa ao comer é forma rara de epilepsia reflexa. Um paciente de 24 anos com crises parciais complexas reflexas ao comer foi submetido a avaliações clínicas, neurológicas, neurorradiológicas e eletrencefalográficas. O exame, neurológico e a tomografia de crânio foram normais. Registros de EEG, incluindo monitorização vídeo-EEG durante a refeição, mostraram presença de anormalidades focais relacionadas a ambos os lobos temporais, predominando à esquerda, e sincronia bilateral secundária, predominando em regiões anteriores. Os achados ictais são semelhantes à sincronia bilateral secundária interictal exceto por sua maior duração. Monoterapias com PR, DPH e VPA não surtiram efeito. Monoterapia com altas doses de CBZ trouxe bons resultados porém com controle incompleto das crises. Como grande número de fatores precipitantes estava potencialmente envolvido, não foi possível determinar com precisão a base fisiopatológica das crises reflexas neste caso.

**The reflex epilepsies include those epileptic syndromes consisting of seizures that are triggered by specific stimuli usually a clearly recognized somatosensitive or sensory one. Eating epilepsy is an uncommon form of these<sup>5</sup>. Several physiopathological mechanisms involved in the genesis of these seizures have already been discussed<sup>1,2,3,4</sup>. We present a case of eating epilepsy, its therapeutical evaluation and clinical follow-up.**

### **METHODS**

**A 24 years-old man (PHC) presenting with atonic, tonic-clonic and complex partial seizures during meals was submitted to clinical, electroencephalographic (EEG), computerized tomography (CT) and cerebrospinal fluid (CSF) examinations and blood biochemistry. EEG studies included routine 16-channel EEG, sphenoidal electrodes recordings and video-EEG monitoring during meals.**

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## CASE REPORT

PHC, a 24 years-old white male, first presented with daily atonic seizures at the age of 15. Eight months later, primarily generalized tonic-clonic seizures appeared with a daily frequency, together with complex partial seizures with automatisms, 2-3 times a day, exclusively during meals. The patient considered these episodes as «absences». Observers described that while starting to eat he suddenly stared, became unresponsive for seconds and started talking illogical words and performing manual automatisms. The post-ictal state included confusion and amnesia for the entire episode. His clinical background was unremarkable except for a febrile convulsion at the age of 2 years. His mother and brother are epileptics, both with primarily generalized tonic-clonic seizures and are under good clinical control in respect to their seizures. When he first presented to us at the age of 19, he was taking carbamazepine (CBZ), phénobarbital (PB), phenytoin (DPH) and primidone (PRM) at non-therapeutic levels. Generalized seizures were controlled but complex partial seizures remained with the frequency of 3-4/day, always related to meals. Physical and neurological examination revealed no abnormalities. CSF analysis and the haematological survey were normal. CT scans were normal. Conventional EEG showed frequent spikes and sharp-waves often associated to slow-waves over the left fronto-temporal region spreading diffusely to ipsi- and contralateral areas. Independent spikes were registered over the right temporal region (Fig. 1). Ictal recordings during video EEG monitoring were similar to the interictal secondary bilateral synchrony except for its longer duration (14 seconds). PB, DPH, VPA monotherapies were ineffective. CBZ monotherapy yielded the best results, lowering the complex partial seizures (CPS) frequency to 2/month. Clobazam add-on therapy (20mg/day) was also ineffective.

## COMMENTS

Eating epilepsies represent an heterogeneous group with variable clinic and EEG findings. However some common features emerge from the reported cases. This young adult male disclosed CPS always related to the onset of eating and unrelated clinically primarily generalized tonic-clonic and atonic seizures.

Reflex seizures precipitated by eating usually begin in the second decade of life. There seems to exist a higher incidence in males. Eating reflex seizures are usually complex partial ones. Associated non-reflex seizures are very often present and frequently appear earlier than the reflex ones, with a lower frequency<sup>1,2</sup>. Focal EEG findings are common in such cases<sup>1,2</sup> specially related to the temporal lobe, but secondary bilateral synchrony appeared to be a landmark in many patients<sup>3</sup> and might correlate to the type of CNS involvement in cases of eating epilepsy<sup>3,7,10,n</sup>.

Non-reflex seizures are easily controlled with current anticonvulsant therapy. On the other hand, reflex seizures are often clinically refractory<sup>2</sup>. Though the use of clobazam is described as effective in eating epilepsy<sup>1</sup>, 1-5 benzodiazepine therapy has shown tolerance and poor clinical control in some cases<sup>1,2</sup>.

Reflex seizures occur at specific stages of the meal in each patient, but can vary from one case to the other. This suggests different pathways and physiopathological mechanisms. Boudouresques and Gastaut<sup>4</sup> and Vizioli<sup>12</sup> pointed to the importance of gastric distention and to the role of the passage of food along the gastrointestinal tract as triggers of visceral afferents which might represent the physiopathological basis of reflex seizures. These previous mechanisms are particularly important when seizures occur at the end of a meal or at the post-prandial state MA<sup>12</sup>. In the case described here, seizures occurred just after starting the meal. The trigger mechanism probably involves other factors such as mastication and swallowing. The role of masticatory movements, together with smelling and tasting food and the role of the amygdala and other structures within the limbic system should be emphasized<sup>2A</sup>.

