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Early childhood prolonged febrile convulsions, atrophy and sclerosis of mesial structures, and temporal lobe epilepsy:

An MRI volumetric study

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Article abstract—We performed MRI volumetric measurements of the amygdala (AM) and hippocampal formation (HF) in a group of 43 patients with temporal lobe epilepsy not controlled by optimal drug treatment. Fifteen patients (35%) had a history of prolonged febrile convulsions (PFC) in early childhood; 30 patients underwent surgery, and histopathology was available in twenty-four. The mean values of AM and HF volumes ipsilateral to the EEG focus were significantly smaller than those of normal controls. The volumetric measurements showed a more pronounced atrophy of the AM in patients with a history of PFC, although the HF volumes were also smaller in this group. Patients with a history of PFC had a higher proportion of more severe mesial temporal sclerosis (MTS) compared with those with no PFC. These findings confirm a correlation between early childhood PFC, the severity of atrophy of mesial structures, and MTS.

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Hippocampal sclerosis is frequent in temporal lobe epilepsy (TLE), as demonstrated at autopsy[1,2] and in tissue resected at the time of surgical treatment.[3-5] The etiology and pathogenesis of hippocampal sclerosis and its relationship to TLE has been a source of controversy over the last century.[1,6] Hippocampal sclerosis may have several causes, but an association with a history of prolonged, usually febrile, early childhood convulsions is a common finding in TLE.[1,7,8]

MRI is currently the most effective method for detecting gross structural lesions in patients with

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TLE. However, many candidates for surgical treatment of seizures have gliosis and neuronal loss in mesial temporal structures that cannot be seen clearly, even on preoperative high-resolution MRI.[9-11]

Quantitative MRI-based volume measurements of the hippocampus improve the detection of unilateral atrophy in patients with epilepsy.[12-16] Since the amygdala (AM) plays an important role in the pathophysiology of TLE, it appeared reasonable to include measurements of this structure as well as that of the hippocampal formation (HF).[1,16-21]

In the present study, we compared the results of volumetric measurements of the AM and HF and the histopathology in patients with TLE who had prolonged febrile convulsions (PFC) in early childhood with those of TLE patients without PFC.

Methods. *Subjects.* We performed MRI volumetric measurements of the AM and HF in a group of 43 patients (19 males and 24 females; mean age, 34.3 years) with TLE not controlled by optimal drug treatment. A clear EEG lateralization was available in 35 patients using surface and sphenoidal recordings. In eight, the lateralization was determined by depth electrode studies because of the ambiguity of the scalp EEG. The degree of lateralization was sufficiently clear to permit a surgical decision in all. Patients were not otherwise selected and represent a group admitted for investigation from June 1991 to August 1992.

The history of PFC was based on detailed accounts from parents and other relatives at the time of hospital admission as well as on a review of the patients' medical records of early childhood hospitalization. The 43 patients were divided into the following groups:

Group I. Fifteen patients with history of PFC in early childhood (35%), with mean age of 29.4 years (range, 18 to 48; SD = 9.0).

Group II. Twenty-eight patients with *no* history of PFC (65%), with mean age of 36.9 years (range, 12 to 62; SD = 10.0).

We used a control group consisting of 13 healthy volunteers (eight men and five women), with mean age of 32.2 years (range, 20 to 50; SD = 11.3), for comparison.

The characteristics of the patient groups are summarized in table 1, and the controls in table 3.

Thirty patients underwent either transcortical selective amygdalo-hippocampectomy or anterior temporal lobe resection including amygdala and anterior hippocampus. Since the surgical removals were done by subpial aspiration, the samples of mesial temporal structures available for pathologic examination were not optimal, but correlation of reduced volumes of AM and HF with pathologic findings was possible in 24 patients.

After excision, the tissue was fixed for 12 to 24 hours in 10% buffered formalin, then cut anteroposteriorly, embedded in paraffin, and sectioned. Staining was carried out using Cajal's gold chloride sublimate, Luxol fast blue, and hematoxylin-eosin.

We used the term "mesial temporal sclerosis" (MTS), as reviewed by Meencke and Veith,[1] to include changes in the hippocampus, the adjacent entorhinal cortex, and the amygdala. The term "MTS" is used here as a qualitative and descriptive one.[1,21] The pathologist was unaware of whether or not the patients had a history of PFC.

MRI acquisition. MRIs were obtained on a 1.5-tesla Philips Gyroscan S15-HP unit. Thirty-two slices on coronal views, perpendicular to the lateral sulcus (Sylvian fissure), were obtained using a 3-D gradient fast field echo sequence with 3-mm contiguous sections, a 75/16/2 (TR/TE/number of signal averages) pulse sequence, a matrix size of 256 × 256, with a 250-mm field of view and a 60° flip angle.

