In unselected patients with intractable temporal lobe epilepsy (TLE), approximately 15% do not have detectable hippocampal atrophy on MRI. The purpose of this study was to evaluate whether T2 relaxometry can identify hippocampal pathology and lateralize the epileptic focus in patients with intractable TLE, who do not demonstrate hippocampal atrophy on volumetric MRI (MRIV). We selected 14 patients with unilateral TLE who had unilateral atrophy and 11 patients with unilateral TLE who had no evidence of atrophy on MRIV. Images were acquired on a 1.5 T MR scan using a dual echo sequence with 23 contiguous oblique coronal slices in all patients and in 14 healthy subjects. Fitting a single exponential decay equation to the imaging data generated T2 maps. Averages of six slices containing the head, body, and tail of the hippocampus were used to calculate hippocampal T2 relaxation times (HT2). The epileptic focus was defined by history, video-EEG, and surgical response. All TLE patients with hippocampal atrophy and 9/11 (82%) patients with normal MRI had abnormally high HT2 ipsilateral to the epileptic focus. Bilateral abnormal HT2 were found in 6/14 (43%) of patients with unilateral hippocampal atrophy and 2/11 (18%) of patients with normal MRI. However, this increase was always greater ipsilateral to the epileptic focus. Qualitative hippocampal pathology showed gliosis and neuronal loss in 10/14 operated patients with hippocampal atrophy on MRIV and in 5/7 operated patients with normal MRI. In conclusion, hippocampal T2 mapping provides evidence of hippocampal damage in the majority of patients with intractable TLE who have no evidence of atrophy on MRI and can correctly lateralize the epileptic focus in most patients.

Key Words: T2 relaxometry; temporal lobe epilepsy; mesial temporal sclerosis; normal MRI.

INTRODUCTION

Hippocampal sclerosis is the most common pathology underlying pharmacologically intractable temporal lobe epilepsy (TLE) (Babb and Brown, 1987). On visual inspection of conventional magnetic resonance imaging (MRI) scans, hippocampal sclerosis is often associated with noticeable hippocampal atrophy and increased T2 signal intensity (Jackson et al., 1993a). Nevertheless, distinguishing a mildly sclerotic hippocampus from a normal hippocampus is often difficult on the basis of qualitative analysis of these MRI features.

The sensitivity of visual MRI analysis can be increased by the use of hippocampal volumetric MRI (MRIV) (Jack, Jr. et al., 1990). Using such an approach, a lateralized atrophy can be found in 70–95% of patients with intractable TLE (Jack, Jr. et al., 1990; Cendes et al., 1993). The hippocampal volume loss detected by MRI correlates with gliosis and neuronal loss in resected specimens (Cascino et al., 1991; Kuzniecky et al., 1987). Nevertheless, hippocampal volumes will be normal in 5–15% of patients with intractable TLE (Jackson et al., 1994)—even though the majority of these individuals demonstrate histopathological evidence of hippocampal sclerosis (Van Paesschen et al., 1995; King et al., 1996; Bronen et al., 1994; Van Paesschen et al., 1996; Jackson et al., 1994).

The sensitivity of MRI analysis can also be increased by quantitative measurement of hippocampal T2 relaxation times. In patients with intractable TLE and hippocampal atrophy, T2 relaxation times within the hippocampal gray matter ipsilateral to the epileptic focus have been shown to increase by at least 10 ms relative to normal controls (Woermann et al., 1998; Namer et al., 1998; Van Paesschen et al., 1995; Jackson et al., 1993). It remains unclear as to whether or not this technique would be useful in lateralizing TLE patients with normal hippocampal volumes. Previous techniques for in vivo measurement of T2 relaxation time have employed a 16-echo sequence that only allowed measurement from a limited number of single slices within the hippocampus (Kalviainen et al., 1998; Jackson et al., 1993). Using a standard dual-echo sequence, it is possible to generate multislice T2 maps (Woermann et al., 1998; Duncan et al., 1996) in a coronal slice containing the head, body, and tail of the hippocampus (Kalviainen et al., 1998; Jackson et al., 1993).
orientation thereby allowing better in-plane resolution of T2 signal in the hippocampus.