Cirignotta et al.<sup>6</sup> called the attention to the possibility of activation of hypothalamic nuclei involved in the cephalic phase of digestion and a CNS mediated reflex. The data obtained on the role of chemical agents in food and its taste, texture and temperature are inconclusive and probably variable<sup>1,2A</sup>. The motor acts associated to the food intake have also to be considered<sup>10</sup>.

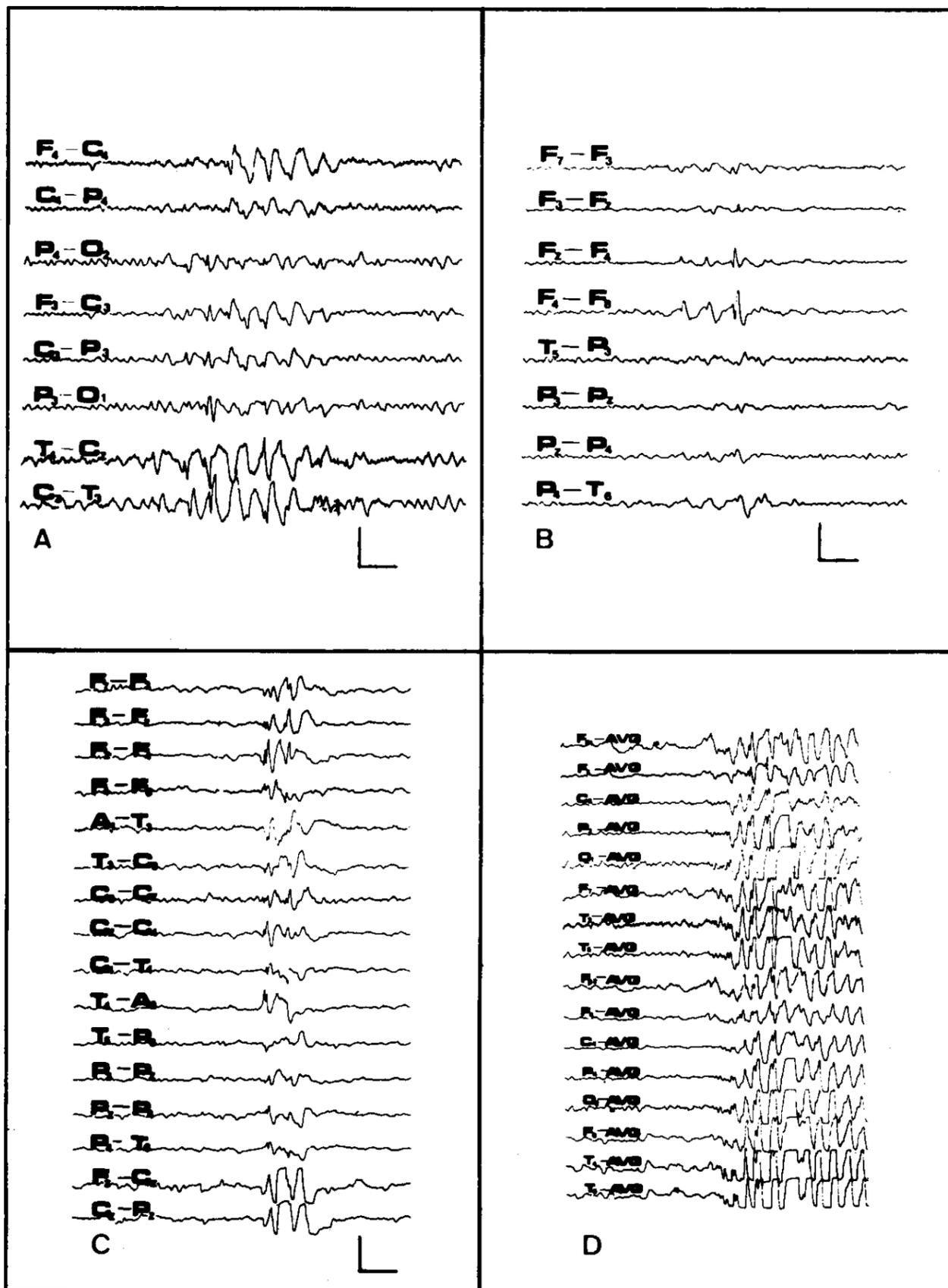


Fig. 1 — Case PHC (A) Spike and slow waves projected over the left mid temporal lobe. (B) Spike and slow wave complex projected over the right anterior temporal lobe. (C) Polyspike and slow wave complex projected over the right anterior and mid temporal lobe. (D) Diffuse bilateral and synchronous spike and wave discharges prevailing in more anterior regions bilaterally. Calibration bars: vertical = 25  $\mu$ v; horizontal = 1 sec.

**Despite the existence of a multiplicity of precipitant factors not only the mechanisms described should be considered. Environmental factors such as the familial atmosphere may play a role<sup>2,8</sup>. Finally, there appears to be regional differences in the prevalence of eating epilepsy around the world, which is for instance especially high in India<sup>3</sup>. This complex of possible genetic, environmental and pathological aspects should be more extensively studied.**

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