The Role of the Temporal Pole in Temporal Lobe Seizure Networks: An Intracranial Electrode Investigation.

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Introduction

A convergence of evidence suggests that the temporal pole (TP) plays an important, and potentially under appreciated, role in the genesis and propagation of seizures in temporal lobe epilepsy (TLE) [1, 2]. Understanding the role of the TP in TLE is becoming increasingly important because selective surgical resections for medically-intractable TLE spare TP cortex [3, 4]. The purpose of this study is to characterize the role of temporopolar cortex in TLE using dense electrocorticographic recordings of the TP in patients undergoing invasive monitoring for medically-intractable TLE.

Methods

In 10 consecutive patients (Table 1) with medically-intractable epilepsy who would eventually undergo cortico-amygdalohippocampectomy (CAH), chronic ECoG recordings of the TP were obtained using an array customized to provide dense coverage of the TP as part of invasive monitoring [5, 6]. A retrospective review of clinical records including ECoG, neuroimaging, neuropathology, and clinical outcomes was performed. A board-certified neuropathologist and neuroradiologist provided reviews of histopathologic and neuroimaging data, respectively. Patients were placed into Group A if seizure onset was from the TP and Group B if seizure onset was anywhere else.

Table 1

Patient /Group	Sex	Age	Feb Sz	Epi On	Epi Lat	Surgical Intervention	Post-op	F/U
1 - A	M	21	No	16	R	R CAH	I	2 mo
2 - A	M	29	Yes	18	L	L CAH	I	40 mo
3 - A	M	26	No	21	R	R CAH	II	57 mo
4 - A	F	50	No	1	L	L CAH + Lesionectomy	II	21 mo
5 - A	M	31	No	23	R	R CAH	II	43 mo
6 - A	M	26	Yes	21	R	R CAH + OF Resection	III	8 mo
7 - B	M	47	No	35	L	L CAH	I	27 mo
8 - B	M	31	No	24	L	L CAH	I	46 mo
9 - B	M	35	No	33	R	R CAH	I	32 mo
10 - B	M	51	No	33	L	L CAH	I	38 mo

Results

Electrophysiological results are summarized for all subjects in Table 2. For six of the 10 patients, the ictal activity of at least one clinically significant seizure type was first detected from an electrode on TP cortex. One such seizure and corresponding electrode localization is depicted in Figure 2.

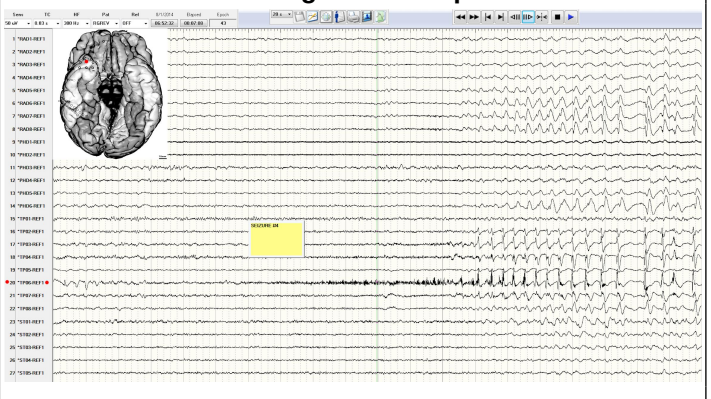
All but one patient who did not have a seizure exhibiting ictal onset of a clinically significant seizure from the TP exhibited other signs of TP epileptogenicity. Patient 9, for example, had clinically silent ictal activity arising from the TP electrodes. Each of these ictal events lasted less than 6s and were not associated with clinical seizure activity. Patient 10 exhibited early spread of ictal activity to TP cortex within 10 seconds.

Interictal discharges

In total, seven of 10 patients had interictal epileptiform discharges (IEDs) from TP cortex. For Group A, 3/6 (50%) patients had temporopolar IEDs. For Group B, 4/4 (100%) patients had temporopolar IEDs. Thus, the presence of IEDs localized to the TP was not associated with seizure onset from the TP.

Neuroimaging data is also summarized in Table 2.

Seizure Arising From Temporal Pole



Electrocorticogram and MR localization of seizure onset from the temporal pole in a given patient.

Table 2

Patient/ Group	PET Hypo	PET Lat	MRI MTS	MRI TPa	MRI AL	TP IEDs	Path Lesion	MTS
1 - A	Y	R	-/-	-/-	-/+	+	FCDIIa	No
2 - A	Y	BL	-/-	-/-	-/+	+	FCDIb	No
3 – A	Y	R	-/-	-/-	+/+	-	GG; FCDIIIb	No
4 – A	Y	L	+/+	-/-	-/-	-	FCDIIa	No
5 - A	N/A	N/A	-/-	-/-	+/+	+	DNT	No
6 - A	Y	R	+/+	+/+	-/-	-	FND	Yes
7 - B	Y	L	+/+	-/-	+/+	+	FCDIIIa	Yes
8 - B	Y	L	-/-	+/+	-/+	+	FCDIb	No
9 - B	Y	L	+/+	-/+	-/-	+	None	Yes
10- B	Y	L	+/+	-/-	-/-	+	None	Yes

Conclusions

These data demonstrate that TP cortex plays a crucial role in temporal lobe seizure networks. Our results, in conjunction with other clinical studies, suggest that seizures originating from the TP are a possible reason for poor seizure control after selective amygdalohippocampectomy. Neuroimaging and IEDs are not indicative of seizure onset in the TP.

References

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