The purpose of the present study was to evaluate whether or not hippocampal T2 relaxometry with a dual-echo pulse sequence (Duncan et al., 1996) would help identify hippocampal pathology in patients with intractable TLE who do not demonstrate hippocampal abnormalities on conventional MRI and hippocampal atrophy on volumetric MRI.

METHODS

Subjects. We studied 11 consecutive patients with intractable TLE (mean age 35 years, range = 21 to 50; standard deviation (SD) = 14) referred because these patients had normal MRI and normal hippocampal MRI volumetry (MRIV). These patients are referred in the text as patients with normal MRI. As a disease control group we selected 14 TLE patients (mean age 36 years, range = 18 to 56; SD = 9) with evidence of hippocampal atrophy on both visual MRI and MRIV.

Hippocampal volumes and T2 relaxation times were obtained in all patients (14 females and 11 males). Hippocampal volumes were also obtained in a group of 18 healthy subjects (7 females and 11 males) whose mean age was 26 years (range = 20 to 40; SD = 6). Hippocampal T2 relaxation times were obtained in a separate group of 14 normal controls (5 females and 9 males) whose mean age was 36 years (range = 28 to 47; SD = 6).

Patients were diagnosed having TLE on the basis of their clinical history, seizure pattern (e.g., aura and complex partial seizures), neuroradiological and neuropsychological findings. All patients had long-term video-EEG monitoring with sphenoidal electrodes. Six patients were implanted with multiple stereotactic intracranial depth electrodes (SEEG) (Olivier et al., 1987) because extracranial EEG recordings did not provide clear localization or lateralization of the seizure onset. Of these six patients, four had normal MRI and two had hippocampal atrophy. In all patients SEEG demonstrated seizure onset in the hippocampus. Patients were classified as having either left-sided or right-sided epileptic focus if 70% or more of interictal epileptiform abnormalities and seizure onset were recorded form that side, and based on response to surgical treatment in 21/25 patients. Based on these criteria, 15 patients were classified as having left-sided TLE (LTLE) and 10 right-sided TLE (RTLE).

Surgical treatment and histopathology. All 14 patients with hippocampal atrophy were operated upon since being studied: 11 underwent a selective amygdalo-hippocampectomy and 3 underwent a temporal cortical resection with amygdalo-hippocampectomy. Seven of the 11 patients with normal MRI were operated, three of who had previous SEEG study. In the remaining four, extracranial EEG was sufficient to lateralize the epileptic focus. Three underwent a selective amygdalo-hippocampectomy and four underwent a temporal cortical resection with amygdalo-hippocampectomy. Qualitative histopathological evaluation (Meencke and Veith, 1991) of the resected tissue revealed astrogliosis and hippocampal neuronal loss in 10 patients with hippocampal atrophy and in 5 patients with normal MRI. The samples of mesial temporal structures available for pathologic examination were suboptimal in four patients with hippocampal atrophy and in two patients with normal MRI because surgical removal was done by subpial aspiration. Mean postoperative follow-up was 24 months (range = 18 to 30). Thirteen operated patients who had hippocampal atrophy have been seizure free since surgery (Engel’s classification I) (Engel, J. r. et al., 1993), and one had rare seizures (Engel’s classification II). Of the seven operated TLE patients with normal MRI, six were seizure free and one had rare seizures (Engel’s classification II).

MRI visual analysis. Visual analysis was performed using spin-echo sequences (FOV 250 mm; matrix 256 x 256) with sagittal and coronal T1-weighted images (TR 550 ms, TE 19 ms), and proton-density and T2-weighted images (thickness 3.0 mm, gap 0.3, TR 2100 ms, TE 20, 78 ms). Fluid attenuation inversion recovery (FLAIR) images were also obtained (slice thickness 3.0 mm, interslice gap 0.3 mm, TR 6000 ms, TE 150 ms, TI 1900 ms, FOV 230 mm).