Volumetric analysis. The images were transferred to a Sun SPARC work station (Sun Microsystems, Mountain View, CA). Volumetric measurements were performed with an interactive software program developed at the Neuroimaging Laboratory of the Montreal Neurological Institute. The regions of interest were outlined using a manual contouring editing function. Once the outline had been defined, the slice volume was calculated automatically by the computer program. A detailed description of the protocol and the anatomic landmarks used in the present work has been given by Watson et al.[17]

Volumes of AM and HF were obtained on each patient and compared with values from the normal control group. Another measure to determine the degree of asymmetry between sides was established by using the right minus left side difference (R−L). This method was utilized by others[12-14] to assess asymmetry of the HF.

Four AM measurements were excluded due to MRI artifacts present in this region.

Statistical analysis. We defined significant smallness of AM and HF as values below 2 SD of the respective mean values of the normal control group. The side asymmetry expressed by R−L AM and R−L HF was considered significant when 2 SD above the mean of control group. The volume measurements were expressed in cubic millimeters.

We performed an analysis of variance (ANOVA) comparing the AM and HF volumetric measurements from all three groups: patients with PFC (group I), patients without antecedent of PFC (group II), and normal controls. We then used a planned comparison with two orthogonal comparisons: (1) PFC patients (group I) versus patients without PFC (group II); (2) normal controls versus all patients (group I plus group II). The same statistical approach was used to analyze the amount of asymmetry between sides (R−L) for AM and HF volumes.

We used Fisher's exact test to compare the pathologic categories in each of the two groups. This analysis was based on the classification given independently by the pathologist as mild, moderate, or severe degree of MTS.

We compared the age (t test) of both groups of patients.

We considered statistical significance to be present at $p < 0.05$.

Results. The volumetric measurements of patients and controls are summarized in tables 1 to 3.

The ANOVA showed a significant difference among the three groups for AM ($F = 22.5$, $p < 0.0001$) and HF volumes ($F = 14.1$, $p < 0.0001$). The orthogonal comparison showed a significant difference between the two groups of patients, with AM and HF volumes being smaller in group I (patients with PFC) ($F = 15.7$, $p < 0.001$; $F = 6.4$, $p < 0.02$, respectively). The second orthogonal comparison showed that AM and HF volumes of patients (group I plus group II) were significantly smaller than controls ($F = 8.9$, $p < 0.005$; $F = 21.0$, $p < 0.001$, respectively).

Table 1. Summary of data from both groups of patients

Pt no.	Age	Sex	PFC	Classif.	R. HF	L. HF	R. AM	L. AM	Pathology
1	39	F	Yes, 6 y	L. TLE	4348.1	4259.9	3096.6	2749.2	MTS severe (+AM)
2	29	F	Yes, 10 mo	L. TLE	4377.8	3228.3	2124.9	1677.6	MTS moderate (+AM)
3	23	M	Yes, 1 y	R. TLE	3509.4	4575.7	2681.8	3014.3	MTS moderate
4	36	M	Yes, 10 mo	L. TLE	4555.2	4004.2	2124	1906.9	MTS severe
5	18	F	Yes, 9 mo	L. TLE	4087.6	3782.5	2247.7	2026.9	MTS mild
6	24	F	Yes, 9 mo	L. TLE	5453.5	3433.5	2796.5	2428.8	MTS moderate
7	22	M	Yes, 7 mo	L. TLE	4466.9	3470	2942.8	2345.5	—
8	37	F	Yes, 6 mo	R. TLE	2549.1	3450.9	1573.7	2145.9	—
9	48	F	Yes, 6 mo	L. TLE	4347	2208.9	2535.7	1701.1	MTS moderate
10	23	F	Yes, 5 mo	L. TLE	4142.9	3613.7	2506.2	1989.4	MTS moderate
11	44	M	Yes, 1 y	R. TLE	3433.2	4179.2	2217.9	2895.7	MTS severe
12	31	F	Yes, 7 mo	L. TLE	5475.1	3688.6	3068.5	2429.7	MTS severe
13	26	F	Yes, 8 mo	L. TLE	4997.2	3272.3	2409.7	1615.2	—
14	19	M	Yes, 2 y	R. TLE	3534.5	4925	2078.1	2412	—
15	22	M	Yes, 6 mo	L. TLE	3600.9	2787.9	2587.5	2183.6	—
16	35	M	No	L. TLE	4251.9	3066.8	2917.2	2259.5	MTS moderate
17	62	F	No	L. TLE	5033.8	3576.6	2429.9	2324.2	—
18	42	M	No	L. TLE	4795.6	5106.6	2875.4	2929.3	MTS moderate
19	27	F	No	L. TLE	4599.1	3355.2	3290.6	2878.5	—
20	38	M	No	L. TLE	5369.4	3674.7	—	—	MTS moderate
21	49	M	No	L. TLE	4556.2	3402	3193.4	2177.1	—
22	33	F	No	R. TLE	4447.4	4853.2	3136.6	3171.7	—
23	30	M	No	R. TLE	4115.2	4599.8	3065.3	3325.9	—
24	38	F	No	R. TLE	2974.5	4656.3	—	—	—
25	33	F	No	R. TLE	2119.1	3131.4	—	—	MTS mild
26	48	M	No	R. TLE	4072.8	4818	2219.5	3134.7	—
27	39	F	No	R. TLE	4760.7	4554.3	3161.9	3149.9	—
28	48	F	No	R. TLE	3564.1	3948.9	2010.5	2208.2	MTS mild
29	30	F	No	R. TLE	5175.9	5362.3	3041.8	3336.5	—
30	22	F	No	R. TLE	3707.2	4204.8	2130.8	2310.4	MTS moderate
31	37	F	No	L. TLE	4090.5	3817.2	3638.9	3587.6	MTS severe
32	56	M	No	L. TLE	4537.3	4191.5	2297.7	2210.5	—
33	35	M	No	L. TLE	4427.5	4480	2545.9	2597.3	MTS mild
34	26	M	No	L. TLE	5076.9	4250.4	3145.7	2909.5	MTS mild
35	38	M	No	L. TLE	4988.8	4543.5	2426.4	2305.8	MTS moderate
36	34	F	No	L. TLE	4755.2	4525.7	2724.7	2506	—
37	12	M	No	R. TLE	2900.4	4399.8	2805.4	3049.7	MTS severe (+AM)
38	34	F	No	L. TLE	4329	2832.3	2859.1	2206.7	MTS mild
39	45	F	No	L. TLE	4700.9	4185.9	3305.6	2616.2	—
40	35	F	No	L. TLE	5132.7	4703.5	2587.3	2271.8	—
41	31	M	No	L. TLE	4498.2	3666.7	2628.1	2280.8	—
42	45	M	No	L. TLE	4801.7	4293.3	3449	3175.9	MTS mild
43	33	F	No	L. TLE	3969	3088.6	—	—	MTS mild

