The Impact of Subcortical Volumes and Cortical Thicknesses on Outcome of Resective Epilepsy Surgery

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CNS 2016 Annual Meeting, San Diego, California, September 24-28, 2016

RWJBarnabas HEALTH

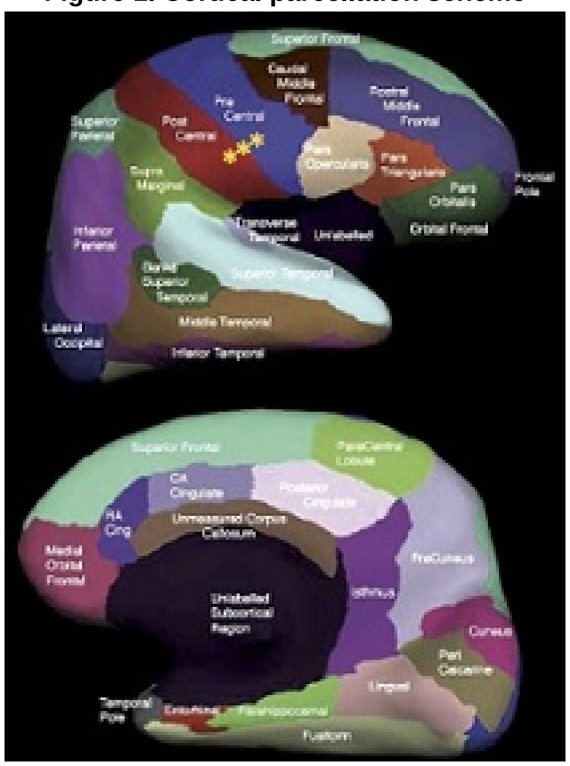
Introduction

Ipsilateral mesial temporal atrophy is a well-known prognostic indicator of temporal lobe resection. The significance of atrophy of other structures in these patients is less clear.

Methods

Structure-based quantification of the MRIs of 48 epilepsy patients undergoing resective surgery and 48 age- and sex-matched normal controls was performed using an automated cortical parcellation and subcortical segmentation algorithm. Chi-square testing of structural atrophy status to seizure control was then performed.

Figure 1. Cortical parcellation scheme



An automated labeling system for subdividing the human cerebral cortex on MRI scans into gyral based regions of interest, Desikan et al., (2006). NeuroImage, 31(3):968-80.

Results

Compared to patients with preserved putaminal volume, patients with putaminal atrophy ipsilateral or contralateral to the side of resection had significantly higher seizure-free outcome. Patients with preserved posterior corpus callosum had significantly higher seizure-free outcome compared to those with posterior corpus callosal atrophy. Patients with preserved pars triangularis and lateral occipital, medial orbitofrontal, and rostral middle frontal cortices contralateral to the side of resection had more seizure-free outcomes than patients with thinning of these structures. Subgroup analysis of cortical thickness in the setting of subcortical structural atrophy revealed that more patients were seizure free who had putaminal atrophy and preserved medial orbitofrontal cortex contralateral to the resected side. Patients with preserved pars triangularis and medial orbitofrontal and rostral middle frontal cortices contralateral to the resected side in the setting of contralateral putaminal atrophy had better outcomes. On the other hand, patients had worse seizure control if atrophied posterior corpus callosum was observed in the setting of lateral occipital and medial orbitofrontal cortical thinning contralateral to the side of resection.

Table 1. Subcortcal atrophy and cortical parcellation thinning to its outcome in post epilepsy resective surgery

Structure, N(%)		Engel 1	Engel 2-4	Total	p-value
Ipsilateral putamen	No atrophy	8 (24.2)	10 (76.9)	18 (39.1)	0.001
	Atrophy	25 (75.8)	3 (23.1)	28 (60.9)	
Contralateral putamen	No atrophy	10 (30.3)	9 (69.2)	19 (41.3)	0.016
	Atrophy	23 (69.7)	4 (30.8)	27 (58.7)	
Corpus Callosum Posterior	No atrophy	10 (30.3)	0 (0)	10 (21.7)	0.025
	Atrophy	23 (69.7)	13 (100)	36 (78.3)	
Contralateral lateraloccipital	No Thinning	26 (78.8)	5 (38.5)	31 (67.4)	0.009
	Thinning	7 (21.2)	8 (61.5)	15 (32.6)	
Contralateral medialorbitofrontal	No Thinning	24 (72.7)	4 (30.8)	28 (60.9)	0.009
	Thinning	9 (27.3)	9 (69.2)	18 (39.1)	
Contralateral parstriangularis	No Thinning	24 (72.7)	5 (38.5)	29 (63)	0.03
	Thinning	9 (27.3)	8 (61.5)	17 (37)	
Contralateral rostralmiddlefrontal	No Thinning	21 (63.6)	4 (30.8)	25 (54.3)	0.044
	Thinning	12 (36.4)	9 (69.2)	21 (45.7)	

Table 2. Cortical parcellation thickness status with concurrent subcortical atrophy status to the outcome

Cortical parcellation, N(%)	concurrent atrophy status	Outcome			p- value
		Engel 1	Engel 2-4	Total	
No thining of contralateral medial orbitofrontal	No atrophy of Ipsilateral Putamen	5 (20.8)	4 (100)	9 (32.1)	.002
	Atrophy of Ipsilateral Putamen	19 (79.2)	0 (0)	19 (67.9)	
No thining of contralateral medial orbitofrontal	No atrophy of Contralateral Putamen	8 (33.3)	4 (100)	12 (42.9)	.013
	Atrophy of Contralateral Putamen	16 (66.7)	0 (0)	16 (57.1)	
No thining of contralateral parstriangularis	No atrophy of Contralateral Putamen	8 (33.3)	5 (100)	13 (44.8)	.006
	Atrophy of Contralateral Putamen	16 (66.7)	0 (0)	16 (55.2)	
No thining of contralateral rostralmiddlefrontal	No atrophy of Contralateral Putamen	7 (33.3)	4 (100)	11 (44)	.014
	Atrophy of Contralateral Putamen	14 (66.7)	0 (0)	14 (56)	
Thinning Contralateral lateraloccipital	No atrophy of Corpus Callosum Posterior	3 (42.9)	0 (0)	3 (20)	.038
	Atrophy of Corpus Callosum Posterior	4 (57.1)	8 (100)	12 (80)	
Thinning of Contralateral medialorbitofrontal	No atrophy of Corpus Callosum Posterior	4 (44.4)	0 (0)	4 (22.2)	.023
	Atrophy of Corpus Callosum Posterior	5 (55.6)	9 (100)	14 (77.8)	

Conclusions

Putaminal atrophy and preserved posterior corpus callosum, as well as preserved par triangularis and lateral occipital, medial orbitofrontal, and rostral middle frontal cortices contralateral to the side of resection were suggestive prognostic indicators for seizure-free outcome. Larger studies are needed to determine the relative contribution of each structure to seizure control.

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