MRI volumetric analysis. Images were acquired on a 1.5 T Siemens Vision Magnetom (Erlangen, Germany) using a T1-weighted 3D gradient-echo sequence (TR 18 ms, TE 10 ms, 1 signal average, flip angle 30°, matrix 256 x 256, FOV 250, thickness 1 mm). Approximately 170 slices with an isotropic voxel size of 1 mm³ were acquired. These images were then analyzed on a Silicon Graphics workstation (Mountain View, CA). Images were automatically registered into stereotaxic space (Talairach and Tournoux, 1988) to adjust for differences in total brain volume and brain orientation, and to facilitate the identification of boundaries by minimizing variability in slice orientation (Collins et al., 1994). This transformation maximized the cross-correlation between each individual’s MR image and a target image consisting of the average of over 300 normal MR images already registered into stereotaxic coordinates (Talairach and Tournoux, 1988). The nine transformation parameters took into account differences in brain position (three translations, three rotations) and brain size (anisotropic scaling in the three principal axes). Registration of the images into stereotaxic space was then accomplished by applying the transformation matrix to the individual’s image coordinates. This process normalized for differences in total intracranial volume. Each image underwent automated correction for intensity nonuniformity due to
radiofrequency inhomogeneity of the MR scanner and intensity standardization (Sled et al., 1998). This correction produces consistent relative gray matter, white matter, and CSF intensities. For each individual, the hippocampus was segmented manually using an interactive mouse-driven software. Anatomical guidelines for outlining the hippocampus have been described previously (Watson et al., 1992).

**T2 relaxometry.** For computation of T2 values, images were acquired using a conventional dual-echo spin-echo pulse sequence (TE 20, 120 ms; TR 3240 ms, 1 signal average, flip angle 90°, matrix 256 × 256, FOV 240, slice thickness 5 mm with 0.5 mm interslice gap. Flow-compensated gradients were used in all three directions. Twenty-three contiguous oblique coronal slices covering the whole brain with a voxel size of 0.94 × 0.94 mm² were acquired. Slices were oriented orthogonal to the axis of the hippocampal body. For each individual, single-exponential-decay equations were fit to the T2 imaging data obtained in each pixel and then T2 relaxation times were calculated for each pixel. For each slice an image was then constructed in which pixel-intensity corresponded to the calculated T2 relaxation time. Averages of 6 slices (range = 5 to 7) containing the head, body, and tail of the hippocampus were used to calculate left and right hippocampal T2 relaxation times (HT2). For each slice, HT2 values were measured by placing the largest possible region of interest (ROI) within the anatomical boundaries of the hippocampus with a mean number of 37 pixels. Boundaries where partial volume effects from cerebrospinal fluid might have occurred were avoided (Fig. 1). Mean T2 relaxation time was calculated in each ROI. Finally a total mean HT2 was calculated by averaging the values of all voxels in all slices. The examiner of the T2 maps was unaware of the MRI and the EEG results.

**Statistical analysis.** Group differences for left and right hippocampal volumes and HT2 were examined using a multivariate analysis of variance (MANOVA) with one between-subjects grouping factor (group: normal controls, LTLE/RTLE with normal MRI, LTLE/RTLE with hippocampal atrophy) and one within-subjects factor (hemisphere: left and right). Each MANOVA described above was followed by Bonferroni-corrected planned-comparisons. For each individual, left-right asymmetries in hippocampal volumes and HT2 were expressed calculated as follows: \((L - R)/[(L + R)/2]\), where L and R refer to the mean volumes or mean HT2 of the left and right hippocampus, respectively.

Group differences in left-right hippocampal volume and HT2 asymmetries were examined using an analysis of variance (ANOVA) with one between-subjects grouping factor (group: normal controls, LTLE/RTLE with normal MRI, LTLE/RTLE with hippocampal atrophy).

**Correlation analysis.** The correlation between HT2 and age was assessed using the Pearson correlation coefficient (r). We compared age at onset of recurrent
seizures, age at evaluation, and duration of the epilepsy between TLE patients with hippocampal atrophy and those with normal MRI using the Student t test.

We analyzed differences in family history, febrile convulsions, seizure pattern, and EEG using Fisher’s exact test.

**RESULTS**

**Group analysis.** Results of group analysis for MRI volumetry and T2 relaxometry are shown in Fig. 2.

**MRI volumetry.** For the 18 normal controls studied: (i) The mean right hippocampal volume (2510 mm³; SD = 285) was significantly greater than the mean left hippocampal volume (2287 mm³; SD = 283) \(t_{17} = -4.63, P = 0.0002\) (paired t test); (ii) the mean left-right hippocampal asymmetry score was -0.094 (SD = 0.085). The mean left-right hippocampal volume asymmetry score was significantly different (i.e., lower) than zero \(t_{17} = 4.72, P = 0.0002\) (one-sample t test).