Classif. = patients were classified as right temporal lobe epilepsy (**R. TLE**) or left temporal lobe epilepsy (**L. TLE**) according to the results of ictal and interictal EEG findings.

PFC = history of prolonged febrile convulsions in early childhood. The numbers denote the age at which these occurred or of the first episode when there was more than one.

R. AM, L. AM, R. HF, and L. HF represent, respectively, the right and left amygdala and right and left hippocampal formation volumes in mm^3.

Mesial temporal sclerosis (**MTS**) was classified as severe, moderate, or mild. **+AM** means that pathologic changes were more important in amygdala than in the hippocampal region.

The ANOVA showed that the amounts of asymmetry between sides (R−L) for AM and HF were statistically different among the three groups ($F = 10.0$, $p < 0.0001$; $F = 13.9$, $p < 0.0001$, respectively). The first orthogonal comparison showed a significant difference between the two groups of patients, with more pronounced AM and HF asymmetry in group I (patients with PFC) ($F = 5.9$, $p < 0.02$; $F = 4.6$, $p < 0.05$, respectively). The second orthogonal comparison showed that the amount of side asymmetry for AM and HF was significantly more pronounced in the patients (group I plus group II) than in the controls ($F = 14.1$, $p < 0.001$; $F = 23.1$, $p < 0.001$, respectively).

Combining the AM and HF measurements, we found a significant asymmetry (with reduced volume on the side of EEG focus) in *all* patients of group I and in 85% of group II.

In 30 patients who underwent surgery, specimens of mesial temporal structures available for

pathologic study were satisfactory in 24—10 from group I and 14 from group II. Patients in group II showed less severe degrees of MTS than in group I, and this almost reached statistical significance, with $p = 0.075$ (Fisher's exact test; table 4).

The mean age of patients of group I was lower than patients of group II ($p = 0.02$, t test).

Discussion. This study showed a reduction of the volume of mesial temporal structures, which coincided with the side of the EEG focus in patients with TLE. This volume reduction was more pronounced in patients with a history of PFC in early childhood than in those without PFC. There was also a higher proportion of more severe MTS in patients who had PFC compared with those with no antecedent of PFC.

Table 2. Mean values of volumetric measurements from the group of patients with PFC (group I) and patients without PFC (group II)

	AM	HF	R–L AM	R–L HF
Group I	2107.0	3385.1	486.8	1080.9
Group II	2617.0	3878.5	309.5	749.4

The mean volumes of amygdala (AM) and hippocampal formation (HF) ipsilateral to the EEG focus are significantly smaller in group I. The amount of side asymmetry, represented here by the absolute values of right minus left side difference (R–L), is more pronounced in group I.

