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Hippocampus and epilepsy

The concept of temporal 'plus' epilepsy

Le concept d'épilepsie temporal 'plus'

P. Kahane [a,b,c,*], *C. Barba* [d], *S. Rheims* [e,f], *A.S. Job-Chapron* [a,b,c], *L. Minotti* [a,c], *P. Ryvlin* [e,f,g]

[a] *GIN, Inserm, U836, chemin Fortuné-Ferrini, 38700 La Tronche, France*
[b] *University Grenoble-Alpes, université Joseph-Fourier, BP 53, 38041 Grenoble cedex 9, France*
[c] *Épilepsie et malaises neurologiques, neurology department, hôpital Michallon, CHU de Grenoble, boulevard de la Chantourne, 38700 La Tronche, France*
[d] *Child Neurology Unit, Pediatric Hospital "Meyer", 24, viale Gaetano Pieraccini, 50141 Firenze, Italy*
[e] *Department of functional neurology and epilepstology, hospices civils de Lyon, 59, boulevard Pinel, 69003 Lyon, France*
[f] *Research neuroscience center of Lyon, centre hospitalier Le-Vinatier, 95, boulevard Pinel, 69500 Bron, France*
[g] *Department of clinical neurosciences, CHU Vaudois, 46, rue du Bugnon, 1011 Lausanne, Switzerland*

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ABSTRACT

The concept of temporal 'plus' epilepsy (T+E) is not new, and a number of observations made by means of intracerebral electrodes have illustrated the complexity of neuronal circuits that involve the temporal lobe. The term T+E was used to unify and better individualize these specific forms of multilobar epilepsies, which are characterized by electroclinical features primarily suggestive of temporal lobe epilepsy, MRI findings that are either unremarkable or show signs of hippocampal sclerosis, and intracranial recordings which demonstrate that seizures arise from a complex epileptogenic network including a combination of brain regions located within the temporal lobe and over closed neighbouring structures such as the orbitofrontal cortex, the insulo-opercular region, and the temporo-parieto-occipital junction. We will review here how the term of T+E has emerged, what it means, and which practical consideration it raises.

RÉSUMÉ

Le concept d'épilepsie temporal 'plus' (T+E) n'est pas nouveau et de nombreuses observations faites au moyen d'électrodes intracérébrales ont illustré la complexité des circuits neuronaux qui impliquent le lobe temporal. Le terme T+E a été utilisé pour unifier et mieux individualiser ces formes d'épilepsies multilobaires, qui se définissent par des caractéristiques électrocliniques évocatrices d'une épilepsie du lobe temporal, un examen IRM dépourvu d'anomalie ou montrant des signes de sclérose hippocampique et des enregistrements intracrâniens qui montrent que les crises sont générées au sein d'un réseau

* *Corresponding author.*
E-mail address: philippe.kahane@ujf-grenoble.fr (P. Kahane).
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épileptogène complexe impliquant une combinaison de structures temporales et juxtatemporales (cortex orbitofrontal, région insulo-operculaire, jonction temporo-parieto-occipitale). Nous verrons dans cet article comment ce terme T+E est né, ce qu'il signifie et quelles considérations pratiques il soulève.

1. Introduction

These last 20 years, besides the delineation of the syndrome of Mesiotemporal lobe epilepsy with hippocampal sclerosis (MTLE-HS) [1], a number of pathological, morphological, and metabolic changes have questioned the exclusive role of the sole hippocampal formation in TL seizure generation. Similarly, a number of studies conducted by means of intracerebral electrodes have illustrated the complexity of neuronal circuits associated with HS, and TLE subtypes covering both the limbic and neocortical systems have been proposed, and referred to as mesial, temporopolar, mesiolateral, lateral and temporal 'plus' [2]. Therefore, it is clear that the notion of focality is too simplistic to suit the variety of TL seizure generators, and the concept of 'epileptogenic network' has been developed to account for the complex spatial organization of several distinct cortical areas generating seizures [3], which possibly depends on disease duration [4] and seizure frequency [5]. Temporal 'plus' epilepsies are a good example of such complex – and possibly gradually evolving [6] – epileptogenic networks, and we will briefly review here how this acronym has emerged, what it means, and which practical consideration it raises.

2. Temporal plus epilepsy: a 'default' concept

Results of TLE surgery have shown that not all TLE patients are cured following surgery, the median proportion of long-term ($\geq$ 5 years) seizure-free patients having been evaluated to 66% [7], with even lower figures after AED withdrawal [8]. Although these results definitely demonstrate that the epileptogenic brain tissue has not been fully removed in a significant proportion of the cases, reasons of TLE surgery failures remain a debated issue [9]. Such failures have been often discussed according to the completeness of the causative TL lesion removal or to the completeness of mesial and/or lateral TL excision, but second TL operations result in a class I outcome in 17–63% of the cases [10–16]. Likewise, an independant controlateral TL seizure onset might explain failures of TLE surgery [17], but it accounts for 18–21% of TLE cases studied by depth electrodes [18,19], and for fewer than 20% of failed TL resections [20]. Failures of TLE surgery might also be due to an extratemporal seizure onset zone mimicking TL seizures [21], a condition described under the term of pseudotemporal seizures [22], where TL resection is consistently associated with a very poor seizure outcome [23,24]. In such a situation, the epileptic generator localized outside the TL can be clinically silent until the discharge propagates to TL structures, therefore mimicking TL seizures both clinically and on scalp EEG [25]. In the absence of a structural lesion, however, these forms of focal epilepsies, although usually suspected, are rarely demonstrated.

Overall, many patients in whom TLE surgery fails do not exhibit evidence of incomplete TL resection, bitemporal epilepsy, or pseudotemporal epilepsy. Our group suggested more than a decade ago that they could suffer from temporal 'plus' epilepsy (T+E), a term which refers to a complex epileptogenic network including a combination of brain regions located within the TL and over the closed neighbouring structures such as the orbitofrontal cortex, the insula, the frontal and parietal operculum, and the temporo-parieto-occipital junction [26–28].

3. Temporal plus epilepsy: an 'observational' concept

The concept of T+E is not new and since a number of decades, observations made by means of intracerebral recordings have shown that an epileptogenic zone could be distributed between distinct (but interconnected) regions of different lobes of one hemisphere, thus leading to the concept of frontotemporal [29–32], temporoperisylvian [5,33,34] and temporo-parieto-occipital epilepsies [35–38].

The term T+E was used to unify and better individualize these specific forms of multilobar epilepsies, which are currently characterized by a prominent involvement of the TL, electroclinical features primarily suggestive of TLE, MRI findings that are either unremarkable or show signs of HS, and intracranial recordings which demonstrate either an ictal discharges originating simultaneously from the TL and the neighboured extratemporal structures (Fig. 1), or two co-existing seizure types in the same patient, with temporal and extratemporal ictal onset, respectively (Fig. 2). T+E therefore raises similar conceptual issues than dual pathology, where an extratemporal lesion coexists with mesial temporal sclerosis, both being epileptogenic [39].

Thus, T+ seizures must be differentiated from seizures that start in the TL and spread more or less rapidily in extratemporal areas, where the additional removal of extratemporal lobe structures is not necessary. The very frequent insular spread of TL seizures [40] is a good example of such a complex issue, since it does not influence the prognosis of TL surgery [41], but can elicit symptoms suggesting an insular involvement, which subsequently can lead to an erroneous suspicion of T+E (Fig. 3).

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