There was a significant interaction of hemisphere and group on mean hippocampal volumes \(F_{4,38} = 16.29, P < 0.00001\). There was also a significant main effect of group on the mean left-right hippocampal volume asymmetry scores \(F_{4,38} = 20.01, P < 0.00001\).

The planned comparisons showed that neither the mean hippocampal volumes nor the mean left-right hippocampal volume asymmetry scores differed signif-

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**FIG. 2.** Hippocampal volume (A) and hippocampal T2-relaxation-time data (B) in normal controls (NC), patients with left temporal lobe epilepsy (LTLE), and right temporal lobe epilepsy (RTLE), either with hippocampal atrophy (Atro) or normal hippocampal volumes (Norm), are presented in grouped box-and-whiskers plots. The center horizontal line marks the median of the sample, the upper and lower edges of the box (the hinges) mark the 25th and 75th percentiles (i.e., the central 50% of the values fall within the box)—the distance between these hinges being referred to as the Hspread, and the “whiskers” extend from the box and show the range of values that fall within 1.5 Hspread. The dots represent individual subjects and the squares represent the group means. The broken lines designate 2 SD from the mean of normal controls (see Results for explanations).
TABLE 1
Clinical and EEG Features of TLE Patients with Hippocampal Atrophy (TLE-Atro) and TLE Patients with Normal MRI (TLE-Norm)

<table>
<thead>
<tr>
<th></th>
<th>TLE-Atro (n = 14)</th>
<th>TLE-Norm (n = 11)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset (mean ± SD)</td>
<td>9 ± 6 years</td>
<td>20 ± 10 years</td>
<td>0.002*</td>
</tr>
<tr>
<td>Age at evaluation (mean ± SD)</td>
<td>35 ± 14 years</td>
<td>37 ± 9 years</td>
<td>ns*</td>
</tr>
<tr>
<td>Duration of epilepsy (mean ± SD)</td>
<td>27 ± 12 years</td>
<td>16 ± 13 years</td>
<td>0.04*</td>
</tr>
<tr>
<td>Family history of epilepsy</td>
<td>23%</td>
<td>50%</td>
<td>ns*</td>
</tr>
<tr>
<td>History of febrile convulsions</td>
<td>31%</td>
<td>0%</td>
<td>0.04*</td>
</tr>
<tr>
<td>Complex partial seizures</td>
<td>Aura had a significantly higher mean</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EEG spike focus</td>
<td>Unilateral</td>
<td>Bilateral</td>
<td></td>
</tr>
<tr>
<td></td>
<td>69%</td>
<td>31%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>70%</td>
<td>30%</td>
<td></td>
</tr>
</tbody>
</table>

Note. ns, not significant (P ≥ 0.05); SD, standard deviation.
* Student’s t test.
* Fisher’s exact test.

significantly in TLE patients with normal MRI compared to normal controls (P > 1.00). Both the left (P < 0.000001) and right (P = 0.0001) hippocampus were of significantly smaller volume in TLE patients with hippocampal atrophy than in normal controls. However, TLE patients with hippocampal atrophy had a significantly lower mean asymmetry score (left hippocampus smaller than right hippocampus) than normal controls (P = 0.0002). The right hippocampus was significantly smaller in RTLE patients with hippocampal atrophy than in normal controls (P < 0.000001).

T2 relaxometry. For the 14 normal controls studied: (i) the mean left HT2 was 79.8 ms (range = 77.4 to 82.3; SD = 1.3); (ii) the mean right HT2 was 80 ms (range = 76.8 to 82.4; SD = 1.4); and (iii) the mean left-right asymmetry score was −0.016 (SD = 0.024). The HT2 in the left hippocampus and the right hippocampus in these subjects did not differ (t13 = −0.25, P = 0.81, paired t test). Correspondingly, the mean left-right asymmetry score did not differ significantly from zero in these individuals either (t13 = −0.24, P = 0.81, one-sample t test). One factor ANOVA showed that the HT2 of the head, body and tail were not different (F = 0.56, P = 0.7). There was no correlation between HT2 and age (r = 0.015; P = 0.94).