The values are expressed in mm^3.

Volumetric measurements showed more pronounced atrophy of AM in patients who had had PFC, although the HF volumes were also smaller in this group. There has been little attention paid to the AM as an important contributor to epileptogenesis in TLE.[6] In this series, all patients who had hippocampal sclerosis had equally severe pathology of the AM when AM tissue was available for analysis. Indeed, the AM was more severely affected than the hippocampus in three patients, two of them with antecedents of PFC. The prior lack of emphasis on pathologic changes in the amygdala could be explained by the fact that this structure is often damaged at surgery because of its location and anatomic conformation.[6] However, a recent pathologic report described a group of patients with sclerosis restricted to the AM as opposed to the HF.[22]

Our findings agree with other reports showing a

Table 4. Pathologic categories in the two groups of patients

	Number of patients	
	MTS mild	MTS moderate to severe
Group I (with PFC)	1	9
Group II (no PFC)	7	7
Total	8	16

$p = 0.075$ (Fisher's exact test).
See table 1 for abbreviations.

Table 3. Normal control data

Control number	Left AM	Right AM	Left HF	Right HF	R–L AM	R–L HF
1	3344.9	3421.4	5091.6	5181.4	76.4	89.8
2	3112.0	3056.3	4634.6	4783.1	-55.7	148.5
3	3315.0	3373.1	4593.0	4496.2	58.0	-96.9
4	2896.1	2877.0	4487.6	4479.9	-19.1	-7.7
5	2530.7	2565.5	4613.3	4717.6	34.8	104.3
6	3297.9	3454.0	4927.4	5034.6	156.1	107.2
7	3519.8	3561.3	4576.9	4734.6	41.5	157.6
8	2694.7	2899.9	4244.1	4433.4	205.3	189.3
9	2840.9	2893.6	4332.1	4460.4	52.7	128.3
10	3264.1	3444.1	4892.2	5087.6	180.0	195.3
11	2744.4	3023.2	4308.4	4564.3	278.7	255.8
12	2805.1	2760.5	4538.3	4601.9	-44.6	63.6
13	2702.8	2921.3	4453.8	4672.2	218.5	218.4
Mean	3005.3	3096.2	4591.8	4711.3	90.9 (109.3*)	119.5 (135.9*)
SD	303.8	304.7	241.2	239.9	102.7 (82.8*)	91.4 (65.3*)

AM Amygdala volumes.
HF Hippocampal formation volumes.
R–L AM Right minus left side difference for AM volumes.
R–L HF Right minus left side difference for HF volumes.

Negative signs indicate that the right side is smaller.

The values are represented in mm^3.

* Mean and standard deviation of absolute amount of asymmetry (ie, ignoring negative signs).

correlation between the occurrence of PFC in infancy and the degree of MTS.[1,4,5,8] We did not find a correlation between the total duration of epilepsy and the severity of MTS.[23] Our data showed that patients with early convulsions, who had MRI volumetric studies in early adolescence, had greater atrophy than those whose epilepsy was of longer duration but who did not have convulsions in early life. This does not rule out the possibility that repeated seizures over a longer period may produce additional, more subtle, hippocampal damage, of the kind described by Mouritzen Dam.[2]

In a previous series from our institution, we found a higher frequency of gestational or birth complications (mostly minor) in TLE patients who had had prolonged febrile convulsions. This suggests that such perinatal or other preexisting factors may make it more likely for a febrile convulsion to be prolonged or complex in children who have a genetic predisposition for febrile convulsions. Such perinatal or preexisting factors, in themselves, may not be sufficient to produce TLE, but the prolonged and often lateralized febrile convulsion leads to hippocampal sclerosis and focal temporal epileptogenicity (Abou-Khalil et al, in preparation).

Patients with TLE preceded by PFC represent a distinctive group. They have fairly homogeneous clinical and pathologic characteristics and constitute 35% to 40% of the patients undergoing surgical treatment for TLE at the Montreal Neurological Hospital. A history of PFC in infancy allows one to predict the likelihood of a good surgical outcome (Abou-Khalil et al, in preparation).

The smallness and pathologic abnormality of the AM may identify a subgroup among patients with mesial sclerosis causing TLE.[24] This may explain the effectiveness, in some patients, of corticoamygdalectomy with minimal or no hippocampal resection.[19] Recognition and quantification of these atrophic changes may be important in planning treatment for patients in whom resection of the hippocampus is not safe because of severe memory impairment.

Further studies are required to establish the relative importance of unilateral or bilateral atrophy of the amygdala, the hippocampus, or both, compared with the EEG, neuropsychological studies, and functional neuroimaging. This may lead to a more rational and less invasive approach to the treatment of TLE.

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