There was a significant interaction of hemisphere and group on HT2 (F4,34 = 55.35, P < 0.000001). There was also a significant main effect of group on the mean left-right HT2 asymmetry scores (F4,34 = 75.16, P < 0.000001). The planned comparisons showed that: LTLE patients with normal MRI had a left HT2 that was significantly higher than that of normal controls (P = 0.009). RTLE patients with normal MRI had a right HT2 that was significantly higher than that of normal controls (P = 0.0033). Both the left (P < 0.000001) and right (P = 0.0043) HT2 were significantly higher in patients with LTLE and hippocampal atrophy than in normal controls. However, LTLE patients with hippocampal atrophy had a significantly higher mean asymmetry score (left HT2 higher than right) than normal controls (P < 0.000001). LTLE patients with hippocampal atrophy had a significantly higher mean left HT2 than LTLE patients with normal MRI (P < 0.000001). RTLE patients with hippocampal atrophy had higher right HT2 than normal controls (P < 0.000001). RTLE patients with hippocampal atrophy had a significantly higher right HT2 than RTLE patients with normal MRI (P = 0.0002).

Individual analysis based on T2 relaxometry. For analysis of individual patients, the following values were considered abnormal: (i) left or right HT2 greater than two SD above the mean of normal controls; (ii) left-right asymmetries greater than two SD above or below the mean of normal controls.

In TLE patients with hippocampal atrophy, 8/14 (57%) had high HT2 ipsilateral to the epileptic focus. Bilateral high HT2 was found in the other 6 (43%) patients. However, all of them were lateralized correctly using the asymmetry score. Therefore, all 14 TLE patients with hippocampal atrophy were correctly lateralized.

In patients with normal MRI, 8/11 (73%) had high HT2 on the side of the epileptic focus. Of the two patients with bilateral high HT2, one was lateralized correctly using the asymmetry score. One patient had bilateral symmetric normal HT2. Therefore, 9/11 (82%) TLE patients with normal MRI were lateralized correctly on the basis of the HT2 data.

In four patients (4/25 = 16%) HT2 was normal in the slice at the level of the brainstem (hippocampal body) as used in previous studies (Jackson et al., 1993b), but abnormally high in the head of the hippocampus. All four patients had normal MRI.

Demographic and clinical data analysis. Results of demographic and clinical data analysis are shown in Table 1. Comparing TLE patients with normal MRI and patients with hippocampal atrophy, we found a
higher age at onset of epilepsy in TLE patients with normal MRI (20 years vs 9 years; P = 0.002). The duration of epilepsy was shorter in patients with normal MRI compared to patients with hippocampal atrophy (16 years vs 27 years; P = 0.04). No TLE patient with normal MRI had a history of febrile convulsions (P = 0.04). There was no statistically significant difference between the two group of patients comparing age, clinical, and EEG characteristics.

**DISCUSSION**

Our study shows that T2 mapping is a sensitive technique for detecting hippocampal abnormalities in TLE patients. Using hippocampal T2 relaxometry we correctly lateralized the epileptic focus in all TLE patients with hippocampal atrophy and most of those with normal MRI.

The conventional dual echo multislice sequence used in our study increased the sensitivity of the T2 relaxation time measurements over single slice techniques (Jackson et al., 1993b) by demonstrating nonuniform abnormalities throughout the length of the hippocampus that would not be detected using a multiecho sequence allowing only measurements from a single slice. In our study, 4 of the 11 TLE patients with normal MRI (36%) had abnormal T2 relaxation time values only at the level of the head of the hippocampus. These findings are consistent with those of Woermann et al. (1998), who reported three TLE patients with abnormal T2 relaxation times in the anterior portion of the hippocampus ipsilateral to their epileptic focus; importantly, these abnormalities were not evident on a multiecho sequence (Van Paesschen et al., 1997b; Jackson et al., 1993b; Van Paesschen et al., 1995b) with a single slice at the level of the body of the hippocampus and two of their patients had normal hippocampal volumes.

Out of the 14 TLE patients with unilateral hippocampal atrophy in our series, 6 (43%) had bilateral T2 abnormalities. In these cases, however, T2 times were always found to be higher ipsilateral to the epileptic focus. Autopsy studies of hippocampi from patients with long-standing TLE have revealed that, although the degree of cell loss is usually asymmetrical, some cell loss is observed bilaterally in the majority of patients even when all the evidence suggests unilateral hippocampal seizure onset (Babb, 1991). Our results are in agreement with previously published data (Woermann et al., 1998; Jackson et al., 1993, 1994; Van Paesschen et al., 1997; Namer et al., 1998) and suggest that T2 relaxometry is superior to volumetric studies in detecting subtle structural damage in the hippocampus.

Compared to TLE patients with hippocampal atrophy, we found that patients with normal MRI had a later onset and a shorter duration of epilepsy, and no history of prolonged febrile seizures in early childhood. Patients with prolonged febrile convulsions in early childhood and a long duration of epilepsy have been shown to have a significantly smaller hippocampus ipsilateral to the seizures focus compared to those without such a history (Tasch et al., 1999; Mathern et al., 1996, 1995). The absence of febrile seizures in our patients with normal MRI, in addition to a shorter duration of epilepsy, could therefore explain a less severe hippocampal pathological change, not sufficient to appear on MRI as reduced hippocampal volume, but detected by T2 relaxometry.

We found that TLE patients with hippocampal atrophy had higher T2 relaxation times in the epileptic hemisphere as compared to those with normal MRI. In our study, in those patients in whom resected tissue was available, histopathological evaluation revealed astrogliosis and hippocampal neuronal loss in 10 patients with hippocampal atrophy and in 5 patients with normal MRI. Because of the qualitative nature of our analysis, we were not able to compare the degree of cell loss between the two groups of patients. However, studies using quantitative cell counts have shown that, compared with patients who have hippocampal atrophy, patients with normal MRI have less cell loss and significantly fewer glial cells in CA1 (King et al., 1996). Therefore, possible causes for a minor degree of T2 abnormality in our patients with normal MRI are either a lower degree of hippocampal cell loss, or equilibrium between cell loss and gliosis.

The identification of mesial temporal sclerosis by MRI greatly influences the clinical management and the postoperative seizure outcome of intractable TLE (Kuzniecky et al., 1993; Jackson Jr. et al., 1992). Seizure freedom can be expected in the majority (80–90%) of patients in whom the ictal onset on scalp EEG coincides with the side of hippocampal atrophy on MRI (Jackson Jr. et al., 1992; Cascino et al., 1995; Engel, Jr. et al., 1998). It is not known how the presence of hippocampal sclerosis without hippocampal atrophy affects the surgical prognosis. Jackson et al. (1994) reported that 5 of 6 such patients were seizure free after surgery. King et al. (1996) reported that 6 of 10 such operated patients were completely seizure free after anteromedial temporal lobectomy, two had >75% reduction in seizure frequency, and two had no worthwhile improvement. In our series, 6 of 7 operated patients with normal MRI were completely seizure free and one had a reduction in the number of seizures. Because of the limited number of operated patients, it is premature to draw a general conclusion concerning the surgical outcome in this subgroup of TLE patients. However, our data show that patients without hippocampal atrophy on MRI but with abnormal hippocampal T2 relaxation times may still have a good surgical outcome, even though the likelihood is proba-
bly somewhat lower than in patients with unilateral hippocampal atrophy.

In our series, 4 of 11 patients with normal MRI underwent intracranial recordings with SEEG and all had seizure onset exclusively in the hippocampus. There was an agreement between the side of increase of T2 time and the lateralization of the epileptic focus as determined by SEEG in all patients. In previously reported series (Jackson et al., 1994; King et al., 1996), the majority of TLE patients without hippocampal atrophy also underwent intracranial EEG recordings, to demonstrate seizure onset in the hippocampus that was eventually resected. Thus, T2 relaxometry could eventually lead to a reduction of chronic invasive EEG recordings in this subgroup of patients.

In conclusion, hippocampal T2 mapping provides evidence of hippocampal damage in the majority of patients with intractable TLE who have no evidence of atrophy on MRI and can correctly lateralize most patients. This technique approach can be a useful adjunct to presurgical evaluation of TLE patients with normal MRI and could lead to a reduced need for invasive recordings in this group of patients. If hippocampal volume measurements are normal in the presence of clinical and EEG evidence of mesial TLE, further detailed MRI assessment is important prior to concluding that the hippocampus is structurally normal.

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